THE

THEORY AND PRACTICE OF MEDICINE

A TREATISE

ON THE

THEORY AND PRACTICE OF MEDICINE

BY

JOHN SYER BRISTOWE, M.D. LOND.

FELLOW AND FORMERLY CENSOR OF THE ROYAL COLLEGE OF PHYSICIANS; SENIOR PHYSICIAN TO AND JOINT LECTURER ON MEDICINE AT ST. THOMAS'S HOSPITAL; EXAMINER IN MEDICINE TO THE ROYAL COLLEGE OF SURGEONS; FORMERLY EXAMINER IN MEDICINE TO THE UNIVERSITY OF LONDON, AND LECTURER ON GENERAL PATHOLOGY AND ON PHYSIOLOGY AT ST. THOMAS'S HOSPITAL

SECOND EDITION

LONDON
SMITH, ELDER, & CO., 15 WATERLOO PLACE
1878

PREFACE

TO

THE SECOND EDITION.

In preparing the second edition for the press, I have taken much more pains than may at first sight be apparent. There is probably not a single page on which I have not made many corrections either of fact or of language, and for the sake of clearness or precision of statement. I have here and there also rearranged my matter when it seemed to me economical of space or otherwise convenient to do so. The more important changes and additions are comprised in the following list:—

In the section on 'Fever' I have introduced modifications and corrections, for most of which I am indebted to Dr. Sanderson's papers on 'The Process of Fever.'

In my introductory remarks upon the infectious fevers contained in the first edition, I incorporated Dr. Klein's observations on the contagium of small-pox. His conclusions have long since been admitted to be erroneous. I have therefore cancelled all reference to these observations, and replaced them by new matter.

In my original account of cow-pox, I somewhat hastily assumed that Chauveau's researches had disproved the identity between cow-pox and small-pox. But a recent reperusal of Messrs. Ceely and Badcock's papers has satisfied me that the evidence, as it at present stands, is in favour of the views which they maintain. Under the circumstances I have thought it right to revise my observations on the subject accordingly.

I have recast my brief sketch of paralytic affections of the larynx, mainly under the guidance of Von Ziemssen's essay contained in his cyclopædia.

The sections on Inflammation and Cirrhosis of the Liver, and those on Nephritis and Bright's disease, have been almost entirely rewritten.

I have now, I trust, attached due importance to Dr. Lewis's observations with regard to the Filaria sanguinis hominis in relation to chyluria, and elephantiasis; and I have made several important changes in that part of the work devoted to the chemical examination of the urine.

I have availed myself largely of Dr. Ferrier's investigations in my introductory remarks on the physiology of the nervous system; and have incorporated several illustrations derived from his work on the 'Functions of the Brain,' and from other sources.

Lastly, I have added: a description of myxœdema, or the cretinoid disease in adults, first described by Sir W. Gull; an introductory thesis on the physical examination of the abdomen; and a brief section on the uses of electricity for diagnostic purposes and as a therapeutical agent.

11 Old Burlington Street:
September 1878.

PREFACE

TO

THE FIRST EDITION.

In placing this work before those for whom it was especially written, namely, the junior members of the profession and students in Medicine, I may be permitted to make a few preliminary remarks, partly by way of explanation, partly by way of acknowledgment, partly apologetic.

The first thought, as I suppose, of everyone who sits down to write a scientific book is bestowed upon the arrangement of his matter. It was my first thought. The classification of disease, moreover, is a subject to which I have devoted a good deal of attention. But I had long formed the opinion that it is impossible, in a work on Medicine, intended to be practical, to arrange diseases on strictly scientific principles; and in this opinion further consideration of the matter only confirmed me. Consequently the arrangement which I have adopted is for the most part artificial, and to be defended only on grounds of convenience. Certain affections I have grouped together as 'Specific Febrile Diseases;' but all others, with in many cases more or less disregard of accuracy, have been classified as Local Diseases. I may add that, in respect of the diseases of individual organs, I have for the most part arranged them, though without expressly indicating the fact, in the following order, namely: Inflammations, Morbid Growths (including Tubercular and Syphilitic Formations), Parasitic Diseases, Degenerations, and Mechanical and Functional Affections. I have not hesitated, however, in many instances to depart from this arrangement.

The selection of subjects to be discussed in a treatise intended to occupy a moderate compass is by no means easy. Medicine is inextricably interwoven with Surgery and with what it is now fashionable to term 'gynæcological medicine.' Moreover, several other departments of practice, especially perhaps insanity, are now relegated to specialists, and have attained such importance as to need special hospitals, and to have a literature of their own. And again, many diseases, and more particularly local diseases, which doubtless have a substantial existence, are either not recognisable by specific symptoms during life, or are of very trivial importance, so that it would be a waste of time and space even to enumerate them. I trust that, under such circumstances, I shall be pardoned for having treated some important subjects superficially; for having omitted many subjects that it may seem to some persons that I should have included in my work; and for having occasionally introduced topics which may appear to be beyond the sphere of Medicine, in the restricted sense of that term.

In discussing each subject, and more especially in discussing each disease, my aim has been to give in a readable form as much information as I could include within a limited space. With that object, my practice has been in every case to read the subject up carefully; to compare the knowledge thus acquired or renewed with the results of my own experience, in those cases in which I had any experience; and then, having taken a more or less definite view of the whole subject, and while my mind was still full of it and of its details, to write as clear and as comprehensive an account as I was capable of. Each article may therefore be regarded as expressing in a condensed form the fulness of my knowledge of its subject at the moment at which it was written. This method of procedure will partly explain both the *ex cathedrâ* tone in which I have, I believe, generally expressed myself, the prevailing absence of notes, quotations, and references to authorities, and perhaps also many inaccuracies and omissions.

I have throughout the work given particular prominence to the pathology and to the clinical phenomena of disease; and in all cases in which the clinical phenomena seem to be the direct consequences of definite lesions (especially therefore in the case of local diseases) my account of the morbid anatomy has been made to

precede the clinical description. It may possibly, however, seem to be an omission that I have only occasionally devoted a special paragraph to the differential diagnosis of diseases. It is so far an omission that I have been driven to it by the exigencies of space. But on the whole I do not regret it; for the distinguishing of one disease from another disease should depend, not on the simple recognition of a few leading characters, which, however carefully selected, are apt not unfrequently to fail us, but on a *bonâ fide* and thorough acquaintance with the collective phenomena of diseases. The more a student is taught to rely on one or two criteria, the less likely is he to investigate diseases intelligently, and the more apt is he to be content with hasty and inaccurate diagnoses.

In respect of the treatment of diseases, again, I may appear to have been in many cases less full and less specific than I ought to have been. The principles by which I have been guided in this matter are easy to explain. In the first place, it seemed to me that works upon the Materia Medica are the proper source from which to learn the doses in which medicines may be administered, and the best modes of combining medicines. And in the second place, in considering the details of treatment, as given in most works of medicine, it appeared to me that their authors had for the most part simply recommended those doses of drugs, those combinations of drugs, and those specific modes of administering them, to which they had accustomed themselves. I admit that the subject of my last objection will be regarded by many from quite an opposite point of view. Nevertheless, while, on the one hand, I should hesitate to force my own routine and trivialities of practice upon students, I should equally hesitate to force upon them those of other people. It seems to me best, having inculcated general principles, and pointed out the specific virtues of certain drugs, to leave the young practitioner generally as much unshackled as possible with regard to his choice of particular combinations and modes of administration. He is far more likely to make a thoughtful physician, and as I think to benefit his patient, if he adapts his drugs and his methods to the exigencies of cases as they present themselves before him, than if he follows the stereotyped procedure of some predecessor.

From first to last I have carefully avoided quoting illustrative

cases. This course has been forced upon me by the necessity under which I laboured of compressing my work within the narrowest possible limits of space. But it is a course which I adopted reluctantly, and with the full knowledge that I was thereby robbing my pages of much that might have been instructive, of much at any rate that would have rendered them pleasanter reading. Everyone who has perused them knows how much of the charm, the freshness, the vigour, the impressiveness, and the permanent interest that characterise the classical writings of Abercrombie, of Graves, of Watson, of Trousseau, and of other masters of our art depend upon the well-told cases with which they are so richly interspersed.

I have already referred to the omission to quote authorities of which I have been generally guilty. The excuses which I have to offer in reference to this matter are mainly the following:—I was anxious to economise space; I felt, moreover, that my work was not an encyclopædia, still less a history of medicine; and again, many important additions which have been made to our knowledge, even during the last few years, have already become classical, and form an integral part of the great body of Medical Science. My indebtedness, however, direct or indirect, to innumerable writers and workers I most fully acknowledge; and among these I must not fail to include my senior colleagues and former teachers of St. Thomas's Hospital, the value of whose teaching to myself I cannot exaggerate. But there are certain works on which I have drawn very largely, and to the authors of which on that account I owe special gratitude: these are, in pathology and morbid anatomy, Rokitansky's 'Pathological Anatomy,' Cornil and Ranvier's 'Manual of Pathological Histology,' and Virchow's writings, including above all his marvellous work on the 'Pathology of Tumours;' in general medicine, Sir T. Watson's 'Lectures on the Principles and Practice of Physic,' Reynolds's 'System of Medicine,' Aitken's 'Science and Practice of Medicine,' Niemeyer's 'Elements of Internal Pathology and Therapeutics,' and Trousseau's 'Clinical Medicine;' and, in special subjects, Duchenne's admirable work on 'Localised Electrisation,' and the no less admirable Lectures by Charcot on the 'Diseases of the Nervous System.'

I must apologise for the many omissions, errors, redundancies, and other faults with which I am only too conscious that my work abounds. Fresh from its completion I feel, perhaps not unnaturally, how much better I could do it were I, from the stand-point of my present experience, now to rewrite it. But this is perhaps a delusion. At any rate I can only take credit for what I have done, and not for what I conceive myself capable of doing. The tree must be judged by its fruits.

In conclusion, I beg leave to record my sincere thanks to my friends Drs. H. DONKIN and GREENFIELD for the kind and valuable assistance I have received from them in the progress of this work through the press. They have each read and criticised nearly every page; and I owe it to them that many mistakes have been corrected, many omissions supplied, and that the reader has been spared the infliction of some grammatical inaccuracies and no little careless spelling.

11 OLD BURLINGTON STREET:
August 1876.

PART I.

GENERAL PATHOLOGY.

GENERAL PATHOLOGY.

I. THE DEFINITION OF DISEASE.

PATHOLOGY, or the physiology of disease, is the science of life under morbid or abnormal conditions. This science, and the arts of applying it in the detection and in the alleviation or cure of disease, and in its prevention, form the subject-matter of works on medicine and surgery.

The question, then, 'What is disease?' naturally arises on the very threshold of a treatise on the practice of medicine. But although doubtless every physician has a notion, sufficiently clear for the practical purposes of his art, of what is implied in the word, the question is one which by no means admits of a ready and explicit answer. Disease, in some at least of its forms, has been regarded by many persons, and is probably still regarded by some, as a real thing or entity. This view implies that it can be either cut out by the anatomist, or extracted by the chemist, or excreted by the patient himself, or in some other way separated from his body, so as to become capable of independent existence and recognition; and might be supported by reference, to the discharge of an intestinal worm or the removal of a vesical calculus, or to a patch of psoriasis, an epitheliomatous tumour, a malformed heart, or probably any other so-called 'local' disease. A little thought, however, will satisfy the mind that the intestinal worm, or the calculus, is of itself the mere cause of disease, and not disease; and that the patch of psoriasis, the epitheliomatous tumour, or the malformed heart, is simply a morbid fragment of the body, and no more the disease itself than the patient who is suffering from scarlet fever or syphilis is the actual embodiment of either of these latter two affections. But, indeed, the opinion that disease is an entity has now been abandoned by all thoughtful physicians. Another view of disease is, that it consists in any deviation from the healthy state, or (at greater length) in any condition of the entire system, or of any part of it, attended with impairment or derangement of structure or function, or both, and tending to render life uneasy, burdensome, or useless, or

to shorten it. It would be difficult perhaps to dispute the accuracy of this definition so far as it goes; at the same time it is obvious that we gain nothing by it unless we have previously agreed upon a definition of health; and in fact, by accepting it, we simply shirk the difficulty which we pretend to solve.

If we consider attentively the various morbid processes and symptoms which separately or in combination indicate the presence of disease, and trace them in each case backwards to their origin, we cannot avoid the conclusion that that origin is some definite or peculiar cause, either innate in the system or acting on it from without, and determining according to its nature and its mode of operation the character and the grouping of the morbid phenomena which ensue—in other words, that the biography of every disease comprises some special cause, and certain resultant phenomena (vital, chemical, or mechanical) which are, or which produce, the symptoms and signs by which we recognise its presence. Let us test the accuracy of this view of disease by a few examples.

A patient is suffering from scabies or tinea tonsurans. In the one case his epidermis is traversed by a lowly form of vegetable growth, and the seats of this growth are indicated by rings of superficial inflammation, by desquamation and the destruction of hair; in the other case, his epidermis is undermined by the burrows of swarming acari, which produce local irritation with intolerable itching, and involve the formation of vesicles and pustules. Now in each of these examples we have an obvious cause, and certain resultant phenomena—the former being the parasite, the latter certain localised inflammatory processes. We have the two factors: namely, the cause and its consequences. We have also the disease. But where is it, and what is it? Is it the parasite, the presence of which is essential in order that the disease shall present its specific characters? Is it the inflammation which the presence of the parasite evokes? The answer to both of these questions must surely be in the negative. The parasite away from the body in which it resides, or apart from the irritation which it causes, is simply a living member of the animal or vegetable kingdom; the local inflammation, dissociated from its specific cause, is inflammation, if you will, but neither scabies nor ring-worm. Obviously then, as applied to such cases as these, the word disease (if it have any real meaning) includes both the special cause of the disease, and the pathological consequences of the operation of that cause.

Again, a person who has never had scarlet fever inhales the particles, or the 'contagium,' which is the specific cause of scarlet fever, and forthwith becomes the subject of that disease. The contagium multiplies within his system, and presently a characteristic rash overspreads his surface; his tonsils and probably his kidneys become inflamed; and, in association with these conditions, there is profound disturbance of his nutritive processes, indicated by heightened tem-

perature, increased formation of urea, and many so-called 'functional derangements.' Now here again we have the cause of the disease, and the various morbid processes which result from its operation. But where is the disease? What is meant by the term 'scarlet fever'? The specific contagious particle of scarlet fever gives scarlet fever, exactly as the acarus scabiei gives itch, or the trichophyton tonsurans gives ringworm: a group of mutually-related phenomena spring up in obedience to their cause as invariably in the former case as in the latter cases. But the contagium of scarlet fever may, as we know, gain an entrance into the living body, and yet be inoperative there; and, on the other hand, several of the more prominent phenomena which form a part of scarlet fever, or symptoms which seem to us identical with the corresponding symptoms of scarlet fever, are occasionally combined in persons who are certainly not suffering from this exanthem. Yet, obviously, in neither of these cases is scarlet fever present. In the former case, the host remains healthy; in the latter case, the disease, though presenting some points of superficial resemblance to scarlet fever, is potentially and essentially distinct from it. Here also, then, it is obvious that, when we speak of the disease, we include in our meaning, not only the symptoms by which we recognise its presence, but the cause upon which those symptoms depend.

Let us take another case. A man is exposed to cold and wet, and shortly afterwards one of his joints becomes swollen and painful; febrile symptoms, attended with abundant sour-smelling perspirations, manifest themselves; presently inflammation attacks other joints: perhaps too the heart becomes implicated. We have here a lot of symptoms which collectively teach us that the patient is suffering from the disease known as 'acute rheumatism.' But what is acute rheumatism? Mere inflammation of a joint, such as that which results from a sprain, does not constitute it; nor even do successive or simultaneous attacks of inflammation of several joints—for if they did, both gout and pyæmia should be embraced within its meaning. Still less are high temperature and profuse perspirations rheumatism; still less acute heart-disease, or any of its various other inflammatory complications. Further, the merely fortuitous concurrence of most, or even all, of the symptoms which have just been enumerated would still not render the case in which they occurred a case of rheumatism. Something more is required for that purpose: a something which shall link all the symptoms together into a common brotherhood, a something which shall constitute their common parentage, a cause from which all shall have directly or indirectly sprung, and which shall have impressed upon them their separate and collective peculiarities. Whether that cause consist in some chemical or other change directly effected in the blood flowing through the part exposed to cold, or in some similar change (induced through the agency

of the sympathetic nerves) in connection with the joints themselves, whether the precise nature of the cause be known or unknown, is immaterial for our argument. In this case, as in the other cases which have been quoted, a cause undoubtedly is or has been in operation; and independently of it the disease 'rheumatism' has no existence.

The relation between cause and effect in disease, and the necessity for not overlooking the cause as an essential part of the disease, are nowhere more obvious than where we have to do with affections in which the cause is tangible, or admits of being weighed, measured, or otherwise tested or examined; as, for example, where mechanical impediments occur in the course of the bowel, urethra, ducts of glands, and other tubular organs; or where poisons received into the stomach act directly upon that viscus, or on distant organs in which they are deposited, or through which they circulate; or where, finally, pathological results follow from excess, deficiency, or unwholesomeness of diet.

Now, in every one of the above examples, it is beyond dispute that neither the collective morbid phenomena or symptoms which indicate the presence of disease, taken by themselves, nor the morbid cause on which these phenomena depend, taken by itself, constitute a disease; that, alone, they are simply factors of disease; and that in each case our conception of a disease is fulfilled only when the cause and its results are, so to speak, welded mentally into one common whole. And hence, if these views be generally true, disease may be defined as *a complex of some deleterious agency acting on the body, and of the phenomena (actual or potential) due to the operation of that agency.*

Regarding it, not as a matter of idle curiosity, but as one of fundamental importance for a clear appreciation of the aims and limits of diagnosis and treatment, that we should have a distinct comprehension of what we mean by disease, we shall pursue the question yet further, mainly with the object of determining how far the word disease is properly applicable (as it often is applied in practice) to mere symptoms or secondary phenomena or incidents of disease.

All diseases involve, some in a greater, some in a lesser degree, certain groups of pathological consequences immediately traceable to their respective morbid causes; but these primary pathological consequences themselves tend to evoke others, these again a tertiary series, and so on continuously. Thus, a person with carcinoma of the bowel may, as a consequence, have stricture, or perforation, or involvement of the glands occupying the retro-peritoneal tissue and gastro-hepatic omentum, or that form of cachexia which cancerous disease so frequently induces: and, as a consequence of these several secondary morbid conditions, various other affections, such as enteritis, peritonitis, jaundice, ascites, melæna, thrombosis or anasarca.

Now all these phenomena, and many others, are obviously integral portions of the carcinomatous affection from which the patient is suffering, and all of them may be regarded as symptoms or incidents of that affection; but many of them are not unfrequently also looked upon as quasi-independent diseases, and treated as such. There is no doubt that they are not diseases. They are clearly, however, elements of disease; and inasmuch as each one of them arises out of some immediately antecedent abnormal condition which is its direct cause, they do obviously enough, in association with their respective causes, fall severally within our definition of disease. Hence the affection which has been selected for illustration, and manifestly also all other primary diseases, may be considered to comprise or involve a number of what, regarded from one point of view, are symptoms or phenomena which are essential parts of it, regarded from another point of view, are component parts or factors of secondary or subordinate diseases, issuing in collateral lines of descent from a common ancestral cause.

II. THE ÆTIOLOGY OF DISEASE.

THE causes of disease have been divided by authors into three classes, namely, the *predisposing*, the *exciting*, and the *proximate*: the first class comprising those conditions which so modify the health of the patient as to render him apt, or predispose him, to contract the disease, to the specific influence of which he happens to be exposed; the second, those causes which immediately impart or excite disease, and give it its specific character; the third, those morbid processes which the action of the exciting cause calls into play, and to which the symptoms of disease are supposed to be directly due. The proximate cause indeed is often, though erroneously, said to be the disease itself. We will illustrate the above distinctions by an example. A woman, who has frequently been exposed to the contagion of scarlet fever without taking the disease, is again exposed at the period of childbirth, and now suffers from a virulent attack. Here, parturition, which renders women peculiarly susceptible of the contagious fevers, is the predisposing cause, the scarlatinal contagium is the exciting cause, and the inflammatory processes going on in the skin, tonsils, and elsewhere, the proximate causes of most of the symptoms which the patient manifests. But the exciting cause of the scarlet fever is obviously the proximate cause of that disease, and the proximate causes of its several secondary phenomena are just as obviously their exciting causes.

The distinction between the exciting cause and the proximate cause is thus purely artificial. That between the predisposing cause

and the exciting cause, on the other hand, is in general well marked; and doubtless if we had an accurate knowledge of the causation of disease, the universality of the truth which underlies these terms would be quite beyond dispute. As it is, however, doubts or difficulties as to their meaning and application are apt to present themselves.

An example will explain our meaning. A man, who has been suffering from privation, is exposed to malarial influence, and contracts ague. In this case, clearly enough, privation is the predisposing cause, malaria the exciting cause. But after a time the ague leaves him, and he is apparently restored to health; and he continues well, until perchance from exposure to the weather in some non-malarious district he catches cold, and straightway experiences another attack of ague. Now which in the latter case should be regarded as the exciting cause? The answer will probably be, 'Exposure to cold and wet,' an answer which necessarily implies that on this occasion malaria is the predisposing cause. Yet, notwithstanding, malaria is equally in both cases the specific cause of the disease, and acts (as we have no reason to doubt) in both cases in a precisely similar manner.

On the whole, however, we mean by exciting cause the specific cause, or element, in disease—that cause (the contagium of an exanthem, the virus of rabies, the parasite of a tinea) which stamps its individuality on the group of morbid processes which ensue, and constitutes with them a definite or specific disease; and by predisposing causes we mean those general, non-specific conditions which by their influence so modify the health of the system, or of parts of it, as to render them (so to speak) a specially suitable soil for the growth of certain diseases, supposing their germs happen to become implanted therein.

A. Predisposing Causes of Disease.

We shall not undertake to discuss the subject of predisposing causes at any length, although it is one of great importance, especially in relation to preventive medicine; but shall content ourselves with enumerating and considering briefly some of the more important and more generally recognised amongst them.

1. *The influence of age* is very remarkable. The period of growth and development, commencing with birth and terminating with the attainment of maturity, and comprising the important physiological epochs of the first dentition, the second dentition and the unfolding of the sexual system, is not only attended with a general aptitude for diseases having a special connection with the physiological processes (general or special) which are going on then, but is liable for less obvious reasons to the attacks of various maladies of other kinds. In

early infancy a remarkable tendency exists to disturbances of the alimentary canal, and to these a very large proportion of infantile mortality is due. Again at this time, and especially during the period of the first dentition, epileptiform convulsions are of peculiar frequency. Rickets is a disease which can manifest itself only during the period of growth of the osseous system, and does in fact occur during the first few years of childhood. It is about this time also that pseudo-hypertrophic paralysis is most commonly met with. True asthma generally comes on in childhood, and not unfrequently disappears before maturity is reached. Chorea affects in large proportion young persons between the ages of 8 or 9 and 15 or 16; and epilepsy, when not immediately traceable to infantile convulsions, commences very frequently about the same time. Acute rheumatism, again, and scrofulous diseases are disproportionately common in young persons. Further, some parasites, such as thread-worms and the trichophyton tonsurans, are peculiarly prone to affect children. Few special liabilities to disease mark the period of maturity, excepting such as are connected with difference of sex, or arise out of habits of life and other circumstances which have only an accidental connection with age. But as the decline of life approaches, and during its continuance, many disorders, and mainly such as are connected with the decay and degeneration of tissues and organs, manifest themselves. Thus the central nervous system becomes affected, and feebleness of mind or fatuity and paralyses supervene; or the heart undergoes morbid changes, and dropsies and hemorrhages result; or the vessels get weakened, and aneurysms and ruptures with extravasations of blood occur; or the stomach, liver, or kidneys suffer, and cease to act efficiently. Gout, too, should probably be included among the proclivities of advancing years.

2. *The differences in the organisation of the sexes* necessitate of course differences as regards some of the diseases to which they are respectively liable. It need scarcely be pointed out that in one sex we meet with disorders connected with the uterus and ovaries, disorders of menstruation, pregnancy, and lactation; in the other sex affections which are peculiar to the male organs of generation. But besides these necessary differences, there are others which are far more difficult to explain and yet are nearly as constant. Thus, chlorosis and hysteria, and nervous disorders related to hysteria, are the almost exclusive heritage of females. And again, certain other affections which occur in both sexes, are yet, for no sufficient reason (so far as we can see) far more frequent in the one than in the other. Thus, erythema nodosum, and exophthalmic goitre, and goitre itself, are all far more common in females than in males. It is possible of course that some of these latter differences may not be due to the influence of sex alone.

3. *Personal peculiarities*, born with the individual, and often hereditary, have an important influence over the relative liability of persons to disease. Children notoriously resemble their parents, not only in the general configuration of the body, but in features, expression, complexion, and mental attributes. Trivial peculiarities in the form of some feature, in the tone of the voice, in the quality of the laugh, small oddities of manner or of gesture, are perpetuated in families. It is not surprising therefore that malformations and other morbid conditions and tendencies to disease should be transmitted also. It is important, however, to note: first, that such inherited peculiarities and tendencies not unfrequently skip a generation, or appear as it were sporadically in families, so that, while out of a family of brothers and sisters some are affected and others escape, the affected and unaffected procreate indifferently healthy and unhealthy offspring; second, that the inherited tendency to disease does not in all cases manifest itself in an exact reproduction of the morbid peculiarity of the parent; and third, that undoubtedly in many cases peculiarities of constitution and special proclivities to disease appear altogether *de novo*.

In some instances the morbid condition is developed, or appears, in fœtal life; in others the child is born healthy, but with a tendency to disease, which becomes realised at some later period. As examples of the former case may be enumerated congenital malformations, idiocy, nævi. Examples of the latter case are more common and far more important for the physician, and therefore need more detailed consideration. Certain functional nervous disorders, such as insanity, epilepsy, hysteria, asthma, neuralgia, undoubtedly run in families, and are apt in some degree to alternate, so that a parent suffering from one of them may beget children in whom one or other of the remaining members of the group replace as it were the particular parental malady. Again, gout, tuberculosis, carcinoma, and other forms of growths, all manifest a tendency to hereditariness. So do many varieties of skin-disease, such as ichthyosis, psoriasis and acne. Degenerative affections, especially those which are characterised by fatty or calcareous changes, also have a tendency to repeat themselves; and thus, in some families the members are apt to be cut off prematurely by extravasations of blood into the brain, due to such degeneration of the cerebral arteries; in others the heart appears to be the selective seat of such changes. Lastly, among inherited or personal peculiarities, we must not forget certain idiosyncrasies characterised by special aptitude to suffer from agencies which are to most persons innocuous, or to remain unaffected by conditions which are generally inimical. The influence of the emanations from fresh hay in producing hay-asthma and of the smell of many flowers in creating nausea, the specially poisonous effects which even the smallest doses of mercury, opium, or other drugs, and which also certain forms of

food (even such wholesome meat as mutton) have upon certain individuals, the unhappy tendency which some persons seem to have to contract all the catching diseases to which they are exposed, and even to take the same one over and over again, and the remarkable way in which other persons seem always to escape, are common examples of the peculiarities referred to.

4. *Occupation, habits of life, quality of food or drink, over-indulgence, privation, and even abstinence,* are all of them potent agents in modifying the constitution and rendering the frame susceptible of disease. We may quote in exemplification of this statement the acquired proclivity of compositors to tubercular phthisis; that of persons who lead sedentary lives to suffer from indigestion and constipation, and the effects of accumulated fat; and that of habitual eaters or drinkers to excess to become gouty, and to suffer from renal and hepatic disorders. It would be easy to multiply examples of the influence of these and like causes in the production of disease, and especially to adduce illustrations which might appear far more striking than any of the few given above—such, for example, as the occurrence in miners and others of special forms of lung-disease, in painters of dropped hand, in drinkers of cirrhosis of the liver, delirium tremens, and so on. But it is obvious that we have here examples, not of any mere predisposition which has been gradually acquired, but of the direct and specific influence of certain exciting causes to which the sufferers have been exposed.

5. *The effects of previous disease* in modifying the tendency to subsequent attacks of disease are in many cases very remarkable. In the exanthemata and allied affections an attack of any one is in a very high degree protective against subsequent attacks of the same malady. On the other hand, many inflammations tend to repeat themselves. Thus, persons who have once had erysipelas of the face generally acquire a liability to attacks of the same malady throughout the remainder of their lives. So it is with rheumatism, pneumonia, bronchitis, tonsillitis, catarrh, renal inflammation, and intermittent hæmaturia. And, indeed, one of the most difficult practical problems with which physicians have to deal is that of the counteraction of such acquired tendencies. But there are many disorders which engender a liability, not to their own recurrence, but to the attacks of other diseases. Thus both scarlet fever and gonorrhœa are curiously apt to be succeeded by attacks of acute rheumatism. Tuberculosis is generally believed frequently to follow on enteric fever and small-pox, and scrofulous enlargement of the cervical glands on mumps. And chorea may certainly be regarded as a sequela of both acute rheumatism and scarlet fever. To these latter examples may be added the fact, which seems beyond dispute, that organs and parts which have been the seats of repeated

or continuous attacks of inflammation, and have in consequence undergone structural changes, and so also pigmentary nævi, often prove the selective sites for the primary development of sarcomatous and other kinds of malignant growths.

It may be convenient to refer here to the special morbid predispositions of different organs and tissues. A very little acquaintance with pathology is sufficient to prove that the different parts of the system are not all equally liable, or liable in proportion to their respective bulks, vascular supply, or importance, to the same forms of disease. Thus, each one of the specific infectious fevers involves in its progress certain organs, altogether disproportionately to other organs, if not to the entire exclusion of some; parasites, whether vegetable or animal, limit their attacks more or less exclusively to certain parts, such as the skin, muscular system, liver, or intestinal canal; the inflammations of rheumatism and of gout are specially wont to seize on the ligaments and other soft parts about joints; and tumours, according to their characters, are prone to originate in different tissues,—tubercle, which is so wide in its distribution, rarely, if ever, appearing in the skin, connective tissue, or muscles; and carcinoma, which is even less exclusive than tubercle in its choice of locality, yet preferring for its primary manifestation certain organs, such as the uterus, the mamma, and particular regions of the alimentary canal.

6. *The influence of heat and cold, of dryness and moisture, and of atmospheric impurity* in predisposing to disease is universally admitted. But here, as in some of the cases previously referred to, we are apt to confound, and it is difficult to avoid confounding, their indirect effects as predisposing agents with their direct effects as exciting causes. And further, when we come to test the relative influences of climates and seasons by their prevalent diseases, our endeavours to arrive at a just conclusion on the subject are seriously hampered by the co-existence with them (but partly no doubt arising out of them) of peculiarities of habit and modes of life, and of malaria or other special conditions of unhealthiness. Thus we shall all acknowledge the influence of temperature in the production of bronchitis, pneumonia, rheumatism, and sunstroke; but in these cases temperature no doubt acts as the exciting cause. Again, we shall all be ready to allow that remittent fever, hepatitis, and dysentery are characteristic diseases of tropical climates; but for the first malaria, not temperature, is wholly responsible, and the latter two are possibly in some cases also of malarious origin. Further, we all know, by personal experience, the ill effects of overcrowded close rooms; and we cannot doubt that deterioration of health must result from that constant breathing of vitiated air to which the children of the urban poor are generally condemned, and we shall possibly rightly attribute much of their early sickliness and prematurity of death directly or indirectly to this cause; but it is

certainly difficult accurately to identify either the morbid state which it produces directly, or the special predispositions to disease which it engenders.

It is of course beyond dispute that certain diseases prevail exclusively or with special severity in certain climates, and that their prevalence varies with season, and also with local telluric or hygienic conditions. Thus, yellow fever occurs in the West Indies and on the West Coast of Africa and some few other localities; dysentery and hepatic abscess are in a peculiar degree diseases of tropical India; Asiatic cholera, dengue, plague, all originate, and prevail chiefly or exclusively, in hot climates; tubercular phthisis is one of the especial scourges of the temperate zone. Again, at least in our own country, thoracic inflammations are most frequent during the cold seasons of the year—acute pneumonia being probably most common in the early spring; diarrhœal affections prevail in summer; and many other diseases have a tendency, difficult to explain, either to undergo exacerbation or to break out, or it may be to subside, at characteristic times—thus ague appears chiefly in spring and autumn, and psoriasis and some other forms of skin-disease have a similar tendency.

It should be added that (fortunately for us) the human frame is adapted to live healthily under great varieties of climate, and under great extremes of heat and cold; and that the effects of climate in the production of disease are probably less due to simple cold or heat, dryness or moisture, than to the neglect, on change of climate, to adapt our habits of life to the altered circumstances in which we find ourselves, and to the effects of sudden and unprepared-for variations of temperature.

7. In close connection with the subject under consideration is the question of variation in the so-called 'epidemic constitution' of different years, and in the 'type of disease.'

By the term '*epidemic constitution*,' Sydenham, who first employed it, meant a peculiar state of the atmosphere, determined by special telluric conditions; to which, as specific causes, he attributed the development of epidemic diseases, such as small-pox, scarlet fever, measles and plague; and by variations in which he explained the epidemic prevalence of one or other of these diseases, and a tendency (which he believed to exist) for all indifferent diseases occurring during such an epidemic to be modified under its influence and to assume some of its characteristics. The advance of pathological knowledge since his day has proved that most, if not all, epidemic disorders spread by contagion, and that there is no atmospheric or telluric influence to which they are due, nor anything beyond actual contagion which can give, during the presence of Asiatic cholera, or of small-pox and the like, any of the special attributes of these diseases to other prevalent diseases. Nevertheless, it must be admitted that there is something

remarkable, and indeed something inexplicable, in the way in which diseases—not contagious and miasmatic only, but simply inflammatory also—become at irregularly recurring intervals prevalent in a high degree over wide areas. In this qualified sense the expression, 'epidemic constitution,' is still not unfrequently, and may on the whole be conveniently, employed.

By the term '*change of type in disease*' is understood, not the transformation of one epidemic disease by gradual steps into another disease—a process in which few now believe; but a change in the quality of diseases, in virtue of which they present cycles of greater and lesser intensity of attack and of other deviations from the normal standard. Such changes are believed to depend partly on variations referrible to the disease itself, partly on 'epidemic constitution,' partly on cyclical changes in the constitution of mankind. There can be no doubt that differences of severity and fatality do not unfrequently characterise different epidemics of the same disease; and further, it is beyond dispute that, even during the same epidemic, some persons are attacked with much greater or much less severity than others, or have the disease in a more or less modified form; and in these senses the fact of variation in the type of disease must be fully admitted. There are many, however, who still believe that all diseases have undergone a change of type during the last fifty years; that they were formerly sthenic, and were to be cured by blood-letting, whereas they have now become asthenic and demand an exactly opposite line of treatment. It would be strange if, while the old descriptions of diseases remain accurately applicable, as in fact they do, to those of the present day, and while the health of the population has been undergoing gradual improvement, as it has done (if, at least, we may judge by the diminishing death-rates and the improved circumstances of the people), the effects of these unchanged diseases on the improved constitutions should be to render these latter more helpless during their attacks, and more likely to succumb from sheer debility. Many will be disposed to admit that the change of type has been rather in the medical practitioner than in the disease or in the bodily constitution, and that the gradual change of treatment has been due, either to the slow advance of knowledge with respect to the effects of remedies in disease, or to fashion.

B. Exciting Causes of Disease.

Amongst the predisposing causes of disease just passed in review are some which act at least as efficiently in the direct production of disease. We refer especially to those discussed in paragraphs 4 and 6. It is certain that to variations of temperature, combined with changes of hygrometric condition of the atmosphere, a very large proportion of local inflammations is immediately due. As examples may be cited

common catarrh, bronchitis, pneumonia, pleurisy, nephritis, rheumatism, inflammation of the portio dura causing facial palsy, erysipelas, and various affections of the skin. Again, over-indulgence in food, even though the food partaken of be fairly wholesome, not only causes sickness and diarrhœa or other forms of gastro-intestinal disturbance, but leads ultimately to accumulation of fat, plethora, indigestion, gout, and various disorders arising out of these. So, on the other hand, deficiency of sustenance, or deficiency of essential ingredients of that sustenance, induces emaciation, anæmia, debility, degeneration, and various special disorders, the direct production of some of which has been demonstrated by experiment on the lower animals, and of which scurvy affords a notable example. Not far removed from such causes as these are the over-exercise or under-exercise, or abuse of the system, or of component parts of it. We need only refer, in proof of their efficacy, to the serious consequences which are apt to ensue on sudden and very violent muscular efforts, or on long-continued over-exertion of the muscular system, to the many injurious effects of sexual excesses, which are not entirely due to seminal losses, and to the many nervous disorders which originate in overwork of the brain, in prolonged wakefulness, in the unconstrained indulgence of the passions, and the like.

Without meaning thereby to exclude the various causes which have just been enumerated from classification among them, we may, with tolerable accuracy, group the remaining specific causes of disease under the heads of 'mechanical,' 'chemical,' and 'vital'; and we may further divide them into the endopathic, or those which originate within the system on which they act, and the exopathic, or those which attack the system from without.

1. *Mechanical Causes.* *Exopathic* mechanical causes embrace all forms of external violence, the results of which fall more particularly within the province of the surgeon. *Endopathic* mechanical causes, on the other hand, are of special importance and interest to the physician. They include mechanical obstructions of orifices or tubes, whether these obstructions be caused by thickening and contraction of their walls, by pressure on them from without, or by impacted concretions: such are intestinal stricture, hernia, intussusception, and the lodgment of gall-stones, and all similar obstructions in the ducts of the liver and pancreas, in the various urinary passages, in the larynx, trachea and bronchial tubes, at the cardiac orifices, and in blood-vessels. They also include impediments, however originating, to the transmission of nerve-currents along the nerves, dilatations of arteries and of other tubes and cavities, perforations or ruptures of their parietes, and extravasations or effusions of blood, serum and other matters. It is obvious, therefore, that agencies of this kind are the direct causes of a very large proportion of the local diseases to which we are liable. But it may be observed that they would probably all have been con-

sidered by the older writers as proximate rather than exciting causes of disease, and that they are in fact in no case the primary causes of the morbid processes from which patients suffer. Thus, the person, who suffers and dies from stricture of the œsophagus or bowel, and whose grave symptoms have all been referrible to the stricture, owes his stricture to previous local inflammatory thickening, or ulceration, or carcinoma; and he who dies from the consequences of mechanical impediment to the passage of blood through the mitral orifice, traces the affection of the mitral valve to a long antecedent attack of rheumatic fever.

2. *Chemical causes of disease* include all poisonous substances, whether they be derived from the inorganic or the organic kingdom, and however variously they exert their influence over the system. The great majority of these are necessarily *exopathic*. Some, like the caustic alkalies and mineral acids, destroy the surface to which they are applied; others, like opium, strychnia, aconite, and snake-poisons, undergo absorption, and quickly exert their chief influence on particular organs, or on the general system; while others, again, introduced into the organism habitually and in minute quantities, slowly induce characteristic organic and other changes, and thus what are commonly regarded as definite diseases. Thus, dropped hand and colic, or plumbism, are the results of chronic lead-poisoning; muscular tremors indicate mercurialism, or the ultimate effect of the inhalation of mercurial vapours; the fumes of phosphorus after a time cause necrosis of the jaws; the habitual use of ergotised cereals for food is believed to bring about a peculiar form of gangrene of the lower extremities; and not improbably endemic goitre and cretinism are due to the constant, slow action of some material agent. We must obviously also include here the poisonous effects of certain articles of food—mussels, fungi, sausages, and the like—and those which flow from the habitual use of alcohol, tobacco, and opium.

Endopathic chemical causes are principally such as depend on defective action of the excretory organs, and the consequent retention in the system of effete matters which then act as poisons. The chief emunctories for the purification of the blood are the kidneys, liver, lungs, and skin. If the kidneys act inefficiently, urea and other excretory constituents of the urine accumulate in the blood, and by their presence there at length induce epileptiform convulsions, dropsy, anæmia, and other symptoms which collectively indicate the presence of Bright's disease. If the liver fail to discharge its normal functions, jaundice follows, and with that, and in some degree in consequence of it, many other grave symptoms. When from mechanical or other impediment to respiration, the blood becomes overcharged with carbonic acid, lividity of surface, delirium, and coma presently supervene. The cutaneous exhalation is for the most part merely complementary

to that of the lungs and kidneys; and hence the injurious effects of its arrest are not very apparent; at the same time, doubtless, serious consequences are often correctly attributed to its suppression. Here we may refer, also, to the ill effects of that accumulation in the blood of the various ill-defined products of decomposition, which attends the specific febrile disorders, and in a greater or less degree most diseases or pathological processes.

3. *Vital Causes.* We now come to speak of that important class or causes to which all contagious or infectious diseases owe their origin—causes, which are specific for each specific disease; which are material; which pass in some way or other from those already affected to those who are sound, and implant themselves in their bodies; which grow and multiply therein at their expense, causing characteristic symptoms; which in a greater or less degree are capable of escaping therefrom, and of then similarly infecting a second series of healthy persons, and so on continually; and of which none (so far as we certainly know) has varied intrinsically in its effects from the earliest record of its operation up to the present time, or upon any part of the earth's surface. It is at once obvious that these causes are essentially and utterly different from those mechanical and chemical causes which have just been discussed. It is impossible to conceive of the contagiousness of a strictured bowel, an apoplectic clot, or an attack of jaundice; it is contrary to all we know of chemistry, that lead or mercury, morphia or the poison of the cobra, or a dose of medicine, should multiply within the system. But here we have poisons or irritants which do multiply in the system, it may be a billion-fold, every unit of whose product is as efficient in imparting disease as was the unit from which it sprung. These facts seem quite incompatible with any other view of the nature of these causes than that they are actual living things.

That some of them are living is absolutely certain; we mean *parasitic animals and vegetables.* Of animal parasites, some live and swarm on the skin, or in it, and readily transfer themselves from one body to another; some live in the alimentary canal or in the solid organs, and these, though still capable of infecting other healthy persons, infect them indirectly only, and after undergoing remarkable transformations external to the body of their host, and often in the organism of some lower animal. Superficial diseases due to the presence of vegetable parasites also are highly contagious.

With regard to the *contagia*, properly so called, namely, the infectious matters to which the several exanthematous and other similarly infectious fevers are due, there is far less direct evidence in favour of their being living things. Nevertheless some such evidence, to the effect that they consist in marvellously minute particles of

living matter or protoplasm, has been adduced, and will at a subsequent page be more fully considered.

The poison or *malaria* on which ague and remittent fever depend, although not communicable from man to man, has a certain resemblance to the contagia, both in its mode of infecting the system, and in the effects which mark its operation there, and hence not improbably is of a like nature with them.

It seems convenient to advert here to the fact that many inflammations, originating apparently in indifferent causes, either are inherently infectious or acquire under particular circumstances an infective character, and that they spread, like the diseases which have just been considered, in some cases by direct contact or inoculation, in others, by atmospheric carriage. Thus, most practical medical men will readily admit the communicability of common catarrh and of tonsillitis, the contagiousness under special conditions of even idiopathic erysipelas, and the readiness with which catarrhal ophthalmia and impetigo occasionally spread. Gonorrhœa furnishes a yet more striking example of the same fact. These cases are important, because they seem to show the possibility of the spontaneous development of contagious elements within the system. It is probable that here the contagious property resides in the pus- or exudation-corpuscles whose development attends the inflammatory process. It must be added, however,—that a very large number of diseases, fundamentally distinct from one another, are yet linked together by the common bond of the occurrence in them of inflammation as a more or less prominent feature: that one tendency of advancing pathological knowledge is to recognise that, in a larger and larger number of so-called 'inflammations,' the inflammation is not the essential element in the disease, but merely one out of a group of several morbid phenomena, all starting from the direct influence of some specific cause: and that hence it may, perhaps, eventually be discovered, that all of these catching inflammations are, in the same sense as scarlet fever or mumps, specific diseases dependent on specific causes.

The causes of carcinoma and other varieties of malignant disease, and indeed of proliferating tumours generally, are very obscure. It is not difficult to understand, that when once a tumour, destined to be malignant, has made its appearance in any part, the subsequent development of secondary tumours in the neighbouring lymphatic glands, and in remote organs, may be due to the conveyance thither from the primary growth of prolific particles of its specific protoplasm; and that, hence, the diffusion of such tumours throughout the organism may, like the diffusion of small-pox throughout a population, be due to a contagium—but to a contagium, in this case (as probably also in certain inflammations), originating in the living tissues. But this explanation throws no light on the primary causation of such growths, and of their specific distinctions from one another. They seem, at

any rate frequently, to be induced by the long-continued local operation of non-specific causes of irritation; and their specific characters, which are perhaps less absolute than they seem to be, may depend in some degree on the nature of the tissue which becomes irritated into overgrowth.

III. PHYSIOLOGICAL PROCESSES IN HEALTH.

The processes of disease, however widely they may seem to diverge from those of health, are merely modifications of them, and their types must be sought in the normal physiological processes by which the body is developed, grows, maintains itself, and finally dies. It will be well, therefore, before considering them in detail, to pass briefly in review the physiological processes out of which they arise.

A. It is now admitted by physiologists, with almost perfect unanimity, that the first origin of every living thing, as also every living particle of the developed organism, is a viscid, homogeneous, colourless, albuminous substance, known as protoplasm or germinal matter; and that this is endowed with remarkable powers, in virtue of which, under appropriate conditions of warmth, moisture and the like, it is capable—first, of throwing out processes or otherwise altering its form, and thus, on the one hand, of investing and absorbing solid particles, and, on the other hand, of actual locomotion: second, of growing, and maintaining itself, by imbibing and appropriating the nutritious matters which surround it, while discharging whatever is superfluous or excrementitious or effete: third, of multiplying by fission or by gemmation: and last, (in dependence on its immediate parentage and other conditions) of undergoing further development or differentiation, so as to take part in the formation of organs, or itself to become an organ performing special functions.

Quiescent protoplasm generally occurs in the form of small round or oval masses, often presenting an imbedded nucleus, or several such bodies, and under many circumstances a thin membranous investment, and hence that combination of characters which we recognise in the typical nucleated cell. The earliest stages in the development of the embryo, and the earliest stages in the development of organs, are characterised by the abundant formation of cells of this kind (without, however, the investing membrane), which are hence termed embryonic cells. These bodies stand, therefore, at the bottom of all growth and all development; and it is by their multiplication and by the changes which they effect, or undergo, that the complex organism of the body is gradually evolved and finally perfected. Thus, in the area germinativa the embryonic cells arrange themselves in three layers—the uppermost or serous, the undermost or mucous, and an intermediate

layer; and, by a process of development or differentiation, from the cells of the uppermost layer are gradually produced the central nervous system and the epidermis with its appendages: from those of the lowest layer, the epithelial lining of the alimentary canal and of the various glandular organs which communicate with it: and from those of the intermediate layer, the vascular system with the ductless glands, and the muscular, osseous, and connective tissues.

B. The result of the processes here adverted to is the formation of a series of simple tissues, which group themselves here and there into complex specialised masses, named organs. These tissues may be arranged, according to Virchow, in three categories, the 'epithelial,' the 'connective,' and those of a higher grade.

1. The tissues belonging to the *first category—the epithelial*—are evolved mainly from the serous and mucous embryonic layers, and comprise—the epidermis, with the hair, nails, and sebaceous and sudoriparous glands; the epithelial lining of the gastro-intestinal mucous membrane, with that of the hepatic ducts and other glandular organs connected with that membrane; the genito-urinary and pulmonary epithelia; and the endothelia, of the serous and synovial cavities of the body, of the blood-vessels and lymphatics. In all these cases, or in nearly all of them, the tissue is composed of typical nucleated cells—that is, of masses of protoplasm containing nuclei and invested in membrane—so arranged as to be in exact contact with one another. Minor differences, yet of great practical importance, are observed between the cells of different epithelia; thus, they vary largely in size and form, and in the thickness and other special characters of their membranous investment. In the case of the outer layers of the epidermis and hairs, nuclei and protoplasm wholly disappear, and each cell becomes a mere lifeless horny flake. The functions of epithelia are very various:—some, as those of the skin and blood-vessels, are merely protective; others, such as that of the mucous surface of the alimentary canal, absorb; while those of glandular organs either manufacture and secrete products serviceable to the economy, or separate from the blood, and excrete, matters which are effete or injurious.

2. The tissues of the *second category—the connective*—are developed almost exclusively from the intermediate embryonic layer, and pervade all parts of the body, with the exception of the epithelia, forming a kind of network, in the interstices of which the higher tissues and the elements of organs are contained. They consist of nucleated masses of protoplasm, which are often exceedingly minute, always surrounded by a wall of greater or less thickness, and either rounded and isolated from one another, or stellate and furnished with processes communicating with those of neighbouring cells. The essential morphological distinction between epithelium and connective tissue is, that in the

former the cells are in absolute contact, in the latter they are separated from one another in a greater or less degree by some intervening substance—either an unorganised or lifeless deposit, or portions of the higher living tissues. According to the nature and amount of this intervening substance, or to peculiarities presented by the cells, connective tissues may be divided into several varieties. In ordinary connective tissue, as also in fasciæ and tendons, the protoplasm is scanty and stellate; and the intervals, which are large, are occupied by wavy bands of white fibrous tissue and more or less yellow elastic fibre, both of which are either simple secretions from the living protoplasmic masses, or the mummies of defunct cells. This variety of connective tissue yields gelatine. In common cartilage, the cells are round or oval, and separated from one another by a dense homogeneous elastic substance, which appears to be formed by the progressive thickening of the cell-walls and by their coalescence, and yields chondrine. In bone, the lacunæ and canaliculi mark the position of the cells and their radiating processes, the proper constituents of the bone occupying the spaces which these include. The central nervous organs and the lymphatic glands possess a peculiar form of connective tissue, termed 'retiform,' in which the essential elements of these organs represent the separating material, and in which the proper cellular elements of the connective tissue are minute and stellate, and the rays passing between them are delicate and homogeneous, and enclose exceeding small spaces. Mucous connective tissue, which is abundant in the developing fœtus, is represented at birth by the tissue of the umbilical cord, and throughout the remainder of life only by the vitreous humour of the eye. In this the intermediate substance is fluid—mucus in fact—and contains mucine. Lastly, passing by some unimportant modifications of connective tissue, it may be pointed out that, in the choroid, spinal pia mater, and elsewhere, the proper cells of this tissue contain pigment, and that in many regions they are distended with oil. In the former case, we have pigmental tissue; in the latter, fat.

It is upon the essential elements of the connective tissue—namely the protoplasmic particles, or cells, and the processes springing from them, which, with certain modifications of character, are distributed nearly universally throughout the organism—that, according to Virchow, the action, growth, and maintenance of the organism immediately depend; and just as (to take bone for an illustration) we find certain districts or territories (the Haversian systems) under the nutritive governance of particular blood-vessels, so we find still smaller territories within them (the lacunar systems) over the welfare of each of which a single cell appears to preside. The latter are termed by Virchow 'cell-districts.'

3. The *third category* of tissues comprises mostly those which are tubular, and formed either by the juxtaposition and coalescence of

cells, or of cells, or protoplasm, which have in some other manner undergone a high degree of specialisation. Among them we may name nerve-cells, and nerves, striped and unstriped muscular fibres, capillary vessels, and lymphatics.

4. Lastly, *complex organs*, such as muscles, bones, glands, brain, and the like, are formed by the association, in various degrees of complexity, of several of the above-enumerated tissues.

Thus, the organism may be regarded as a combination of vital and non-vital elements:—the latter comprising various more or less complex chemical compounds, which have been prepared and deposited through the agency of the living matter, and whose subsequent changes and duration are regulated by the action of the living elements which are in their immediate vicinity; the vital elements being the protoplasmic masses or nucleated cells, which, thickly disseminated, carry on between them all the living functions, and form—the universal network of connective-tissue corpuscles: those laminated aggregations which constitute the various epithelia, and endothelia, and the walls of capillary vessels and lymphatics: the massive accumulations which are observed in the central nervous organs, liver, lymphatics, and other glands; probably striped muscular fibre, and the axis-cylinders and peripheral ends of nerves: and lastly, the corpuscles which are free in the circulating fluids.

It is important to note that the vital properties of protoplasm differ in degree, and in quality, according to its age and the functions to which, by process of development, it has become subservient. Thus, embryonic protoplasm, and its nearest representatives in the mature organism—namely leucocytes and connective-tissue corpuscles—especially possess the power of multiplication and of differential development; whereas muscular fibres and nerve-cells, which stand at the opposite extremity of the scale, probably never, at any rate in health, undergo proliferation or development except in their own special groove.

C. The development, growth, and maintenance, therefore, of the entire organism depend essentially on the healthy circumstances, as to nutrition and the like, of the protoplasmic elements which constitute its living parts. All actively living matter is unstable and short-lived, and needs for the due performance of its vital acts (which are always attended with a certain amount of waste of tissue) suitable food, which it can imbibe and transmute into its own substance, so as at least to supply the place of that which was lost. But it needs, also, the removal of the spent nutritious fluids in which it is bathed, and of those effete and excrementitious matters which it continually emits.

1. For the purpose of providing a constant supply of nutriment, we have the blood, impelled by the heart, slowly coursing through the capillary blood-vessels, and ever sweating all save its morphological elements through their delicate parietes into the extravascular

tissues around, and occasionally perhaps exuding these morphological elements also; and for the purpose of maintaining a constant removal of the spent pabulum, and of effete matters, we have the extravascular fluids ever undergoing absorption, partly by the agency of the venous radicles, but mainly by the lymphatic vessels, which have their origin in the meshes of the capillary network, and in the very spaces in which the protoplasmic elements themselves are situated.

2. The nutritious matters of the blood are supplied to it primarily from the alimentary canal. Food, after having been triturated and swallowed and acted on by the secretions of the various glandular organs which discharge their contents into the stomach and bowels, is absorbed at the surface of the mucous membrane—the fluid and more readily diffusible parts by the capillary blood-vessels, the fatty and albuminous matters by the lymphatics. Those substances which enter by the former route, after passing through the liver and perhaps undergoing some change there, mingle with the general mass of the blood; those which enter by the lymphatics first traverse the lymphatic glands, carrying thence with them the white corpuscles which these glands manufacture, and then like the former blend with the circulating fluid. But the surplus nutriment, which escapes from the capillary vessels into the tissues external to them, is also taken up mainly by lymphatic vessels; and this again, after passing through lymphatic glands, and deriving thence morphological elements, mingles, like that derived from the alimentary canal, with the blood-stream. Lastly the important secretions furnished by the mucous membrane of the alimentary canal, and by the viscera which discharge into it, are reabsorbed in large proportion with the food, and thus re-enter the circulation.

3. Effete matters derived from the waste of the organism are dissolved in the fluids which are also the carriers of nutritious matter; and hence are removed from the parts in which they are produced by the same channels, namely the veins and the lymphatics; and then mingling with the blood are there further reduced by the reducing agency of the oxygen, which it is the function of the lungs to furnish to the blood. Thus, they get converted into diffusible compounds of comparatively simple constitution, which are then separated from the blood by appropriate emunctories—carbonic acid by the lungs, nitrogenous compounds and salts by the urine and by the skin, and the colouring matter of the blood by the kidneys and the liver.

4. Presiding over the processes of nutrition, and to a great extent regulating them, yet itself entirely dependent upon them for the means of its material and functional activity, is the nervous system, comprising the central organs, the nerves, and the end-organs of the nerves. By means of the nerves every part of the organism, probably almost every protoplasmic mass, is brought directly or indirectly, through the intervention of ganglia or of the central organs, into

relation with the other elementary parts of the organism. Sensations or impressions received at the peripheral terminations of afferent nerves are conveyed instantaneously either to some nerve-ganglion, or to the spinal cord, or to the brain, or to all of them; and then, reflected thence along the efferent nerves, certain responsive influences are transmitted which, according to their destinations, result in muscular movement or in glandular action. Thus, the central organs are kept informed of what is going on throughout the organism; and thus (to omit all reference to their influence over the voluntary muscles),—by acting on the walls of the heart and blood-vessels, they regulate the supply of blood to parts, and so control their nutrition and the activity of their special functions: by acting on the walls of gland-ducts, they modify the rate of escape of the products of the glands: and by means of the trophic nerves (which many physiologists now believe to exist), they probably exert a direct influence over the action of the essential elements of secreting organs.

D. Ere we bring these preliminary physiological remarks to a conclusion, a more direct reference must be made than has hitherto been done to the fact that decay and death are essential elements in the normal processes of life. It has already been pointed out that every act of life is attended with waste of tissue, and that living protoplasm is essentially unstable and short-lived. It must be added, that every part of the organism has a limited duration, which is far shorter than that of the normal duration of the body which it contributes to form, and that the parts are removed either by slow disintegration and degeneration or are cast off in mass. We need only advert, in exemplification, to the shedding of the epidermis and of the elements of excretory glands, to the removal and re-formation of bone-tissue, to the generation and destruction of blood-corpuscles, to the atrophy of the uterus and the fatty degeneration of its muscular elements after parturition, and to the even more complete destruction by similar processes of the Wolffian bodies during fœtal life, and of the thymus gland during the first few years of extra-uterine existence. Lastly, it must never be forgotten, that atrophy and degeneration of organs and tissues are normal physiological processes of old age, and that somatic death, in which they culminate, is their normal termination.

IV. PHYSIOLOGICAL PROCESSES IN DISEASE.

If we carefully consider the intimate processes of disease, we cannot fail to recognise the fact, that they consist essentially in nutritive modifications of the protoplasmic or vital elements of the tissues—that under the influence of abnormal or unwonted stimuli (including the stimulus of excessive nourishment), these enlarge, or multiply, or

differentiate; that when insufficiently stimulated or fed, they undergo atrophy or degeneration, or perish; and that, as a necessary consequence of such changes, their functional attributes become heightened, or impaired, or more or less profoundly modified. Thus, on the one hand, we get simple hypertrophy, inflammation, or heterologous growth, and, on the other hand, fatty or calcareous conversion, or other forms of degeneration; and, again, functional derangements too numerous to mention, which constitute so large a proportion of the symptoms of disease.

But when we look to the marvellous complexity of the organism, to the intimate anatomical relations which subsist between the vascular and the nervous and other subordinate systems and organs, and to the correlation and mutual dependence of the various functions which all these different component parts of the organism are called upon to perform; and consider that the healthy structure and function of each is involved in a greater or less degree in the similar integrity of every other; we must admit (what the slightest practical experience will confirm), that we cannot limit our view of morbid processes to these intimate changes alone, but must embrace within it the structural and other modifications of organs to which such changes give rise, as well as those further nutritive and functional disturbances which, in a variety of ways (mechanical, chemical, and other), disease of one part necessarily evokes in a greater or less degree in all other parts of the system. We proceed to discuss at length the several matters here adverted to.

A. Morbid Growth.

1. *General Observations.*

a. Growth and development of cells.—Whenever the protoplasmic particles or cell-elements of a part are stimulated to unwonted growth, they first increase in bulk, and become turbid, or minutely and indistinctly granular, and if stellate, fusiform or caudate, at the same time retract their processes, and assume a more uniformly rounded shape; and then, by internal gemmation or fission, each cell gives origin to two or more smaller cells, which in their turn repeat more or less accurately the processes of growth and proliferation. The results of such stimulation, so far as regards the cells themselves, are, that sometimes the newly-generated cells acquire in all respects the same characters as had formerly belonged to their immediate ancestors, that sometimes they retain permanently the immature or embryonic condition which represents the early or indifferent stage of nearly all cell-growth, and that sometimes again they undergo development into cellular bodies which differ materially in size, form, and attributes, from those which gave them origin. Simple hypertrophy or hyperplasia furnishes an example of the first of these alternatives, inflammatory cell-production of the second, and heterologous tumours of the last.

b. Conditions associated with over-growth.—But where there is exaggeration of cell-growth, there necessarily is also at least proportionate exaggeration of the various conditions which are subsidiary to such growth—namely, exaggerated afflux of blood, exaggerated accumulation of nutrient fluid, exaggerated molecular destruction, and exaggerated efflux of superabundant and effete materials.

Increased afflux of blood is determined mainly by reflex dilatation of the arteries, capillaries, and veins, which minister to the needs of the affected part, and in a subordinate degree by increased force and frequency of the heart's contractions, and produces one form of what is known as 'congestion.'

Increased accumulation of nutrient fluid in the extravascular tissues is due to the preternaturally abundant escape of it from the dilated capillaries—an escape doubtless dependent in some degree on the vital influence exerted by the protoplasm of the capillary-walls, and by the over-growing protoplasm external to them. The tissues consequently get swollen, soft and juicy, and in a greater or less degree 'dropsical.'

All vital activity, whether this manifests itself by material changes or by functional excitement, is attended with molecular disintegration, which has some exact quantitative relation with it; and hence increased vehemence of growth, and of reproduction, is necessarily accompanied with a proportionately increased production of effete and excrementitious matters. But, in addition, undue rapidity of cell-growth and development always involves a corresponding tendency to fall into premature decay and dissolution; and hence arise fatty and other forms of degeneration, the products of which accumulate, and mingle with those of molecular disintegration. It is thus that the fluids of the affected region tend to become surcharged with innutritious, waste, and often noxious materials.

The increased absorption which takes place is probably dependent, in some measure, on the more active passage of fluid by endosmosis through the walls of the venous radicles, but is certainly due mainly to the more direct action of the lymphatic vessels. Indeed, it is almost impossible to suppose that those slightly diffusible substances, albumen and fibrinogen, should, in the face of the opposing pressure from within the blood-vessels, be capable of re-entering them, or that solid particles, whether indifferent or specialised, should be removable by any other route than that furnished by the open mouths of the lymphatics. And that these really are the main agents in the removal of probably everything, save a variable proportion of water and dissolved salts, is shown by the tendency which, when largely overworked, they and the glands in their course have to become enlarged and presently inflamed, or involved in the identical processes going on at the seat of absorption.

c. Migration of leucocytes.—One of the most interesting phenomena,

connected with the subject of local proliferation, is the fact, stated many years ago by Dr. Addison, and since then clearly established by the experiments of Cohnheim and the later observations of many other physiologists, that in artificially produced irritation or inflammation of the tissues of the frog, after retardation of the current of blood in the vessels of the part has taken place, the white corpuscles gradually penetrate the vascular walls, and presently pass completely through into the tissues external to them. It has further been shown that these emigrant corpuscles take an active personal part in the proliferation which ensues; that is to say, that they then, as well as the proper protoplasmic masses of the part, give origin by gemmation or fission to new generations of cells. How far this process contributes to inflammatory proliferation in warm-blooded animals, or may be regarded as an essential element in the development of non-inflammatory growths, is at present in great measure a matter of inference. Still there are many good grounds for regarding it as an important item in all cases of abnormal cell-proliferation. And it is far from unlikely that it may be equally importantly concerned in the normal processes of growth and development.

d. Tendency of morbid growth to spread locally.—Morbid cell-development, occurring primarily at any one spot, generally has a tendency to spread in the neighbourhood of that spot. The direction of local spread is in most cases largely determined by the structure and connections of the tissue or organ in which the growth has originated. Thus, growths beginning in the cutis or mucous membrane are prone to limit their extension to these structures: and the same rule applies to the kidney, ovary, and other organs. Nevertheless, in many cases the morbid process tends gradually to involve all adjoining parts. This local spread is sometimes effected by the progressive involvement of the healthy tissues immediately surrounding the focus of disease; and very often partly by this process, but partly also by the appearance of new foci of disease in the vicinity of the primary focus, and by their gradual coalescence with it and with one another. It is sometimes determined by the lines of capillary lymphatics and blood-vessels.

e. Tendency of morbid growth to become generalised.—The tendency to the simultaneous or consecutive occurrence of the same kind of morbid proliferation in different, and even remote, parts of the organism is traceable to a variety of causes, presents obvious and characteristic differences, and has therefore a widely different significance in different cases. The matter is one which deserves, and indeed demands, consideration; and we proceed, therefore, to discuss it in some detail. A person, in apparently the best of health, finds that he has a fibrous or fatty tumour in the subcutaneous connective tissue, or an osseous or cartilaginous tumour growing from the shaft of some bone; and probably in a short time it is ascertained that many other

tumours, identical in character with the one first detected, are making their appearance in the connective tissue or the bones (as the case may be) of different parts of the body. Now it is indubitable that we have here a curious tendency in certain tissues of the body to undergo special morbid changes. To what is this tendency due? The first-formed tumour may be distinctly traceable to some local injury; has the growth which resulted from that injury so infected the system as to have led to the multiple development of similar growths throughout the same tissue as that which was primarily involved? Or have all the tumours (including the first) resulted from the common operation of some independent morbid irritant or poison diffused generally throughout the system? Or is there some inherent weakness or vice in the particular tissue, which has become thus largely affected, rendering it liable to take on specific morbid proliferation under the influence of mechanical violence or any other indifferent cause? In the examples which have been adduced (and many similar ones might be added), the last of the three suggested explanations will doubtless be regarded as the only tenable one; and probably it is the correct one. At all events, we have no grounds for assuming—from the presence of cachexia or other associated abnormal conditions, that any poisonous matter either is or has been present in the system; or, from the presence of lymphatic implication, that the primary seat of disease was the source of infection.

The case, however, is not quite so simple as it appears to be at first sight. The skin, like the bones or connective tissue, constitutes a special constituent of the organism, and like them (though in a still higher degree) is liable to many morbid conditions which are peculiar to itself, and which may be distributed at intervals over its surface. Now a patient may have psoriasis, beginning perhaps in a patch on the elbow or knee, and diffusing itself in spots over the greater part of the body. His father may have suffered from the same disease, and his brothers and sisters also may be subject to it. The case is one of hereditary predisposition. Now, probably no one would dream of suggesting that the spread of the disease was due here to the infecting influence of the patch which first appeared on the knee or elbow; and certainly no direct evidence could be adduced in favour of its dependence on any morbid irritant carried by the blood. The case would doubtless be regarded as equivalent, in point of origin, to that of multiple fibrous tumours or exostoses. But another patient has psoriasis, differing a little in details of distribution and colour, but (unless we go into the previous history and subsequent progress of the case) probably in no other respect from that observed in the former patient; and further, at the time of observation he may in every other sense be perfectly healthy. He had a chancre, however, some time previously, and his skin-disease is due to the syphilitic poison. Or, to take another example—an apparently healthy person becomes liable, with-

out obvious cause, to urticaria, and suffers from it off and on for years, perhaps for the remainder of his life. It is little, if at all, influenced by diet or habits and altogether uncontrollable by medicinal treatment, and moreover may be readily induced by a pinch or scratch. There seems no reason to regard this, any more than simple psoriasis, as the result of a specific irritant working from within. But another person takes a meal of mussels, and presently presents, together with more or less violent constitutional disturbance, an abundant urticarial eruption. Now here the relation between cause and effect is as obvious as in the case of syphilitic psoriasis. We have, thus, clear evidence that both psoriasis and urticaria are producible by the local operation of special poisons, which have been introduced from without, and have infected the system, and that the former may appear without necessary contemporaneous manifestation of other symptoms of disease. But do not these facts throw doubt on the non-specific origin of so-called 'idiopathic' psoriasis and urticaria, and hence also on the assumed non-specific causes of fibroma, exostosis, and the like?

Nevertheless, while many specific affections of particular tissues are certainly traceable to the influence of specific irritants, it seems not improbable that other such affections are due simply to the influence of indifferent causes acting on parts which have acquired special aptitude to take on such morbid action. At the same time it must be admitted, that the absence of collateral evidence of the presence of systemic poisoning by no means proves the absence of such poisoning; and, further, that the apparent commencement of the above or any like lesions from injury does not make it certain that this injury was its essential cause.

The difficulties which have just been briefly considered are equally apparent in the case of carcinoma and other infecting tumours. These, like exostoses and fibromata, become multiplied throughout the organism, and like them repeat in each newly-formed growth the characteristics of the growth which was first developed. But they differ from them essentially in being heterologous in structure from the tissues wherein they first make their appearance, and in the fact that they are not, or not so obviously, limited in their further distribution to one special form of tissue. They differ from them also in the fact that, however we may explain their origin, the first-formed mass inoculates the system with the disease, as truly as the inserted variolous contagium inoculates a person with small-pox, and exactly in the same way as a chancre infects its subject with constitutional syphilis. Thus, if a carcinomatous tumour makes its appearance in the testicle, the patient for a time seems, and probably is, free from disease elsewhere; but presently other organs get implicated, and in a certain sequence. First, the lymphatic glands, into which the testicular lymphatics run, become involved—these are the lumbar glands; and then, after an interval, the disease appears simultaneously in

many tissues and organs. If a patient has carcinoma of the glans penis, the next manifestation of the disease occurs exactly where the effects of syphilis first reveal themselves, subsequently to a chancre of the same part—namely in the inguinal glands. And in this case, again, at a later period the disease becomes generalised. The same rule applies equally to cancer of breast, uterus, or pylorus, and indeed to any primary cancer no matter what its seat:—first, the lymphatic glands in the neighbourhood, and especially those which lie in the direct route between the tumour and the thoracic duct, suffer; and, later on, patches of carcinoma appear, distributed throughout the organism. It must be added that, in diseases of this kind, every secondary tumour is equally infective with that which was first developed; and consequently that, just as the primary tumour causes disease in the lymphatic glands related by position to its seat, so each secondary tumour tends sooner or later to infect those lymphatic glands which are in immediate connection with it.

f. Tendency of certain morbid growths to limit their distribution to certain tissues or organs.—But although carcinomatous tumours, and such growths as are related to them by their mode of dissemination from a primary focus of disease, undoubtedly tend, when they become generalised, to involve a much wider range of tissues and organs than do fatty tumours, exostoses, and the like, it is nevertheless certain that they have preferences or elective affinities, and that these are in some degree characteristic for each species of tumour; and further that, as Virchow distinctly points out, the parts in which such affections usually originate are especially the parts which their secondary manifestations seem to avoid, and conversely. Thus tubercle and carcinoma, although severally disposed to involve secondarily a large number of organs, and many of them in common, present obvious peculiarities of distribution; for while both of them are specially apt to attack the lungs, brain and serous membranes, carcinoma is yet more disposed to attack the liver, which tubercle generally avoids, and tubercle has a marked affinity for the mucous membrane of the bowels and for the spleen, in both of which situations secondary cancer is certainly rare. And thus, again, while primary carcinoma is common in the breast, womb, and alimentary canal, these parts rarely get involved when carcinoma originates in some other part of the system. The cause of the apparent capriciousness of distribution of secondary growths is very obscure. It is of course easy to understand why the lungs, which form a kind of filter to the universal blood, should be peculiarly liable to them; and why organs, such as the liver and kidneys, which receive a specially copious supply of blood or have such arrangements of vessels as retard or lengthen its passage through them, should be affected more frequently than others. But neither such conditions, nor others connected with the relative functional activity of organs, influential, though they be, are alone sufficient to

explain the phenomenon. It has recently been ascertained that lymphatic tissue is very abundantly distributed throughout the organism; and there is some reason to believe, that the generalisation of both tubercle and lympho-sarcoma is connected with this fact, and depends either on some special proclivity to morbid processes which this tissue acquires under certain constitutional conditions, or else on the circumstance that it is the appropriate soil for the germination of the seeds of lympho-sarcoma and of tubercle. The latter is probably the correct explanation; and indeed, probably also in other cases, apparent capriciousness is mainly dependent on the special suitability of different tissues and organs for the reception and growth of different specific morbid elements—an explanation which is in entire accordance with all we know of the behaviour, of the contagia of the exanthemata, of animal and vegetable parasites, and indeed of other organic and inorganic poisons admitted into the organism.

g. Connection of dyscrasia with the origin of morbid growths.—Nothing which has yet been said relates in any degree to the question of the primary origin of infecting growths; it has simply been shown that when once developed they become sources of specific infection to their unfortunate possessors. This primary origin is referred by many persons to a 'dyscrasia' or morbid condition of system, itself supposed to be produced by the presence of some morbific matter or influence residing in the blood; and indeed Mr. Simon, who formerly adopted this view, regarded a carcinomatous tumour as a newly-developed organ, whose express purpose was to effect the separation of such poison from the organism. There are several considerations which lend countenance to this hypothesis:—when a person exposed to atmospheric changes contracts pneumonia or any other variety of internal inflammation, an interval elapses between his exposure and the commencement of the inflammation, during which some abnormal condition of the system—a dyscrasia—is present; so again the incubative stage of small-pox or measles is a period of specific dyscrasia; and further, at any rate as regards tuberculosis, we know that it is apt to come on in individuals who have fallen into general ill-health. But, on the other hand, these examples are none of them strictly analogous to that of carcinoma; and one indeed (that of the exanthem) fairly considered tells the opposite way—for its incubative period corresponds, not to the supposed incubative stage of carcinoma, but to the period which elapses between the first appearance of a tumour and its generalisation. Besides, in the great majority of cases in which we have the opportunity of observing the first manifestations of carcinoma, these are certainly not preceded by any evidence of ill-health; and, moreover, no such evidence becomes apparent until the patient is obviously beginning to suffer, directly or indirectly, from the effects of his disease.

The existence, then, of initial carcinomatous and other such specific

dyscrasiæ may fairly be denied—at all events, the only proof of their existence is the appearance of those very lesions which are attributed to their influence. And hence the only sense, in which such a dyscrasia can be conceded, is the sense in which we should admit a preliminary dyscrasia as the source of enchondromata, exostóses, fibrous tumours, leprous patches, and the like—a dyscrasia, that is to say, of limited distribution, and consisting simply in a tendency (congenital or acquired) in certain parts of the body to undergo a special kind of proliferation under the operation of various forms of irritation. It need not of course be denied in this case, any more than in that of non-infective growths, that such a tendency may exist simultaneously in various parts of the body; and that hence, although it is certainly not the rule, there may be a concurrent primary outbreak of infective growths in two or more localities.

h. Secondary dyscrasia.—But although a state of cachexia, or a dyscrasia, is not an essential antecedent of primary infective growths, there is no doubt that a condition of cachexia speedily follows upon their appearance. The fact has already been adverted to that, from any focus of morbid proliferation, there is an abnormally large reflux of nutrient fluid into the general circulation, partly by the veins directly, but chiefly by the lymphatics, and that this nutrient excess is largely charged with effete and morbid products, generated in the diseased area. These products comprise—the ordinary waste-materials, such as carbonic acid, and urea; materials which are traceable to the special chemical constituents of the part involved, earthy matter if it be bone, phosphates if it be brain; and probably also fibrine or fibrine-producing substance, which, as Virchow suggests, is manufactured at the seat of disease, and being removed thence by the lymphatics overcharges the blood and gives it its inflammatory character. But, in addition, specific affections yield specific elements, which also traverse the lymphatics, and presently mingle with the blood. What these are is not accurately known; but probably (judging from the analogies afforded by the infectious fevers) they are living protoplasmic particles evolved by the primary growing mass, which get arrested in the lymphatic glands and then infect them, by either growing parasitically among their elements, or (sperm-like) imparting to them specific properties, and which presently are shed thence in new generations, through the thoracic duct into the blood-stream, to sow themselves in distant organs. Now, in all these processes, it is obvious that we have ample sources of deterioration of the general health, and of functional disturbance of various parts of the organism—in other words, of a secondary dyscrasia. But it is obvious, also, that the degree and character of the dyscrasia will vary according to the peculiarities of the morbid process to which it is due, and especially that that accompanying the development of infective growths will be attended with specific characteristics. Further, more or less in most

cases, but in the last more particularly, dyscrasia will probably be largely increased, by the constant drain of nutriment which the growth and ulceration of tumours necessarily involve, and by the obstacles which, by pressure or otherwise, these so often interpose to the due performance of important or necessary functions.

When secondary dyscrasiæ are present we often find that some mechanical injury, or the result of some such injury, attracts, as it were, specific morbid processes. When, for example, a patient is suffering from constitutional syphilis, a local outbreak is often thus determined. It is probable that this phenomenon is due to the fact that parts, in which certain non-specific morbid processes are in progress, furnish a specially suitable soil for the growth and development of specific elements of disease, which happen to be circulating in the blood. The interesting experiments of Chauveau seem strongly to confirm this view. He found that, on injecting putrid fluids containing bacteria into the blood of healthy animals, no special consequences beyond some constitutional disturbance necessarily followed; but that if, after injecting them, the operation of twisting, and thus strangulating, one testicle was performed (an operation common in France and leading to the gradual wasting of the organ) violent inflammation with sloughing, probably attributable to an abundant development of bacteria, took place in the injured part, the opposite uninjured testicle remaining altogether unaffected.

i. Meaning of terms malignant and innocent.—It may be well here briefly to explain the meaning of the terms 'innocent' and 'malignant,' as applied to morbid growths. Malignant is almost synonymous with infecting; but not quite—for a chancre and an inoculated variolous pustule are both infecting growths, yet not malignant. The word implies, therefore, something more than is presented by either of these affections. It implies in fact, additionally, that the morbid process going on in any one locality has a tendency to invade all the tissues which are about it, and none whatever towards cure, or even to remain quiescent. A malignant tumour may, therefore, be defined as one which tends to involve all surrounding structures, and to disseminate itself through the agency of the lymphatics and veins, and has no disposition to spontaneous cure. The term 'innocent' is mostly understood to signify simply that a tumour is non-infective. Malignant tumours often present other characters which, though not necessarily associated with malignancy, are yet highly suggestive; these are, aptitude to recur after removal, abundance and rapidity of cell-growth, softness and juciness of tissue (the juice being milky), great vascularity, and marked differences of texture as compared with that of the parts in which they originate.

A very characteristic feature of most morbid proliferations, whether they be malignant or innocent, is their quasi-parasitic nature—their disposition to grow and to maintain themselves, independently of the

general health of the body in which they are developed, and from which they derive their sustenance. Thus, a large abscess, so far from becoming starved by the gradual emaciation of its possessor, will often go on increasing even more rapidly as his body dwindles away. And so also, enchondromatous, fatty and carcinomatous tumours, and tubercle, show no signs of impaired vigour of growth, even while the patient is progressively wasting under their influence. Over-nutrition and under-nutrition of the body of their host are alike without obvious influence over their progress.

2. *Hypertrophy. Hyperplasia.*

The term 'hypertrophy' is commonly used loosely of all organs or tissues which, from no matter what cause, have undergone abnormal increase of bulk. Thus, a liver enlarged by fatty deposit or lardaceous infiltration is often said to be hypertrophied, as also is an ordinary swelled testicle, or a lymphatic gland affected with tubercle or carcinoma. But in such cases as these, the enlargement is due essentially to the deposit of some extraneous matter, or the development of some inflammatory or other morbid growth; and the normal structure of the organ, so far from being increased in quantity or size, has probably undergone atrophy or degeneration.

True hypertrophy of an organ consists, either in an enlargement of its essential elements, or in an increase in their number. By Virchow the latter variety of over-growth has been distinguished as 'hyperplasia.' The former process is exemplified by the enormous enlargement of the unstriped muscular fibres of the womb which takes place during the progress of pregnancy; the latter by the over-growth of bone, which is effected simply by the multiplication of its elementary parts. It is very difficult, however, in many cases to determine positively by which of these two processes an over-grown organ has become enlarged; and doubtless they frequently co-operate.

Of all morbid processes, simple hypertrophy is that which seems to approach nearest to the processes of health; indeed it is mostly due to the operation of the very causes which produce normal increase of bulk, and in a very large number of cases is, for a time at least, protective or otherwise beneficial. Hence, it is difficult to draw the line between that normal growth of the heart, which comes with advancing years and activity of body, and that excess of enlargement which sustained and over-violent exertion brings about, and which presently reacts injuriously. Again, how much more speedily would obstructive disease at the cardiac orifices prove fatal, if hypertrophy of the heart's walls did not naturally follow upon their efforts to overcome that obstruction. Similar morbid hypertrophies of the muscular parietes of hollow viscera are always apt to arise under circumstances which compel them to long-continued unwonted action. We may refer to

the hypertrophy of the stomach which occurs when the pylorus is diseased, to that of the intestine in cases of intestinal obstruction, and to that of the bladder, or ureter, or other ducts when mechanical impediments prevent the due escape of their accumulated contents. Such consecutive, and often beneficial, hypertrophies are not confined to muscular organs, but may occur in glands, in bones, and elsewhere:—in the kidney, for example, when in consequence of the destruction of one its fellow attains unwonted dimensions, or when both undergo enlargement under the influence of diabetes; in the bones, as when a protective buttress is formed in the concavity of a curved rickety tibia.

Not all forms of hypertrophy, however, are a consequence of the attempts of organs to adapt themselves to conditions of increased work. Hypertrophies which are essentially abnormal, and have no beneficial tendency whatever, arise, in some cases from the direct influence of the nervous system; in others (and these are the most frequent), from the stimulus of excessive supply of nourishment. Amongst the former may be included the hypertrophy of the heart which long-continued nervous palpitation induces, and that form of goître which occurs in 'Graves's' disease; amongst the latter, that general enlargement of the lower extremity (in which the bones get longer and thicker than those of its fellow, and the other structures of the limb proportionately increased) met with in cases where, owing to obstruction and dilatation of its lymphatics, the whole member is succulent with nutritious fluid. A particular form of hypertrophy of the tongue in children, and the overgrowth of the skin and subcutaneous connective-tissue in elephantiasis, also are largely due to this last condition.

3. *Inflammation.*

General account.—The collective morbid phenomena which are included under this term occur as an essential, or as an accessory, part of the great majority of diseases. They represent the reaction of the system, or of parts of it, against the injurious effects of irritants which are morbid either from their amount or from their quality; the efforts by which nature endeavours to destroy, counteract, or throw out what is noxious; and those by which she strives to repair what has been injured, and to restore what has been destroyed. It need scarcely be added that inflammation often goes far beyond, or falls far short of, its aim, and often acts as it were capriciously and blindly.

The classical local signs of inflammation are *redness, swelling, heat* and *pain*. These no doubt are all present in the majority of cases—the redness being due to accumulation of blood in the dilated blood-vessels; the swelling, partly to this dilatation, partly to simple effusion and growth of tissue; the heat, in some degree to the increased afflux of blood, in some degree to the rapid disintegration that is in progress; and the pain to pressure on the sensory nerves, or to

their implication in the morbid processes. But neither redness, swelling, heat nor pain, is absolutely essential to inflammation; they are simply to be regarded as common results or accompaniments of that process.

Inflammation consists primarily and essentially in an unnatural irritability, and tendency to undue proliferation, of the protoplasmic elements of a part—these giving rise, not as in simple hyperplasia to a mere increase in the number of the normal elements, but to cells which tend to resemble leucocytes, or embryonic cells, and which never go beyond the formation of simple granulation-tissue or some variety or modification of the various forms of connective-tissue. The connective-tissue corpuscles are those in which inflammatory proliferation chiefly takes place; but all protoplasmic masses, including those of the epithelia, those connected with the nerves and striped muscles, and also those which by their coalescence form the walls of capillary vessels, readily participate in the process. As doubtful exceptions may be named the special cells of the central nervous organs, the proper liver-cells, and other cells which have attained a high phase of development.[1] But, in connection with these extravascular changes, vascular phenomena speedily ensue, and at once take an active share in the processes which are going on. Among the incidents which occur in the course of inflammation or follow upon it are, exudation, suppuration, ulceration, gangrene, and granulation or repair.

a. Extra-vascular processes.—The extra-vascular processes of inflammation may be best observed—observed freest from complication—in parts which are devoid of vessels, such as the cornea, cartilage, and certain portions of the mesentery. If a costal or articular cartilage be excited to inflammation by the mechanical removal of a bit of it, the injured surface becomes covered at the end of about a week by a soft, greyish pulp, which consists entirely of embryonic tissue, or a mass of embryonic cells, together with some newly-formed blood-vessels. If now a cross-section of the cartilage be made, so as to include its whole thickness, together with the wounded surface and the pulp covering it, the following appearances will be detected on microscopic examination:—first, in the region farthest removed from the seat of injury, the cartilage-cells and the hyaline intervening substance in a perfectly normal condition; but, on gradually advancing thence to the diseased surface (second), simple enlargement of the cells and of their

[1] It is not intended to suggest, that these highly endowed cells are incapable of undergoing any form of inflammatory change, for recent observations by M. Charcot seem to prove that the proper cells of the nervous centres may be the primary and chief seats of such changes: still less that they take no active part in non-inflammatory morbid growth, for the investigations of Dr. Croighton tend to show that heterologous growths in the liver commence with vacuolation and internal gemmation of the proper liver-cells.

nuclei, and of the cavities in which the cells are contained; third, fissiparous multiplication of the enlarged cells and nuclei, and the appearance therefore of several closely-packed nucleated cells in each originally unicellular cavity—each young cell, moreover, being invested in a thin cartilaginous capsule, and so still presenting the essential characters of a cartilage-cell; fourth, continued proliferation—the cells becoming smaller and much more numerous, losing their cartilaginous capsules, and assuming all the characters of simple embryonic cells, and the cavities containing each group of embryonic cells still enlarging at the expense of the hyaline cartilaginous substance, and hence approaching one another and here and there coalescing; fifth, an irregularly scalloped border, to the whole surface of which is attached, and from the whole surface of which grows, the grey film of embryonic tissue covering the injured surface of the cartilage—each scallop representing a portion of a primitive cartilaginous capsule, the cavity of which has come to blend with those around it; and the continuous embryonic mass representing the united proliferating contents of these and other lost cartilage-capsules. We thus see the effects of injury to be: first, growth and proliferation of the protoplasmic or living parts of the cartilage—the newly-formed cells gradually losing the anatomical and other attributes of cartilage-cells, and degrading into simple embryonic cells; and, second, progressive deliquescence and removal of the hyaline or non-vital constituent of the cartilage under the influence of this cell-growth and multiplication, culminating in its entire disappearance from those parts in which proliferation has attained its most advanced stage.

The mesentery of the adult animal forms, not a uniform lamina, but a delicate network, of which the trabeculæ are in many cases exceedingly fine, without blood-vessels, and consisting solely of a core of connective tissue, and an investing layer of polygonal tessellated epithelium. If a little solution of nitrate of silver be injected into the peritoneal cavity of such an animal, inflammatory changes take place in that epithelium, as they have just been shown to take place under analogous circumstances in the cells of cartilage. At the end of about twenty-four hours, turbid fluid is found in the serous cavity—the turbidity being due to the presence of cellular elements, presenting all varieties between ordinary pus-corpuscles on the one hand, and larger cells containing two or more oval well-defined nuclei on the other; and the epithelial cells at the surface of the trabeculæ have become plumper and larger, have lost their cell-walls, and in many cases have undergone proliferation, giving rise to pus-cells and such other forms of cells as are found floating in the peritoneal fluid. The cells adhere irregularly to their points of origin, and are invested, and to some degree retained *in situ*, by bands of coagulated fibrine which has exuded from the inflamed surface. If no further irritation be excited, at the end of a few days the cells floating in the peritoneal

fluid get opaque and fatty and perish, while those which are still adherent to the trabeculæ flatten and resume the ordinary characters of serous epithelium.

In the above two cases we have proliferation simply of the cells which are proper to the irritated tissues; in the case of the cornea, however, the results of irritation are more complex and more remarkable. The cornea of the frog consists mainly of a network formed by the union of the rays of stellate cells—the meshes being occupied by indifferent non-vital material, which corresponds to the hyaline matrix of cartilage, and to the white fibrous trabeculæ of ordinary connective-tissue. If the living cornea be irritated by the application of a point of nitrate of silver to its centre, changes presently take place in it, which soon spread, and before long involve the whole extent of its tissue, rendering it more or less obviously milky and opaque. The first changes discoverable by the microscope are in the immediate vicinity of the injured spot. Here the stellate cells first become unnaturally well-defined, and a little more granular or turbid than in health; then they swell, their branching processes at the same time growing thinner; presently these are retracted, and the still-growing cells, assuming a somewhat nodulated or botryoidal form, become as isolated from one another in the substance of the corneal matrix, as are normally the cells of cartilage in the cartilaginous matrix. Whilst these changes are in progress the cells grow more and more opaque, and their contents more and more difficult to discriminate; but soon, obvious proliferation occurs within them, the nuclei divide and subdivide—each subdivision carrying with it its own particular envelope of protoplasm—until every corneal cell becomes the mother-cell of an irregular group of embryonic corpuscles. This increase of the vital elements of the cornea is attended, as is the equivalent process in cartilage, by the liquefaction and removal of the intervening matrix, and ultimately by the coalescence of neighbouring groups of cells and their discharge from the surface of the organ. So far the process is essentially the same as in cartilage, and indeed as in serous membrane also. But something more occurs. Whilst the changes above described are going on in the centre of the cornea, and gradually spreading from that point outwards, other changes are taking place at the periphery of the cornea and creeping thence in the centripetal direction. These consist, in the gradual escape of leucocytes from the now dilated marginal vessels, and their immigration (in virtue of their amœboid properties) into the interstitial spaces of the adjoining parts of the cornea. These spaces they soon crowd, rendering the corneal tissue opaque; and soon breed, mingling their offspring with those of the proliferating corneal cells, from which they become undistinguishable. Cohnheim, who first recognised this immigration of leucocytes into the inflamed cornea, attributes all the morbid cell-development occurring in it to their presence and action, and considers that the proper

corneal cells remain perfectly passive. The active share, however, which these latter take in the inflammatory process, has been so often witnessed and described by competent observers, that there can be no reasonable ground for doubt upon the matter. The concurrence of these two processes, not only in inflammation of the cornea, but in the greater number of inflammations, seems now to be thoroughly well-established.

Processes, essentially identical with the above, mark the occurrence of inflammation in the intervascular spaces of the so-called 'vascular' tissues:—they are, growth and multiplication of the protoplasmic elements, immigration and multiplication of leucocytes; and, concurrently with this over-growth, the liquefaction or degeneration, and disappearance, of the non-vital parts, and indeed of living parts which have attained their highest phase of development. Thus, we find the earthy and organic matrix of bone eroded into cavities, the trabeculæ of white fibrous tissue attenuated into a comparatively delicate network, and muscular and nervous tissues undergoing fatty metamorphosis.

b. Vascular processes.—The condition of the blood-vessels in and about an inflamed part has long engaged the attention of pathologists. The important share which they take in inflammation is indicated by the redness which attends the process, and by the dilatation and throbbing of the arteries which lead to the spot in which it is going on. The latter fact indeed sustained, if it did not originate, the belief that the increased flow of blood to an inflamed part was determined by the active movements of the vessels of the part, in the same way that the general distribution of the blood is governed by the alternate contractions and dilatations of the heart.

That the active processes going on outside the vessels in an inflamed area create a demand for an increased supply of nourishment, has already been pointed out. This demand can only be satisfied through the medium of its blood-vessels, which consequently soon dilate, and thus attract thither an excessive amount of blood. This phenomenon, indeed, so speedily follows the event which calls it into operation, that in inflammation produced experimentally it is often the very first indication of the presence of inflammation. If the web of a frog's foot, or its mesentery, or any other convenient tissue of one of the lower animals, be irritated, and the processes which follow carefully observed, it will be seen—that the small arteries of the irritated area, gradually dilate and probably after some hours attain their maximum diameter, which may be double that originally presented by them; that, subsequently to the commencement of the arterial dilatation, perhaps some hours afterwards, the capillaries and veins of the part follow suit; and that thus at length all its vessels get proportionately enlarged. It will further be seen that, while these changes of dimension are going on in the vessels, equally remarkable

changes are occurring in the blood-stream within them:—at first, while only the arteries are affected, the rate of flow is increased; then, as general dilatation of the vessels supervenes, the stream flows more and more slowly through them (oscillating, perhaps, in some of the capillaries), and the white corpuscles congregate and cling to the vascular walls; at length the blood stagnates, and loses its serum, and the red and white corpuscles get wedged together into an apparently homogeneous or amorphous mass. While, however, this condition of stasis has been coming on in the area of inflammation, the vessels immediately around it have become dilated, and through them the blood is still circulating with unwonted rapidity.

It is at the period of stasis, or rather perhaps just previous to it—at the time when the white corpuscles or leucocytes are adhering in large numbers to the inner surface of the vessels—that that emigration of corpuscles, which has already been adverted to, and plays so important a part in the inflammatory process, chiefly occurs, and may be best observed. If at this time the small veins be narrowly watched (for it is in them that the process commences and chiefly to them that it is confined) small, transparent, button-like bodies will be seen to spring here and there from their outer surface; these gradually increase in bulk and number, and assume a pyriform shape, and presently, having acquired the form and size of white corpuscles, detach themselves from the surface from which they seemed to grow—their connection therewith having previously been reduced to a mere thread. Prior to their complete detachment they often throw out delicate processes which aid them in their ulterior movements. In this way vast numbers of white corpuscles pass in a short time from the interior of the vessels into the tissues external to them, without leaving behind them a trace of the route by which their escape through the parietes was effected.

It is obvious, then, that variations in the dimensions of vessels, and in the rate of flow of blood through them, are very important incidents in the collective phenomena of inflammation. But it is not at all easy to determine upon what cause, or on what combination of causes, the several variations depend; and especially it is difficult to trace the exact relation between the varying diameters of vessels and the varying rates of the passage of their contents along them. We know that the smaller veins, and still more the smaller arteries, are capable of contracting and dilating within comparatively wide limits, and thus of regulating to a considerable extent the amount of blood to be admitted into, or discharged from, the area to which they minister; and that this function is effected by means of their muscular walls, which, when they contract, diminish the calibre of the vessels, when they relax, permit of their dilatation. We now know also, chiefly through the labours of Stricker, that the capillary vessels are not merely passive organs, contracting and dilating in obedience to

the various degrees of blood-pressure to which they are subjected; but that, in virtue of the endowments of the living protoplasm of their walls, they possess, like the arteries and veins, a power of active contraction. And, further, we now have good reason to believe that arteries, veins, and capillaries possess, in addition to the power of active contraction and the capability of passive dilatation, a distinct power of active dilatation, or at any rate of dilatation with retention of tonicity. Again, we know that the muscular tissue of the vascular system, like that of all other parts, is under the dominance of nerves—in this case the nerves of the vaso-motor system. Contraction of vessels may be caused, either by the direct application of irritants to them, or by exciting the cut surface of the distal portion of a divided motor nerve, comprising vaso-motor fibres, distributed to them. Active or tonic dilatation seems specially to be induced by reflex action, excited by stimulating the sensory nerves of the part in, or near, which the vessels undergoing dilatation are situated. Passive dilatation takes place whenever the influence of the vaso-motor nerves is abolished or weakened, or the vascular walls lose their proper contractile power. We may gather from this statement, that the primary dilatation of the vessels of inflamed parts is due to reflex stimulation, traceable to the inordinately active vital processes which are taking place in the extra-vascular tissues; and that the later dilatation is probably merely passive. As regards the question of the variations which take place in the rate of the blood-flow in the vessels of inflamed parts, it will be sufficient for our purpose to point out, that the increase, which occurs in the early stage of inflammation in the centre of the inflamed area, and which is maintained continuously in the immediate neighbourhood of the lesion, is in obvious accordance with the physiological fact that dilatation of the smaller vessels, not only admits of a larger presence of blood in them, but allows of a more ready transit of blood through them; and that the stasis, which takes place after a time in the still dilated blood-vessels of the inflamed area, is obviously connected with the tendency which the corpuscular elements of the blood have then acquired to adhere to, and pass through, their walls—which conditions in their turn doubtless depend on the altered nutritive relations then subsisting, between the walls of the vessels and tissues external to them on the one hand, and the blood within them on the other.

c. Exudation.—The abundant fluid which sweats from the vessels during inflammation, though consisting essentially of the serum of the blood, presents modifications of constitution determined by the tissues in connection with which its escape occurs, and further involves different results according to the circumstances attending its escape. The swelling, which always accompanies inflammatory processes going on in the substance of organs and tissues, is mainly dependent on this exudation; and indeed if the parts involved be lax, serous infiltration, or œdema, is apt to spread far beyond the limits of actual inflamma-

tion. In inflammation of mucous membranes, the membrane itself, and the tissues which are subjacent to it, all get infiltrated; but, in addition, there is generally a copious discharge of fluid from the free surface. The most abundant discharge, however, takes place into serous cavities when the membrane which invests them is the seat of inflammation. It is thus that hydrothorax and ascites are often produced. The most common distinction between inflammatory fluid-exudation and blood-serum is the presence in the former of a comparatively large quantity of fibrine, or fibrinogen. This is observed to a greater or less extent in all cases, but is especially remarkable in the inflammations of serous membranes, in which the great bulk of the exuded fibrine coagulates at the moment of its escape, entangling morphological elements, and forming the false membrane which adheres so characteristically to the surface. Another, but less frequent, peculiarity is the appearance in it of mucine; this is observed chiefly when the mucous and synovial membranes are affected, and is due to the direct influence of the cells of the diseased surface. We have pointed out that the exudation of white corpuscles probably is an essential element in the inflammatory process; small but variable numbers of red corpuscles also are apt to exude in company with them; but at times the escape of blood-cells is much more abundant than can be explained by this process, and is manifestly due to actual rupture of blood-vessels—generally vessels of new formation.

d. Suppuration.—A frequent event of inflammation, and one that marks one of its recognised stages, is the formation of pus. 'Laudable pus,' as it is termed, is a thick, creamy, mawkish-smelling, alkaline fluid, containing a great abundance of corpuscles, to the presence of which its opacity and whiteness are due. The fluid part, which is called the 'liquor puris,' contains, like the serum of the blood (from which it is derived), albumen, salts, &c., and differs little from it in composition. It sometimes also presents a peculiar albuminoid substance, named 'pyine.' The corpuscular part consists almost entirely of bodies termed 'pus-cells,' which, as generally seen, are globular in form, varying between $\frac{1}{2500}$ and $\frac{1}{3000}$ inch in diameter, and differing little, if at all, from leucocytes, or so-called 'mucous corpuscles,' or embryonic cells. They are transparent, colourless, more or less granular masses of protoplasm, without investing membrane; which, though globular when dead or as usually examined, present active amœboid movements of locomotion and change of form, while still living and under appropriate circumstances. Under the influence of water, or still better dilute acetic acid, the general substance of each corpuscle swells up and becomes more transparent, and one nucleus, or more frequently two, three or even more nuclei, are revealed within it.

It is obvious, then, that there is little or no microscopical difference between typical pus-corpuscles and the corpuscles developed, previous

to the suppurative stage, by the breeding of connective-tissue cells and other stationary protoplasmic bodies, or of immigrant leucocytes; and that they both have a common origin. Indeed at every suppurating surface the gradual transition of the one into the other may be readily observed. There is, however, some reason to doubt whether pus-corpuscles ever multiply, and some reason to believe that the groups of small nuclei they contain are to be regarded as the last abortive attempt at reproduction.

It is not difficult to trace some of the steps which lead to the development of pus. It has already been shown, that when inflammatory proliferation is going on, the indifferent or non-vital tissues between the groups of swarming cells gradually get eroded and removed; and that presently, as these disappear, the neighbouring groups of cells come into direct relation with one another, and thus constitute an almost uniform mass of embryonic tissue. They still cohere, however, either as epithelial cells do, or through the intervention of some scanty adhesive material. It needs only the loss of this cohesive property, and the addition of the liquor puris, to convert this inflammatory hypertrophy of tissue into orthodox pus. It is thus, indeed, that suppuration takes place at the surface of an ulcer; it is thus, also, that abscesses arise. In the latter case, softening occurs in the centre of some proliferating region; and the cells, which would otherwise have formed an ingredient of solid living tissue, change into pus-corpuscles; by extension of the softening, the abscess enlarges, and more corpuscles are added to its contents; and, further, the existence of a cavity induces towards it a rapid migration, both of the extravasated leucocytes, and of the other embryonic cells which crowd the periphery. By a continuance of the above processes, abscesses approach neighbouring surfaces, point, and presently rupture. The pus-corpuscles contained within abscess-cavities speedily undergo degenerative changes, and perish—they get studded with fatty particles, swell, and subsequently break up into a detritus; or they contract and become opaque and angular; or they undergo calcareous impregnation. And thus the contents of abscesses are gradually, sometimes absorbed, sometimes converted into caseous, mortary, or other such stuff; and a more or less perfect cure ensues.

Pus of recent formation does not always present the exact characters above assigned to it, but sometimes is thin and watery (*ichor*), sometimes contains a greater or less admixture of blood (*sanies*), and sometimes is distinctly fetid. These obvious peculiarities are dependent on something special, either in the condition of the patient or in that of the part which is suppurating, and are connected with peculiarities of microscopical and chemical constitution. Thus, we find sometimes, that all the pus-corpuscles have already undergone degenerative changes, and that in place of the orthodox cells we have only granule-cells, or it may be a mere molecular debris; sometimes,

that abundant blood-corpuscles are mingled with the other elements of pus; sometimes, that fragments of tissue, bone and the like, are contained in it; and sometimes again, that bacteria and other minute living organisms, are present. The admixture of visible particles of tissue implies the association, with the suppuration, of somewhat rapid destruction of parts, and often indicates necrosis or gangrene; the presence of bacteria and the like is a proof, either that the pus is undergoing putrefaction, or that the blood generally is infected with them. Under all these latter conditions fetor is pretty certain to be present.

e. Destructive processes.—The destructive effects of inflammation have already been adverted to. They are shown in the softening and disintegration which takes place in the hyaline substance of cartilage, in the white fibrous element of connective-tissue, and in the earthy matrix of bone, during the gradual multiplication of cellular elements, and especially during the formation of abscesses. They are shown also in the fatty and other degenerative processes which, under similar circumstances, go on in muscle, nerve-cells, and other higher tissues, taking no part in the inflammatory proliferation. Destruction occurs, however, in a yet more marked form in the various processes termed 'ulceration' and 'necrosis' or 'gangrene.'

In *gangrene* a larger or smaller portion of tissue perishes, and is probably separated in mass from the neighbouring living textures. The death of the part is due essentially to its deprivation of nourishment; which deprivation depends mostly on the obstruction of the arteries leading to it, either by clot in their interior, or by thickening of their walls, or by external pressure arising from accumulation of inflammatory products or other causes. In inflammatory gangrene the parts involved usually are swollen and succulent, for the reason mainly that, like all inflamed tissues, they were previously infiltrated with abundant exudation.

In *ulceration* the destruction of parts is molecular, or by small fragments, and progressive. It has long been a question whether, in the common forms of ulcer which gradually extend in area and in depth, the apparent melting away of tissue, on which their extension depends, is due to absorption by the vessels or to discharge from the surface. It is obvious, in any case, that this gradual disappearance of tissue must be preceded by its liquefaction, degeneration or death; for these are normal and necessary processes by which, even in health, the worn-out portions of the body are prepared for removal by absorption, and equally the processes by which, during inflammation unattended with ulceration, the more lowly-organised structures—the matrix of cartilage, cornea, bone, and the like—melt away and disappear; and indeed it is impossible to conceive of any other. Looking then to the fact, that the molecular destruction, which is going on at the surface of ulcers, presents no real difference from that which is

going on in the non-ulcerating stage of inflammation (the products of which are certainly removed in chief measure by absorption), it seems not improbable that a portion of the effete products of ulceration also may be removed in this way. But, on the other hand, since the destruction takes place at a free surface, which is exuding a considerable quantity of fluid, and even of corpuscular elements—conditions which are highly favourable for the discharge from that surface of any effete matters which are produced there—it seems hardly likely that these should be removed by absorption only. Indeed it seems most probable, on physical grounds alone, that the chief removal of ulcerative detritus should be effected in the manner last described. That it is mainly thus removed is now generally acknowledged. It may be added, in confirmation of this view, that the discharge from ulcers involving bone contains earthy matter, and even small fragments of bone; and that generally when ulceration is extending rapidly, fragments of disintegrated tissue are suspended in the fluids which exude from the ulcerated surface. In sloughing ulcers, such as those attacked with hospital gangrene, extension is attended with an abundant separation of shreds and flakes of dead tissue from the diseased surface.

It will of course be understood that the above remarks apply only to those cases in which ulceration is in progress. Excavations, whether termed ulcers or not, in which the surfaces are granulating are examples, no longer of ulceration, but of repair and restoration.

f. Organisation and granulation.—It has already been shown that, at an earlier stage than that at which suppuration occurs, the results of inflammatory proliferation are the production of a greater or less quantity of embryonic tissue, or tissue at a low phase of organisation. The intervening matters melt away, and the newly-formed cells come into near, if not absolute, relation with one another; or, if the process is occurring at the surface of a serous membrane, the new-formed cells are retained in connection with that surface by entanglement in the fibrine which coagulates there. In the progress of organisation important changes ensue. In the latter case the embryonic corpuscles, entangled in the fibrine, throw out delicate processes, by which they presently unite with one another to form a network, in the meshes of which the fibrine is then contained. At the same time, new vessels, starting from the normal vessels of the subjacent serous membrane, shoot into the adventitious tissue. Later, the fibrine undergoes liquefaction and removal, and the interspaces between the cells get occupied by a form of white fibrous tissue, which they are instrumental in manufacturing. In the case of the organisation of inflammatory products occupying the substance of organs, essentially the same series of events happen:—the embryonic cells undergo conversion into connective-tissue corpuscles; new vessels are formed; the fibrine which has coagulated, and in a greater or less

degree the proper or special highly-endowed elements of the parts, got removed or impaired, and the non-vital elements of connective-tissue are deposited in their place. In both cases, the new-formed tissue belongs to the connective-tissue series, and in both tends to get contracted and dense and hard in texture.

The processes, here briefly described, take place also in the healing of wounds, and in the filling up of ulcerous or other excavations by granulation. Granulations are hemispherical masses of cells, produced, and increasing in size, by constant cell-breeding and immigration of leucocytes. The cells in the first instances are purely embryonic in character, and many of those growing at the free surface, and others which migrate thither, are shed as pus. But presently those which remain undergo differentiation; the majority elongate or send out processes and gradually evolve connective-tissue; whilst others also elongate, but become aggregated into solid cylindrical loops, soon to be hollowed into channels of communication with previously-existing vessels, and thus themselves to become blood-vessels, and important agents in the further growth and vitality of the granulation-tissue. Rindfleisch describes and figures the formation of lymphatic tissue in the over-grown vegetations of 'proud flesh.' Neighbouring granulations, as they grow, run together and blend, and thus at length cavities get filled up with a tolerably homogeneous mass of new-formed tissue. But when the granulating mass attains the general level of a free surface, such as that of the skin, its further growth under ordinary circumstances becomes arrested, epidermis begins to shoot from the normal epidermis at the margins over the edges of the granulating area, which at the same time contracts, and soon, if it be of small size, gets completely covered. It is even now a disputed point, whether a granulating surface has any power of itself to generate epidermic cells. It is certain, however, that the chief development of new epidermis begins from old epidermis, that very large breaches of surface never become thus covered unless aided by artificial means, and that the grafting here and there, upon such a surface, of small fragments of epidermis results in the formation of a number of epidermic islets, from which new epidermis spreads radially. In the healing of a clean cut, of which the edges are placed in close apposition, the process is nearly the same as that of the organisation of false membranes. The divided vessels pour out blood and serum, containing fibrinogen; and this coagulating entangles corpuscular elements, and cements the divided surfaces; presently, the white corpuscles thus entangled, and others which migrate among them, emit processes and form a network, mapping out the fibrinous cement into comparatively small islets. The further steps of the process present no peculiarity.

The ultimate product of inflammatory organisation is generally what is commonly termed 'cicatricial tissue'—a form of connective-tissue presenting much hardness and compactness, comparatively little

vascularity, small and widely scattered plasmatic cells, and relatively abundant and dense interstitial substance; which becomes bony when developed in connection with bone, and contains fat when it replaces normal fatty tissue; but which, while it is capable of reproducing, with more or less imperfection, the various tissues comprised in the connective-tissue group, rarely results in the reproduction or development of higher tissues, such as muscle, and probably never in the formation of organs. Hair and glands, for example, never appear in entirely new-formed skin.

In some cases the results of inflammatory proliferation are somewhat different. The process gets chronic, cell-generation goes on comparatively slowly, and the newly formed tissue, instead of contracting and hardening, becomes swollen and perhaps softer than natural, and forms, in fact, an increasing projection or lump, in which the cell-elements remain predominant, but tend to fatty and other forms of degeneration. Such results are seen in keloid and in some forms of arterial atheroma.

g. Spread.—The tendency which inflammations have to spread is at least as remarkable as that presented by other proliferating affections. If a patient has local eczema, produced by the application of some irritant, presently other patches of eczema appear in the neighbourhood; if he has a boil, it commences in a point, and increases by involving more and more of the surrounding tissues, and soon other boils arise in its vicinity; in erysipelas and pneumonia, and in inflammations of serous and mucous membranes, the same rule of local spread, or spread by simple continuity, is even more obvious. But inflammations also tend, in many cases, to spread through the agency of the lymphatics and veins, and thus to involve remote parts, and other tissues besides those first affected. Thus, suppuration, occurring in a toe or finger, is apt soon to be followed by inflammation in the course of the lymphatic vessels, and of the lymphatic glands in the groin or axilla; and indeed generally there is a tendency, if the local inflammation be sufficiently intense, for the nearest lymphatic glands to get implicated. And thus again, in certain cases, inflammatory processes become generalised by means of the circulating blood, so that tracts of inflammation, secondary to some primary tract, appear, either simultaneously or in quick succession, in various parts of the body. Ordinary pyæmia furnishes a typical example of this connection; and it is not impossible that the frequent association of inflammation in different organs, and even the invasion of successive joints in acute rheumatism may admit of similar explanation.

h. Constitutional effects.—We must not forget to consider, however briefly, the influences which inflammatory processes going on in one part of the system exert on the system generally. Patients who are suffering from acute inflammations are soon affected with febrile symptoms. To what are they traceable? In some degree, no doubt,

to the direct influence which abundant local proliferation of tissue exerts generally upon nutrition. It will be recollected, however, on the other hand, that the copious and active proliferation attending the formation of an extensive surface of granulations, or the development of the fœtus, produces no such constitutional disturbance. But, indeed, the inordinate consumption of nutrient matter is certainly not the main cause of the constitutional symptoms of inflammation. It has been proved by direct observation that a part generates much more heat when inflamed than when in its normal state; and that the blood in the veins coming from an inflamed area is distinctly hotter than the blood brought thither by the arteries. It is certain, therefore, that a part of the febrile temperature of the system must be due to the dispersion of this excessive locally-produced heat. Again, as we have already pointed out, wherever inflammatory proliferation is active, there also the processes of effusion from the blood-vessels, of molecular disintegration, and of lymphatic absorption are specially active; and thus large quantities of modified nutrient fluid, and of products of decay, alike, are being constantly removed from the seat of disease and poured through the thoracic duct into the systemic veins. It seems highly probable that here is the source of the comparatively large presence of fibrinogen which is so characteristic a feature of the blood of inflammation, and that here also is the main source of the excess of urea and other products of retrograde metamorphosis, which are presently discharged by the various emunctories. There can be no doubt that the general symptoms of inflammatory fever are largely due to the heightened temperature, and to the alteration and deterioration of the blood, which have been thus produced—conditions which, according to their amount, must necessarily influence in a greater or less degree the nutrition and the functions of all parts of the system. It is certain too, that the nervous system, mainly by its vaso-motor branches, plays an important part in the production of febrile disturbance, though what that part is is not easy to identify; and that the symptoms of inflammatory fever are largely modified, chiefly in the way of complication, by the interpolation of other symptoms, due to the modification, impairment, or destruction of the normal functions of the organ which happens to be affected.

i. Varieties.—In the foregoing pages we have discussed the phenomena of inflammation in the abstract; our account of inflammation would scarcely be complete, however, if we failed to point out some of its varieties—varieties depending, partly on the intensity of the process, partly on the organ implicated, and partly on the nature, and mode of operation, of the cause; and revealing themselves as such, either by their extent and arrangement, or their special tendencies, or their duration. It need scarcely, perhaps, be pointed out, that we trench here upon the domain of specific diseases, or diseases in which the inflammation is a mere secondary phenomenon, excited and kept

up by the operation of some specific irritant, which has been received into the system and then distributed through it. But indeed, as knowledge advances, we see more and more clearly that, in every case of inflammation which comes before us, the inflammation has been excited by some cause which imparts to it certain distinctive features—that it is specific—and we recognise the fact, half unconsciously perhaps, by distinguishing most varieties of inflammation by specific names.

First. *Varieties as to extent and arrangement.*—In many cases, inflammation pervades, with tolerable uniformity, the whole of an organ or tissue—such is the case in pneumonia, peritonitis, erysipelas, and pityriasis rubra; in many cases, it is irregularly distributed in patches or spots, as in the rashes of typhus and enteric fevers, in urticaria, shingles, and lobular pneumonia; in other cases, it assumes certain definite patterns—discs in lepra, rings in erythema circinatum, and ringworm, crescents in measles, and sinuous bands in some cases of secondary syphilis.

Second. *Varieties as to result and intensity.*—It is certainly a striking fact, that some forms of inflammation, no matter how severe they may seem, or threaten, to be, never pass beyond the earlier stages of the process; while others, which commence probably with the mildest indications, invariably go on to suppuration or gangrene. In such diseases as measles, pityriasis, and lepra, the local phenomena of inflammation are always exceedingly slight, and consist in little more than hyperæmia in patches, followed by modification, and then detachment, of the overlying epidermis. In urticaria, the process, if more intense for the time, is far shorter in its duration; for here we get pretty intense congestion, with rapid effusion of serum into the congested tissues, which subsides in a few hours or even in a few minutes, and is rarely followed even by desquamation. In eczema, herpes, and pemphigus, the local congestion is always attended with abundant effusion of serum beneath the epidermis. Now, in all the above cases, notwithstanding the marked differences of detail which they exhibit, the changes are rung only on mere congestion and effusion, together with (as is of course always the case) a certain amount of nutritive change, if not of actual proliferation. In other cases, suppuration seems to occur almost invariably; it is so with small-pox and cow-pox, impetigo and ecthyma; and in inflammation affecting the periosteum, and the womb immediately after parturition, this suppurative disposition is extremely well-marked. In other cases, again, the tendency of the inflammation to end in the death of tissues, that is, in ulceration or gangrene, is a characteristic feature; as examples we may adduce erysipelas, carbuncle and hospital gangrene.

Third. *Varieties as to duration.*—Inflammations are acute or chronic in their progress. Acute inflammations are sometimes, as in factitious urticaria, remarkably evanescent. Chronic inflammations are chronic in different fashions:—in some instances the inflammatory process, as

in the case of a patch of psoriasis on one of the knees, or of a sinus constantly discharging pus, is continuous and of long duration; in a larger number of cases chronicity is due to a succession of acute attacks, each one of which may have but little intensity. It is thus that urticaria assumes the chronic form of urticaria evanida, and that erysipelas and eczema become perpetuated; we may add to the list rheumatism and gout. It seems probable also that cirrhosis of the liver, referrible to alcohol, is rendered chronic by the repeated irritation induced by the repeated application of the alcoholic poison. It is in these latter forms of chronic inflammation, more especially, that the proliferation of tissue, which attends all inflammations, becomes constant, and leads to a substantial addition to the normal bulk of a part; that bones acquire increased thickness and density; and that the interstitial tissue of the liver, kidneys, lungs, and nervous centres gets augmented in quantity, and by its augmentation leads to the gradual destruction of the essential glandular elements.

4. *Tumours.*

General Account.—It would be foreign to the purpose of this work, and to a great extent out of place, to enter into anything like a minute account of the various forms of tumours which are described by pathologists. We purpose, however, to pass them generally in brief review—describing at greater length those of them which have a special relation to the practice of medicine, and a special interest therefore for the physician. Tumours, in the proper sense of the term—that is, morbid proliferating growths, or neoplasms—have a very close affinity with simple hypertrophy or hyperplasia on the one hand, and with mere inflammatory overgrowth on the other. Structurally considered, they are in truth, in many cases, a simple hyperplasia or overgrowth of normal tissue, differing, however, from true hyperplasia in the facts—first, that they are overgrowths occurring in a limited district; and, second, that their growth has no relation to the general growth of the tissue out of which they spring, or to the general nutrition of the body. In many cases, again, tumours and simple inflammatory overgrowths are structurally identical; but generally the latter are more rapid in their development than tumours are, and at the same time, much more ephemeral in their duration.

Tumours have been variously classified. They have been divided into the two large groups of *cystic* and *solid* tumours. But cysts, although a very characteristic feature of some new formations, are for the most part merely incidental to them, and their presence or absence can in no sense furnish the basis of a scientific classification. Again, they have been distinguished into those which are *innocent* and those which are *malignant*. It need scarcely be said, that the question of the malignancy or non-malignancy of a tumour is always, in a practical

point of view, of supreme interest; and it may be allowed that, in a large number of cases, malignancy is linked to special structural characters, and may be predicted from them. But, on the other hand, it is now generally admitted that malignancy varies in degree, and that few if any proliferating growths are wholly free from liability to assume malignant properties. Virchow, accepting the law which J. Müller enunciated—namely, that 'the tissue which constitutes a tumour has its type in one of the tissues of the organism, either in its embryonic condition or at the period of its complete development'—classifies tumours according to their structural relations with the normal tissues of the body. Such a classification is at once scientific and intelligible; and although many difficulties, and much room for difference of opinion, present themselves when it is attempted to carry it out in detail, there can be little doubt that it is sound in principle, and will ultimately be universally adopted. But, admitting that all tumours have their types in the normal tissues, it does not always happen that a tumour has its type in the very tissue in which it originates. When a tumour arises in a tissue from which it takes its pattern, it is regarded by Virchow as 'homologous;' when, on the other hand, it is developed in a tissue which it does not thus resemble, he calls it 'heterologous.' The latter term has often been used of malignant tumours, in the belief that they are something altogether different and distinct from the normal elements of the body—something in fact of the nature of parasites; and it is well to know that, even in the more accurate and limited sense in which Virchow employs it, it still carries with it the sense of malignancy. Most malignant tumours are heterologous.

Virchow divides tumours into four groups, as follows:—1, tumours formed at the expense of the elements of the blood, or tumours by extravasation and exudation; 2, tumours referrible to the retention of products of secretion, and the consequent dilatation of ducts or cavities; 3, tumours originating in proliferation, which he subdivides into *histioid* tumours, or such as are formed out of a single tissue, *organoid*, or such as are characterised by greater complexity and an approach to the structure of organs, and *teratoid*, or those comprising a combination of organs; and 4, or lastly, complex tumours, in which features characteristic of two or more of the foregoing groups are combined. The first two of Virchow's groups embrace a series of pathological results which can only be regarded conventionally as tumours; all true tumours are included in his third and fourth groups.

We shall not discuss the details of the above classification, nor shall we reproduce here the convenient modification of it which MM. Cornil and Ranvier have published; yet, in the brief account of tumours which we are about to give, we shall be guided in a very great degree by the views of these authors. Indeed the modifications, mainly of arrangement and proportion, which we shall introduce, will have

reference almost entirely to convenience of description and to clinical considerations. We shall arrange tumours (omitting, as will be observed, all further reference to the teratoid and complex forms) in the following groups:—

a, tumours which have their type in the various forms of connective tissue; this includes the fibrous tumour or *fibroma*, the fatty tumour or *lipoma*, the mucous-tissue tumour or *myxoma*, and one or two less important varieties;

b, tumours composed of cartilaginous tissue, or *chondromata*;

c, osseous tumours, or *osteomata*;

d, tumours formed of nervous tissue, or *neuromata*;

e, tumours consisting of muscular tissue, or *myomata*;

f, vascular tumours, or *angiomata*;

g, tumours consisting of lymphatic tissue, or *lymphomata*;

h, *tubercle* and *granuloma*, including syphilitic gummata, and farcy;

i, *sarcomata*, or tumours which resemble embryonic tissue;

j, tumours presenting an alveolated structure—the alveoli being formed of connective tissue, and occupied or lined by closely packed epithelium-like cells; all these are embraced in the general term '*carcinoma*' or '*cancer*.'

a.—Connective-tissue Tumours.

i. *Fibrous tumour, or fibroma.*—Tumours of this kind consist essentially of connective tissue—that is of a network of plasmatic cells, separated from one another by bundles of white fibrous tissue and different proportions of elastic fibres; the last, indeed, are often absent. They are rosy, greyish, yellowish, or white in tint; are sometimes dense and close-grained like fibro-cartilage, sometimes soft, loose in texture, and succulent; are provided for the most part with scanty and small blood-vessels, and are sometimes non-vascular; and very often, when involving a mucous or a serous surface, involve also the glandular and papillary structures, which then undergo hypertrophy. Fibrous tumours often originate in the subcutaneous connective tissue; often, too, in the substance of the skin—producing, sometimes warts or papillomata, sometimes '*molluscous*' tumours, and sometimes pedunculated masses of enormous bulk. Again, they are frequently developed in connection with mucous surfaces, forming mucous polypi. The opaque cartilage-like patches often seen on the surface of the spleen, heart, and other viscera, are fibromata; but their plasmatic cells are scanty, indistinct, and much flattened, the fibrillated intermediate substance is densely stratified, and they are without vessels. The thickening and induration of the skin and subcutaneous connective tissue, in elephantiasis Arabum, are chiefly due to the growth of connective tissue, and constitute a diffused form of fibroma. It is very difficult to separate, by a defined line, the results of chronic inflammation from

fibromatous tumours, especially from the diffused forms of fibroma. And, indeed, papillary growths and polypi are frequently a simple sequela of ordinary inflammatory processes; and further, there is little if any real difference between the forms of fibroma involving the pyloric extremity of the stomach, or the substance of the mamma, which we generally regard as of the nature of tumours, and the fibrous growth invading the liver in cirrhosis, which is commonly considered to be simply inflammatory. Fibrous tumours are apt to undergo various forms of degeneration, especially the fatty, mucous, and calcareous. They are almost invariably free from malignant tendency.

ii. *Fatty tumour, or lipoma.*—Fat is a mere modification of connective tissue, in which the plasmatic cells have become distended with oil, so that their protoplasm and nuclei can only be recognised with difficulty, and they themselves are transformed into globular, or (from mutual pressure) polyhedral, bodies. Fatty tumours consist, for the most part, simply of newly-developed fat-tissue, and present little if any structural differences from normal fat. They vary in size, and generally are lobulated, and capable of pretty easy enucleation from the tissues in which they are imbedded; but sometimes their limits are ill defined, and they pass gradually into the normal textures. Lipomata often originate in the subcutaneous connective tissue, and occasionally in the submucous and subserous tissues; also in the neighbourhood of glandular organs; and indeed generally wherever fat exists naturally. Not unfrequently they form polypi or pedunculated tumours. There are several well-defined varieties of fatty tumours:—one, which may be called *fibrous lipoma*, is characterised by the presence of abundant fibrous tissue; another—the *myxomatous lipoma*—presents the combined characters of myxoma and lipoma; a third is the *cystic lipoma*; and the last which we may enumerate is the *erectile* or *cavernous lipoma*. Further, fatty, like fibrous tumours (to which they are closely related), are liable to undergo calcareous and other forms of degeneration, and are probably always innocent.

iii. *Mucous tumour, or myxoma.*—Mucous tissue, which is common in the fœtus, exists permanently only in the vitreous humour. The tissue of the umbilical cord furnishes a typical example of it. It consists of plasmatic cells, which are generally stellate like those of connective tissue or bone, and of an intercellular substance, which, instead of being solid, as in these latter cases, is transparent and fluid and contains mucine, or the characteristic constituent of mucus. Myxomata are lobulated tumours, gelatinous in consistence, translucent, and yielding a transparent, glairy, never milky, fluid. Under the microscope they are seen to consist of scattered cells, sometimes round or oval, often stellate, and an abundant network of capillary vessels, separated from one another by the structureless fluid, or semi-fluid,

mucus which gives them their specific character. They vary in colour and consistence according to the relative proportions of cells and mucus which they contain, being more opaque and denser as the cellular element predominates. They originate in most places in which normal fat occurs, and indeed there seems to be some definite relation between them and fat. But they occur elsewhere. Their most common seats are the subcutaneous and submucous tissues, and the connective web between muscles; but they are not unfrequently met with in the brain and in the course of nerves, in glandular organs such as the breast and kidney, and beneath the periosteum. In connection with the skin and mucous membranes, they often form papillary or polypoid outgrowths. Placental hydatids are a good example of this latter variety. Sometimes myxomatous tumours contain cavities (*cystic myxoma*), or their cells get distended with fat (*lipomatous myxoma*), or their intercellular mucus tends to condense and become cartilaginous (*enchondromatous myxoma*), or their vessels are extraordinarily abundant and large (*vascular* or *erectile myxoma*). Myxomata, when not occurring in situations where fat is normally present, must be regarded as heterologous; and they then occasionally present malignant characters. Generally, however, they are innocent, and do not even return after removal.

iv. *Glue-like tumour, or glioma.*—This is a tumour which, according to Virchow, consists of connective tissue resembling that of the nervous centres; and in fact it originates almost exclusively in these centres, in the course of nerves, and in the retinæ. The neuroglia consists of very small and delicate cells, imbedded in a finely granular or amorphous substance. These have a tendency to be stellate, and, in carefully prepared sections, appear to unite with one another by their rays, so as to map out the intervening substance into small polygonal areæ. Gliomatous tumours present the same structure, and are generally white and medulla-like in aspect, and exceedingly soft. They vary no doubt considerably, in respect of the relative proportions of their cellular and intercellular elements, and in their tint, consistence, and vascularity; and they run, on the one hand, into myxoma, on the other into the small round-celled variety of sarcoma, with one or other of which it is difficult to avoid confounding them. They are apt to undergo mucous, caseous, or fatty degeneration, and to become cystic. The situations which they affect, and the tendency they have to attain a large size, render them dangerous; but they are rarely malignant.

b.—Cartilaginous Tumours, or Chondromata.

Chondromata consist of cartilaginous tissue—that is of cells surrounded by lamellated thickenings, and separated from one another by intercellular substance, yielding chondrine, which is generally hyaline,

as in ordinary auricular cartilage, but may be reticulated as in yellow cartilage, or fibrous as in fibro-cartilage. Cartilaginous tumours have for the most part a slightly translucent or pearly aspect, and a whitish, greyish, or yellowish hue. They vary greatly in consistence, being sometimes dense and hard and crisp, sometimes forming a diffluent pulp. They are generally distinctly lobulated, the lobules being separated one from another by connective tissue, which conveys their nutrient vessels; for the cartilaginous tissue itself is entirely extravascular. The tumours are often perfectly well-defined; but they are sometimes irregularly diffused through the tissues or organs in which they originate. Under the microscope they present many varieties of character. Their cells vary in size and number, and are always encapsuled; they are generally round or oval, but occasionally branched or stellate like those of the cornea; further, they not unfrequently undergo fatty or calcareous degeneration. The intercellular substance, which, as previously stated, may be hyaline in character, or consist in part of either white fibrous tissue or elastic fibres, sometimes softens into a mucous fluid in which the cartilage-cells are simply suspended. Chondromata in this latter condition have a resemblance to the intervertebral cartilages; and it is by such softening in patches that they occasionally become cystic. Virchow divides chondromata into *ecchondroses* and *enchondromata*. The former are merely outgrowths from the normal cartilages, and are therefore homologous; th · never attain important dimensions, are invariably innocent, and very apt to be converted into true bone. The most interesting examples of ecchondrosis are the cartilaginous outgrowths which take place in joints affected with chronic rheumatoid arthritis. Enchondromata are heterologous; they occur most frequently in bones, especially in the long bones; but they are also met with in the subcutaneous connective tissue, and in the aponeuroses, in the lungs, parotids, testicles, ovaries and mammary glands. Enchondromata generally no doubt are innocent; but they certainly are sometimes distinctly malignant, extending along lymphatic vessels, involving lymphatic glands, and ultimately invading remote organs.

c.—Osseous Tumours, or Osteomata.

Osteomata are generally divided into three species—namely, *ivory osteomata*, *compact osteomata*, and *spongy osteomata*. The first species is met with on the inner surface of the skull, and at the joint ends of bones and elsewhere; it is characterised by remarkable compactness of tissue, and under the microscope presents bone-corpuscles and canaliculi (which latter run radially to the surface), and a total absence, or great deficiency, of Haversian canals, and hence of vessels. Compact osteomata present the ordinary characters of compact bone. Spongy osteomata, as their name implies, resemble more

or less closely the spongy or cancellous tissue. Osteomata springing from the surfaces of bones are known as *exostoses*; those originating in the substance of bones may be named *enostoses*. Both varieties are clearly homologous. But osseous tumours are sometimes heterologous. Thus, they appear in the connective tissue, in the membranes of the brain and cord, in the brain itself, in the choroid and vitreous humour of the eye, in the lungs and in the skin. True osteomata, even when heterologous, are probably never malignant. Nevertheless tumours, which have undergone more or less perfect conversion into true bone, are sometimes malignant in a very high degree. Such tumours, however, are made up in great measure of cartilaginous or embryonic tissue, and should probably be regarded as chondromata or sarcomata which have undergone calcareous or osseous transformation.

The teeth occasionally present outgrowths of their own tissue, which have been named *odontomata*.

d.—Nervous Tumours, or Neuromata.

The term 'neuroma' is often applied loosely to all growths occurring in the course of nerves; and thus myxomatous, fibrous and various other equally distinct tumours have, to a large extent, been regarded as varieties of neuroma. Neuroma, in the strict sense of the word, means a tumour formed of nervous tissue—either vesicular like that of the ganglia or central nervous organs, or fasciculated like that of the nerves or medullary substance of the brain. The former variety is exceedingly rare, but has been described as occurring in the brain and spinal cord. The latter variety is more common, but nevertheless of unfrequent occurrence, and is met with only in the course of nerves. True fasciculated neuromata generally are small white hard tumours, occurring singly or in numbers along a nerve-trunk, or more commonly at the extremities of nerves, which have been divided in the amputation of a limb. They are invested with, and permeated by, very dense fibrous tissue, the presence of which makes them difficult of examination; but their essential character is, that they contain a large number of newly-developed nerve-fibres, which form an abundant and intricate network. These generally have the double contour; but neuromata containing only pale fibres have been described.

e.—Muscular Tumours, or Myomata.

Striped muscular fibres have been discovered only in congenital tumours. Unstriped muscular fibres, on the other hand, are of common occurrence in morbid growths. Myomata are most frequently met with in the uterus, and it is in connection with the uterus that their characters may best be studied. The so-called 'fibrous tumours' of this organ are, almost without exception, muscular tumours. These

vary greatly in size, have a reddish or greyish fleshy aspect, are generally exceedingly dense, and present a lobulated character with curvilinear bands of fibres interlacing with great complexity. They always originate within the walls of the uterus, and hence, in the early stage, are surrounded by the uterine muscular tissue; but if seated near either the mucous or the serous surface, they are apt ere long to protrude through the fibres which embrace them on that side, and presently to become pedunculated. Microscopically, they are found to be identical in structure with the uterine muscular walls. Further, like them, they are capable of hardening in contraction, and again of undergoing relaxation. Moreover, they increase during pregnancy as the uterus itself increases—their muscular fibres undergoing similar and equal hypertrophy; and when, after parturition, the uterine walls suffer involution they also suffer in the same sense. Uterine muscular tumours frequently degenerate:—the muscular fibre-cells get fatty; or their tissue undergoes mucous transformation—considerable patches becoming softened and infiltrated with mucous fluid, and not unfrequently converted into cysts; but the most frequent and important change is due to the deposition of calcareous matter, partly in the connective tissue of the tumour, partly in its muscular fibres, by which means nearly its whole substance may at length be converted into a hard calcareous mass. This latter form of degeneration generally commences in the interior; occasionally, however, it starts from the periphery, and it may remain limited to the periphery. Myomata rarely, if ever, originate except in tissues which themselves contain muscular fibres. After the uterus, they are most frequently met with in the prostate, and alimentary canal. They have also been found in the scrotum, labia majora, and ovaries. They are always innocent.

f.—Vascular Tumours, or Angiomata.

Several of the tumours which have already been described, and several of those which we shall presently discuss, are liable to be exceedingly vascular—partly from excessive formation, partly from general and irregular dilatation, of blood-vessels—and thus to assume an erectile or cavernous character. And, indeed, although we have adopted the name 'angioma' for a group of tumours, there are few, if any, in which vascular hypertrophy or hyperplasia constitutes the sole, or even the essential, characteristic. Angiomata may be conveniently divided into two species, in the one of which the newly-developed vessels are properly formed arteries, veins, and capillaries, and, in the other of which the blood traverses a series of lacunar spaces, like those of erectile organs. The former may be called 'simple angiomata,' the latter 'cavernous angiomata.' *Simple angiomata* form violet or red, more or less elevated, patches, the general seat of which is the skin or subcutaneous connective tissue. Their vessels are

abundant, tortuous and dilated, and often present irregularities of calibre, and even pouch-like protrusions. Amongst these must be reckoned the small racemose knots, which often make their appearance on the face and elsewhere—sometimes in considerable numbers—and in which the chief morbid phenomenon is dilatation of small arteries and veins. *Cavernous angiomata* are also known by the name of *erectile tumours*. They occur in the skin and subcutaneous connective tissue, in the neighbourhood of the external mucous orifices, and in some of the internal organs, more especially the liver and spleen. They have a spongy character, which is due to the comparatively large size of their vascular lacunæ, and the comparatively small amount of their solid tissue. The lacunæ are irregular in size and shape, communicate freely with one another, and are lined with a layer of flat epithelial scales. The solid or trabecular element consists mainly of connective tissue, in which the ramifications of small vessels and unstriped muscle are sometimes contained. Angiomata are often congenital, and are entirely free from malignancy.

g.—Lymphatic Tumours, or Lymphomata.

The important relation which subsists between the lymphatic vessels and glands, on the one hand, and morbid proliferation of tissue, on the other, has already been explained. We have shown that, when inflammatory processes are taking place in any part, the nearest lymphatic glands tend very soon to get inflamed; that, if the local inflammation has specific characters, the resulting affection of the lymphatic glands shares in these characters; and that, in all cases of malignant tumour, it is the neighbouring lymphatic glands which, next in order of sequence, become the seat of malignant growth. So that, in fact, in the morbid proliferations of these bodies, we have an epitome of the morbid proliferations of the whole organism; and to describe their tumours would be equivalent to writing a complete treatise on tumours. What is meant, however, by the term lymphatic tumour, or lymphoma, is an hypertrophy or hyperplasia of lymphatic structure, and the new formation of similar structure in parts where normally lymphatic organs have no existence. Under the name 'lymphoma' may be included two perfectly different morbid conditions:—the one, an abnormal development of lymphatic vessels, or *lymphangioma*; the other, an abnormal development of lymphatic gland-structure, or *lymphadenoma*.

i. It is doubtful if *lymphangioma*, as an independent morbid growth, has any existence. There are many cases, however, in which enlargement, and possibly over-development, of lymphatic vessels forms an important ingredient in the morbid conditions which are present. Virchow has shown that, in elephantiasis Arabum, hyperplasia of the

connective tissue is largely associated with a dilated and hypertrophic state of the lymphatic vessels, and especially of the lymphatic spaces in which they originate. This change seems, however, to be secondary to obstruction of the lymph-paths through the inflamed lymphatic glands, to which the dilated tubes converge. In congenital hypertrophy of the tongue and lips, the same authority has pointed out the presence of a similar condition of the lingual lymphatic vessels. Further, cases are occasionally observed, in which the penis and scrotum, or corresponding parts in the female, or the lower part of the abdomen, or the thigh or leg, are thickened and brawny; and in which groups of depressed vesicles appear here and there, and, rupturing from time to time, yield large quantities of pure lymph. Here, the hypertrophy of the skin and subjacent parts, and the formation of vesicles, are doubtless all due to dilatation of the lymphatics, and their distension with lymph—phenomena which probably are themselves secondary to some proximal obstructive disease.

ii. *Lymphadenoma.*—There are at least three morbid conditions of the lymphatic glands which, if we have regard only to anatomical characters, are extremely difficult, and often impossible, to distinguish from one another. These are—simple inflammatory hyperplasia, the so-called 'scrofulous' form of enlargement, and that morbid condition now generally known as lymphadenoma, or lympho-sarcoma.

Simple inflammation of lymphatic glands may be induced by causes acting directly upon them, but is much more commonly the result of irritation propagated to them along the lymphatic vessels. They enlarge and get painful, assume a homogeneous aspect and a yellowish or faint rosy tinge, and under the microscope are found to differ but little from healthy glands—their enlargement being due to simple hyperplasia of their cell-elements, or leucocytes, and hypertrophy of their reticular connective tissue. Lymphatic glands thus affected may suppurate, or undergo other of the changes which are apt to follow on inflammation; but their general tendency is to resolution.

The term '*scrofulous*' is commonly applied to the slow and painless enlargement of groups of lymphatic glands, which occurs for the most part in children, and almost invariably ends in the destruction of the glands by an imperfect kind of suppuration. Scrofulous glands are generally met with in either the neck, thorax, or abdomen, and are commonly limited to one of these regions. Indeed, in the neck, where their progress can best be followed, we often see that the enlargement commences in one gland only; that the glands in the vicinity are successively affected, and often at long intervals; and that, after a while, the morbid process ceases with the destruction of all the implicated glands—those on the opposite side of the neck possibly remaining all the time perfectly healthy. In the earlier stages of this affection, the glands differ little, either to the naked eye

or under the microscope, from such as are simply hyperplastic from inflammation; but they tend soon to become opaque, yellow and friable—to undergo caseous degeneration. This change commences in the central parts, and gradually involves the whole mass, which presently breaks down into a semi-fluid detritus and thus forms the imperfect pus previously adverted to. Occasionally the caseous lump dries up, earthy salts are deposited in it, and it becomes an inert earthy concretion. There is a good deal of vagueness in the sense in which the term 'scrofulous' is generally employed. It is taken for the most part to imply that the morbid process, to which we attach it, is dependent on some peculiar condition of the constitution, and further that there is some close affinity, if not actual identity, between it and tubercle. But the so-called 'scrofulous glands' are certainly not tubercular; and, although their appearance is sometimes followed by that of tubercle, in a very large number of cases no such sequence is observed. And as regards cachexia, it is certain, that 'scrofulous glands' often develop in persons who appear in all other respects in the best of health; and further (if we may judge by the limitation of the morbid process), that if we admit their dependence on a pre-existing state of cachexia, that cachexia must in many cases be limited to a definite part or district of the organism. It is well known, however, that when a single gland has undergone scrofulous proliferation, there is a remarkable tendency for the morbid process to spread thence to other glands, in its immediate neighbourhood, and thence again to others; it seems in fact to spread from gland to gland, through the agency of some infective material, which the diseased organs evolve. It is well known also, that scrofulous enlargement of the glands of the neck not unfrequently follows upon certain diseases affecting the throat, such as mumps, diphtheria, and scarlet fever. Now, basing his arguments upon such facts as these, Virchow maintains (and we think with reason) that scrofulous proliferation of lymphatic glands, like ordinary inflammatory hyperplasia of the same organs, is always secondary to some peculiar process going on at the mucous surface, or other part, which is in direct relation with them by means of the lymphatic vessels—that scrofulous disease, of the glands of the neck is traceable to some inflammatory condition of the throat, fauces, or contiguous parts; of the bronchial and mediastinal glands, to pulmonary or bronchial inflammations; and of the mesenteric and retro-peritoneal glands, to similar conditions of the alimentary canal. He considers that there may be some specific quality or element in the primary inflammation, and a tendency in its products to undergo rapid decay, similar to that which characterises the morbid products of the diseased lymphatic glands; but that generally they are not recognisable, from the fact, that in this case the cells are mostly developed at a free mucous surface, and are speedily shed from it. But he considers, further, that there may be some special aptitude or weakness, con-

genital or acquired, in the lymphatic glands of certain persons, or of certain parts of them, which makes their inflammations, induced by indifferent causes, assume the scrofulous character.

The affection now generally known as '*lymphadenoma*,' differs but little anatomically from the morbid conditions which have just been described. It is characterised like them by a simple overgrowth of lymphatic tissue—that is, by a development of cells, which essentially resemble ordinary leucocytes, in the meshes of a trabecular tissue like that of normal lymphatic glands. The cells here, as in healthy glands, are so abundant that, in an unprepared section, they conceal all other elements; but if they be removed by pencilling or washing, the fibrous matrix and vessels come into distinct view. The lymphatic glands in this affection, and other parts which become implicated, rapidly increase in bulk, acquire for the most part an opaque milky aspect, soften, and yield, like carcinoma, a milky juice. They are liable also to fatty and caseous degeneration, and to be the seat of hemorrhage. There are two ways in which lymphadenoma tends to produce important and characteristic results—the one by generalisation, the other by modifying the quality of the blood.

Lymphadenoma is generally distinguished, from both simple inflammation and scrofulous proliferation, by the following important facts:—first, that the morbid process tends pretty rapidly to involve the lymphatic glands distributed throughout the organism; and second, that there is a disposition to heterologous development of identical morbid gland-tissue in situations in which normal gland-tissue has no existence. In other words, lymphadenoma must be looked upon as a variety of malignant disease, in which the secondary as well as the primary growths assume the microscopical characters of lymphatic tissue. It should be remarked, however—in the first place, that by lymphatic or adenoid tissue is not meant the whole complicated organism of lymphatic glands, but merely that comparatively simple arrangement of reticulated fibres and leucocytes, which is found in the solitary intestinal glands, and in the Malpighian bodies of the spleen; and, secondly, that the recent investigations of several German physiologists, and of Dr. Burdon Sanderson in this country, have shown that lymphatic tissue is very abundantly distributed throughout the body (amongst other places in the subserous tissue, in the submucous layer of the intestine, and along the bronchial tubes and the hepatic ducts) and that hence arises a possibility that, notwithstanding the diffusibility of lymphadenoma, its heterologousness and malignancy may, in the strict sense of these terms, only be apparent. Lymphadenoma not unfrequently affects the bronchial and mediastinal glands; and it may extend thence, along the connective tissue which invests the bronchial tubes, into the substance of the lungs, or may invade the parietes of the heart, insinuating itself between its muscular fibres, without necessarily forming any distinct tumour. The mesenteric glands also are often

chief seats of the disease; which is then apt to transgress their limits, to involve the substance of the mesentery, and to creep thence into the intestinal walls, which consequently become thickened in all their layers, and probably at length present flat tubercular elevations on both the mucous and serous surfaces. The liver, spleen, and kidneys also are peculiarly liable to suffer. Here, as in the heart, the growth tends rather to infiltrate the tissues than to form defined and independent tumours. In the fresh condition, the affected tracts of these organs present an opaque milky aspect, which may be in striking contrast with that of the surrounding healthy parts; and if they abut on the surface they probably form a slight convexity there. When however the contrast of colour has been impaired or lost by maceration, it is sometimes impossible by the naked eye alone to distinguish the healthy from the diseased parts. In the spleen, the microscopical characters of the morbid growth are almost identical with those of the healthy gland-tissue; in the liver and kidneys, however, the growth infiltrates the texture of the organs, and separates their proper elements from one another. In the kidneys especially this may be well observed; for the lymphoid growth spreads through the intertubular tissue of the organs, separating the still healthy tubes and Malpighian bodies from one another, until at length they appear to be sparsely distributed in a nearly homogeneous mass of adventitious cell-growth.

The other special characteristic of lymphadenoma is its tendency to influence the quality of the blood. Knowing as we now do that the lymphatic glands and the spleen, and probably also lymphatic tissue, wherever it may be situated, are the laboratories in which the corpuscular parts of the blood are manufactured, and further that they are the media through which, in chief measure, the elements and the products of local morbid processes are thrown into the system, we should naturally expect that anything which impairs or modifies their functions would soon lead to impairment or modification of the quality of the blood, and hence to various affections of the general organism. The influence of the morbid glands in lymphadenoma over the constitution of the blood is, however, quite special. Some years ago now, Dr. Hughes Bennett and Professor Virchow discovered almost simultaneously that in certain cases of morbid enlargement of the lymphatic glands or spleen, the circulating blood was characterised by containing a comparatively small proportion of red corpuscles, and a comparatively large number of leucocytes. These observations have since been confirmed and extended by many pathologists, and by Professor Virchow himself. And it may now be regarded as established that, in the disease under consideration—lymphadenoma—the blood gets gradually and permanently deteriorated in quality by the addition to it, from the diseased lymphatic glands, of the morbid leucocytes which they produce—leucocytes which microscopically differ little from those

proper to the blood, but fail to undergo conversion into red corpuscles; and that hence the red corpuscles diminish in number, while the white corpuscles accumulate, until finally, in extreme cases, they outnumber them. This change in the blood is attended with increasing pallor of that fluid, which reveals itself by increasing pallor of the skin and mucous membranes; and has obtained for the disease in which it occurs the name of *leukæmia* or *leucocythæmia*.

Cases in which leucocythæmia is present may be divided into three classes:—in one the disease producing it is limited to the lymphatic glands; in another it is splenic only; and in a third both spleen and lymphatic glands are implicated. Virchow distinguishes lymphatic from splenic leucocythæmia by the circumstance, that in the former the white corpuscles are generally smaller than normal white corpuscles, while they contain for the most part solitary and comparatively large nuclei; whereas in the splenic form of the disease, the leucocytes more closely resemble those of the normal blood, being equally large with them, and generally containing two or more nuclei of small size, which become obvious under the influence of acetic acid. When the spleen and lymphatic glands are simultaneously affected, both varieties of leucocytes may be discovered mingled in the blood. Lymphadenoma may be present, however, without the co-existence of leucocythæmia.

h.—Tubercle and Granuloma.

The latter term has been employed by Virchow to include the specific growths of syphilis, lupus, elephantiasis Græcorum, and farcy, because anatomically they differ but little from ordinary granulation-tissue, and it is often difficult to decide from mere inspection whether such growths are tumours or mere inflammatory products. Tubercle he regards as a species of lymphoma; and many pathologists are of the same opinion. It is by no means improbable that they are right, and certainly weighty argument may be adduced in favour of their view. On the other hand, many good observers by no means admit the adenomatous nature of tubercle. And, therefore, since it is generally acknowledged to be closely allied to gummata and to the tubercles of farcy, it seems reasonable to classify it, at all events provisionally, with these latter affections.

i. *Tubercle.*—From the time of Laennec down to within a very recent period, tubercle was regarded as a mere exudation or deposit from the blood, consisting in large measure no doubt of cells, but of cells which were degenerate from the beginning and never had any vitality; and it was recognised as occurring in two forms—one, the grey granulations or miliary tubercles (hard, greyish, translucent bodies, varying from the size of a small pea downwards, and tending to become opaque, yellow, and soft or friable internally); the other,

the so-called 'crude' tubercles, which are generally of larger size, of a nearly uniform opaque buff colour, and friable or cheese-like in consistence, but which were commonly believed to take their origin in the general caseous conversion of grey tubercles, and therefore to represent a comparatively late stage of the tubercular process. It is now, however, generally admitted—that tubercle is no mere deposit, but on the contrary, equally with sarcoma and carcinoma, a living growth, consisting essentially of cells, but having, above all other growths, a tendency to undergo rapid degeneration and death, and especially that form of degeneration which is termed 'caseation;' that the grey semi-transparent material which often forms the whole bulk of miliary granulations, and may often be recognised at the periphery of larger masses, is alone living and growing tubercle; and that the yellow caseous substance which has frequently been taken for its essential part is merely effete and dead matter, often no doubt tubercular in its origin, but often also the detritus of quite other kinds of cell-growth.

Grey granulations take their rise in the connective web of most organs and of many tissues, and, as will presently be shown, not improbably affect specially the lymphatic tissue distributed throughout the organism. They are common in serous membranes and in the pia mater, and it is probably here that their development may best be studied. If a minute tubercle from one of these situations be placed under the microscope, it will be found to consist mainly of an aggregation of cells, mostly of small size and of the embryonic character, of which those towards the centre will probably even now be angular, withered, and opaque from granular fatty deposit. A close examination will reveal other facts:—the growth will be found almost certainly to have taken place in connection with some minute vessel, probably to encircle it; and further, beyond the margins of what may perhaps be regarded as the actual growth, a zone of connective tissue will be recognised in which hypertrophy and proliferation are commencing—the plasmatic cells being larger than those of the normal tissue, and in many instances containing in their interior broods of two, three, or more secondary cells. It would seem, therefore, that the morbid process commences with proliferation of the connective-tissue elements of the adventitia or outer wall of blood-vessels, that it gradually involves more and more of the neighbouring connective tissue, and that as it spreads at the margins the central parts fall rapidly into decay. It follows that the chief microscopic elements of tubercle are—first, simply enlarged connective-tissue corpuscles (fusiform and stellate); second, these same cells containing two or more new cells within them; and third (and probably far most abundantly), small shrivelled granular embryonic corpuscles. But during the last few years it has been distinctly ascertained that, although neither peculiar to tubercle nor essential to it, certain cells presenting remarkable characters are commonly to be found, either in the centre of elementary

tubercles, or distributed in the peripheral parts of agglomerated tubercles. These are large irregular branching bodies, termed 'giant cells,' of which each contains from twenty to forty distinct nuclei. Their source is not clearly determined. In some cases possibly they result from the fusion of smaller cells; but M. Brodowsky has recently shown it to be probable that, at any rate, some of them are to be regarded as morbid modifications of protoplasmic buds from the walls of vessels, which, under other circumstances, would have become vessels. It should be added, that new blood-vessels seem never to form in the tubercular process; that no higher stage of development, in fact, than the mere over-production of new cells of a low grade of organisation is ever attained; and further, that the vessels around which tubercles form become at a very early period obstructed by the coagulation of fibrine, and the accumulation of leucocytes, in their interior. The intercellular substance of tubercle is, in the first instance, that of the particular form of connective tissue in which it originates; it soon, however, gets scanty and indistinctly fibrous or granular. Rindfleisch has described a reticulated connective tissue, in the meshes of which the corpuscular elements are contained—an arrangement, in fact, almost identical with that which obtains in adenoid tissue, and which, if generally present, goes far to confirm the views of those who regard tubercle as an adenoid growth. It is extremely difficult, however, to satisfy oneself of the presence of any such fibrous stroma, and MM. Cornil and Ranvier distinctly deny it. They admit that a kind of reticulum, probably of artificial production, may be recognised in sections which have been hardened with chromic acid or with alcohol; but they assert that it never contains protoplasmic particles (as lymphatic stroma does) at the points where the fibres intersect, and moreover that in the unprepared tubercle it has no visible existence.

But even if tubercular growths be not, like lymphadenomatous tumours, mere overgrowths or reproductions of modified lymphatic-gland structure, there can be no doubt at all that they are in very large proportion adventitious growths originating in lymphoid tissue. At all events, many physiologists, and particularly Dr. Sanderson, have shown satisfactorily that adenoid tissue is far more generally distributed throughout the body than was formerly suspected, and that it is especially abundant in all those parts in which tubercle is most frequently developed; and indeed, as regards tubercles produced experimentally, Dr. Sanderson seems to have clearly demonstrated their origin in hyperplasia of these normal lymphatic accumulations. We need hardly quote, in favour of this doctrine, the fact of the frequent development of tubercle in the lymphatic tissue of the solitary and agminated glands of the intestines, and in that of the spleen and lymphatic glands. We will discuss two cases, however, which Dr. Sanderson has specially investigated, in the course of his experiments on the artificial production of tubercle. In the first place, he has shown that in the

peritoneum, as indeed in all serous membranes, small masses of adenoid tissue are distributed abundantly, in some cases unconnected with vessels, but more commonly adherent to their walls, or encircling them, or even investing whole groups of capillary vessels; he has also shown that, in animals dead of acute peritonitis, all these masses have become soft, tumid, and enlarged; and further that, when tuberculosis is in progress, it is in them and by the multiplication of their cells, rather than by that of connective-tissue corpuscles, that miliary tubercles are gradually developed. Secondly, as regards the lung, it is now generally held that grey tubercles originate in the matrix of the organ, and not, as was formerly believed, within the air-cells; and it is generally admitted, we believe, that the part which they chiefly affect is the connective tissue surrounding the bronchioles at the point at which these lose themselves in the air-cells, and that the growth of tubercle-cells gradually extends thence into the substance of the tissue, which separates the air-cells from one another, and limits each pulmonary lobule. Now, according to Dr. Sanderson, there always are normally, in the situation here indicated, masses of adenoid tissue, and the early stage of pulmonary tuberculosis consists in a kind of hyperplasia of such masses.

The frequent connection between tubercle and adenoid tissue must be admitted. Nevertheless it is certain that, like lymphadenoma, tubercle does not take its origin exclusively in adenoid tissue. The general result, deducible from recent observations with respect to the genesis of tubercular products, seems to be that, like the products of inflammation, they are not derived from a single source—that they are not the results of specific hyperplasia of connective-tissue corpuscles alone, as Virchow teaches, nor yet simply overgrowth of the lymphatic cells of adenoid tissue—but that they are probably derived, in varying proportions, from both of these sources, from the other cellular elements which happen to form part of the affected tissue, and even from immigrant leucocytes.

The view here expressed has an important bearing on the question 'what is and what is not to be regarded as tubercle?'—a question of the highest interest, in reference to the status of the morbid condition of lung commonly known as 'pulmonary phthisis,' and to the nature of closely related, if not identical, morbid conditions of other organs. According to views generally accepted until within the last few years, the grey miliary tubercle and the yellow cheesy tubercle (of which both are common in the lungs, and the latter occasionally, by coalescence, infiltrates large tracts) were regarded as being, not so much varieties, as different stages of the same disease; and it was held that, in the dead-house, all the intermediate conditions, by which the minutest miliary tubercles lead up to the most extensive caseous infiltration, can be readily recognised. At the present day, Virchow and many other distinguished pathologists deny this relation, and maintain that caseous disease, which comprises probably all the cases

recognised clinically as pulmonary phthisis, is of pneumonic origin—the consequence of catarrhal or lobular pneumonia. The grounds of this opinion are mainly, that in caseous infiltration of the lung, the presence of tubercular proliferation of the interstitial tissue is not a very obvious anatomical feature; and that the great bulk of the morbid mass consists of degenerate epithelial cells accumulated in the air-cells and smallest bronchial passages. Many important considerations, however, may be adduced in favour of the opposite view. It is a fully recognised fact that, even in undoubted examples of miliary tubercles, the proliferation of cells in the matrix of the pulmonary lobules, which constitutes their commencement, is soon attended with dense accumulation of cells, probably due to epithelial proliferation, within the pulmonary loculi. Now, unless we start with the assumption that tuberculosis consists in nothing else than proliferation of connective-tissue corpuscles, or of the elements of adenoid tissue; what right have we to assume that the protoplasmic bodies, which fill the air-cells, are specifically different from those which occupy the substance of the matrix? It is admitted that pus-cells may originate in epithelial as well as in other kinds of cells; why should tubercle-cells have a more exclusive parentage? It is a recognised characteristic of tubercle that its specific cells very rapidly fall into degeneration; but this is even more remarkable in the cells which fill the loculi, than in those which crowd the pulmonary matrix. Again, the caseous masses of pulmonary phthisis certainly do not occupy those parts of the lung, which either lobular or lobar pneumonia specially affects; but they do occupy those situations (mainly the upper portions of the lungs) in which miliary tubercles generally originate, and are most advanced. And, lastly, caseous tubercles in the lungs are constantly associated with tubercular formations elsewhere in the body, and indeed in those very parts in which generalised miliary tubercles are specially apt to manifest themselves. For many reasons, therefore, of which we have only indicated the more important, we are disposed to maintain the relationship between miliary tubercles and caseous infiltration, to regard them simply as varieties or different stages of the same disease, and to support the claim of 'pulmonary phthisis' or 'caseous pneumonia' to be called also 'tubercular phthisis.'

The recent investigations of Dr. Klein[1] and Professor Charcot[2] are strongly confirmatory of the views here advocated. Dr. Klein shows that, in miliary tubercles of the human lung, 'the first changes take place in the alveoli and inter-alveolar septa;'—that, as regards the alveoli, the epithelial cells become swollen, granular and detached, that 'they then proliferate, and that generally, either by their coales-

[1] 'On the Relation of the Lymphatic System to Tubercle.' *Report of the Medical Officer of the Privy Council.* New Series, No. 3, 1874.

[2] 'Revue Mensuelle de Médecine et de Chirurgie,' 1877, p. 876.

cence or by the disproportionate enlargement of one or more of them, each cavity becomes filled with a multinuclear lump of protoplasm or giant cell, which subsequently undergoes fibrillation, caseation, or other form of degenerative change; and that, as regards the inter-alveolar septa, these thicken with the growth of a tissue containing branched and spindle-shaped cells and a few lymphoid cells. He adds, that, at a somewhat later period, cords of adenoid tissue are formed upon the walls of the larger vessels in the vicinity of the tubercles. He further points out, in reference to tuberculosis of artificial production, that although the ultimate changes are identical with those just described, they take place in an inverse order—the development of the perivascular adenoid cords preceding the changes in the inter-alveolar septa and in the air-cells; and he concludes that, in artificial tuberculosis, the process commences from the arteries and veins, in the idiopathic affection from the pulmonary capillaries. Professor Charcot's observations are still more to the point, for he expressly shows that there is no essential, genetical, or structural difference between miliary tubercles of the lungs and so-called 'caseous pneumonia.' He point out, however, that in the latter variety of pulmonary phthisis the tubercular process commences in the *parietes of the bronchioles* where they lose themselves in the air-cells; and that softening takes place here before any trace of inflammation is visible either in the epithelial lining of the tubes or in the pulmonary lobules connected with them; and further, that when a caseous patch is examined, it presents a central degenerate area, and a marginal zone of embryonic cells, and scattered giant cells, infiltrating the normal tissues of the lung.

The quasi-malignant character of tubercle is generally admitted; although the fact, that it appears often to originate, almost simultaneously, in many points of one or more organs (in both lungs, for example), might seem to imply the existence, in some cases, of a widely-diffused tendency of organs to become tubercular, independently of specific infection. The proof of its malignant attributes lies, partly in that disposition to general diffusion which it shares with growths which are unquestionably malignant; and partly in the facts, that its local spread is due chiefly to the establishment of new foci of disease in clusters around the primary growths, and that the nearest lymphatic glands always become secondarily affected at an early period. It was considered by Laennec (and his view in a very slightly modified form has been advocated by Dittrich and Niemeyer) that a degenerate mass of tubercle—a caseous lymphatic gland, for example—is a common, if not the invariable, source of generalised tuberculosis—that the degenerate particles taken up by the blood become distributed by it, and then act as specific irritants to the parts which they infect.

The remarkable experiments, in reference to the production of tubercle by inoculation, first made by Villemin, and since repeated

and extended by Wilson Fox, Sanderson, Cohnheim, and others, have a very interesting bearing on the points considered in the last paragraph. Guinea-pigs and rabbits were inoculated with tubercular matter; and it was found that, after the lapse of some weeks, small indurated caseous nodules had become developed at the seat of operation; the next lymphatic glands had undergone hyperplastic enlargement, and the lungs, liver, serous membranes, and some other organs presented a greater or less number of small, grey, translucent, hard bodies, which accurately resembled the miliary tubercles occurring in man; and it was assumed that all these secondary formations were really tubercle, and that tubercular detritus taken up by the absorbents, and then distributed throughout the organism, had a specific influence in the production of tubercle. It was soon proved, however, that the inoculation of other forms of growth, or of decomposing healthy tissue, or of the products of local inflammations excited by mere mechanical irritants, was quite as efficient in generating general tuberculosis, as was the inoculation of tubercular matter itself. And hence it became obvious, that the exciting cause of the tubercular development was, not the matter which was inserted or applied locally, but the products of the inflammatory process which this matter evoked. The experiments failed, therefore, to prove the inoculability of tubercle, but they proved that tubercle might be produced locally by direct non-specific irritation, and that tubercle so engendered had the capacity for becoming generalised. To a certain extent then, these experiments may seem to favour the views of Laennec, and Niemeyer, as to the infective quality of caseous matter. It is more in accordance, however, with what is now known of morbid proliferation, and of contagium, to assume that the infective element of tubercle is not effete and dead material, but rather living (even though degenerating) particles of protoplasm.

The most common seats of tubercle are the lungs and the mucous membrane of the intestines. But tubercles are generally largely distributed throughout the bodies of those who die tuberculous; and we may enumerate as their seats of election, after the lungs and bowels, the serous membranes, the spleen, the kidneys and liver, the brain and its membranes, the mucous surface of the genito-urinary organs, the supra-renal capsules and the bones, and of course the lymphatic glands.

ii. *Syphilitic gummata* have a close anatomical affinity with tubercle, on the one hand, and with inflammatory products on the other. They resemble granulation-tissue, in the general character and arrangement of their cellular structure, and in the facts—that they are provided with permeable vessels, and that at an early period of their growth they are capable of conversion into cicatricial tissue. They tend, however, like tubercles, to undergo early caseation and death; and if their progress be not modified by medical treatment, this may be regarded

as their normal termination. It is in this latter condition, that they are almost invariably found *post mortem* in the liver, testicles, brain, bones, and other internal organs. They then form opaque, buff-coloured, toughish masses, imbedded in dense connective or cicatricial tissue. They are especially common in the skin and subcutaneous connective tissue, but here they generally undergo ulceration and leave indelible cicatrices. Excepting by their toughness, by the size which they attain, and by the paucity of their numbers, it would be exceedingly difficult to distinguish caseous gummata from tubercles in the same condition. In the brain and testicles especially, the resemblance between gummata and tubercles is remarkably close.

i.—Sarcomata.

The term 'sarcoma' was formerly applied to all tumours which were supposed to have a fleshy character, and hence came to be used indiscriminately, and to have no precise meaning. It is now, however, limited in its application to those growths which consist, not in their beginnings merely, but throughout the whole term of their existence, of embryonic tissue. Virchow regards them as belonging to the series of connective-tissue tumours which have already been described, and shows that the latter, especially when they undergo generalisation, tend to get more or less obviously sarcomatous,—that is, tend to become more and more exclusively cellular, and to lose more and more their several distinctive characters. Sarcoma differs structurally little, if at all, from simple inflammatory granulation-tissue; both of them consist essentially of embryonic cells, which in the first instance are small, and round, and separated from one another by the least possible quantity of intercellular substance; in both cases there is a tendency, as organisation proceeds, for the cells to grow fusiform or spindle-shaped while still retaining their embryonic characters; in both cases the anatomical and other features of the new-formed cells are modified, to some extent, according to the nature of the tissues in connection with which they arise; and in both cases the growths become abundantly vascular from the development of new vessels, the parietes of which are formed of cells, little if at all modified from those which constitute the general mass. They differ materially, however, in the fact, that inflammatory formations tend to subside or to form mere cicatricial tissue, while sarcomatous tumours maintain a continuous vitality of growth, present a wider range of variations from the primitive type of structure, and are in large proportion malignant.

Many varieties of sarcoma may be described. If it affects a bone, or an osseous tumour, or is attended in its progress with osseous transformation, we have what may be termed an '*osteo-sarcoma*'; and if, under analogous circumstances, we find sarcomatous growth associated with simple fatty or mucous or gliomatous tissue, we have tumours

which may be named respectively '*lipomatous sarcoma*,' '*myxo-sarcoma*,' and '*glio-sarcoma*.' Again, sarcomata may undergo fatty or calcareous degeneration, or mucous softening, and hence acquire special characters. The occurrence of degeneration, and especially of mucous softening, often leads to the formation of cysts; and thus arises that variety of sarcoma commonly known as '*cysto-sarcoma*.'

Sarcomatous tumours are often, and perhaps best, classified according to the characters presented by the cells which predominate in them —the presence of any of the modifications, which have been above indicated, then marking only subordinate divisions or varieties. There are at least four such species of sarcoma which we may briefly consider: namely (i.) round-cell sarcoma; (ii.) spindle-cell sarcoma; (iii.) large-cell sarcoma; and (iv.) melanoid sarcoma.

i. *Round-cell sarcoma*.—In this species the structure of the growth approaches nearest to that of ordinary granulation-tissue—the cells being small, round, distinctly nucleated, and separated by little intercellular substance. Such tumours are nearly homogeneous, but soft and pulpy in texture, greyish or white in hue, opaque or slightly translucent, and (if they have been removed some hours from the body) yield a milky juice. They are very vascular, often attain enormous dimensions, and are malignant in a very high degree. They originate almost indifferently in all parts of the organism; but especially perhaps in the skin and subcutaneous connective tissue, in glandular organs, particularly the breast and testicle, in bones and muscles. They comprise most of the tumours which were formerly called 'medullary sarcoma' and 'encephaloid,' and many of those which were termed 'fungus hæmatodes.'

ii. *Spindle-cell sarcoma*.—In this case the growth consists of cells which have become elongated and fusiform, or spindle-shaped, and hence present a higher grade of development than those of the round-cell sarcoma. The cells vary a good deal in size, and contain each from one to two or three nuclei. They are arranged side by side in bands or bundles, which take a curvilinear course and cross one another in various directions; so that, on examining a microscopic section, we see round or oval groups of apparently round or oval cells, surrounded by bands of fusiform cells—the former being simply cell-bundles which have been cut across more or less obliquely. Spindle-cell sarcomata are harder and denser than round-cell sarcomata, greyish or white, slightly translucent, and of a more or less distinctly fibrous or lobulated character. They yield but little juice. They have a tendency to recur, and even to present malignant characters; but their malignancy is far less pronounced than that of round-cell sarcomata, and they rarely reach the size which these latter attain. Spindle-cell sarcoma is synonymous with 'fasciculated sarcoma,' and includes Paget's 'recurrent fibroid tumours.'

iii. *Large-cell sarcoma.*—In some cases the cells of sarcomatous tumours attain unusually large dimensions. The most characteristic example is that furnished by Paget's 'myeloid tumours' of bone. These originate only in bones, destroy them extensively, and grow to a large size. They are made up to a considerable extent of embryonic cells, both of the round and spindle-shaped varieties; but that which distinguishes them from all other forms of sarcoma, is the presence of a greater or less abundance of large cells containing many nuclei. These cells, which are obviously derived from the many-nucleated cells of the healthy medulla, present much variety. They may measure as much as the hundredth part of an inch in diameter, and thus be objects distinguishable by the naked eye; they may be round or oval, but generally are irregular, and present a more or less complex arrangement of buds or tails; and they may contain any number of nuclei between two or three and two or three hundred. They consist of masses of protoplasm, unbounded by distinct cell-wall, and with the nuclei imbedded in their substance. Although myeloid tumours have unlimited powers of local development, and even invade and grow along the veins, they are very rarely malignant in the true sense of that word.

iv. *Melanoid sarcoma.*—In this form of tumour the embryonic cells, which constitute it, are more or less loaded with minute pigment granules. The cells are round, oval or fusiform—generally the last—and separated from one another by a small amount of intercellular substance. Each contains one or two distinct oval nuclei. The pigment granules are roundish or angular, and separately might pass for oily or cretaceous particles; they are deposited chiefly in the extra-nuclear protoplasm, and sometimes in such abundance that the cell under the microscope appears black, and the nucleus is altogether concealed; but they are found also in the substance of the nucleus. Melanoid sarcomatous tumours are generally soft, and present—if large, a mottled sepia-brown or black appearance; if small, a more or less uniform black or brown hue. They take their origin almost invariably in structures which normally are pigmented, such as the choroid coat of the eye and congenital pigmented nævi; and when they become generalised, the secondary growths repeat the pigmented character of the primary growth, thus furnishing a good example of the tendency, which secondary growths always have, to reproduce the specific characters of the parent tumour. Melanoid sarcomata are generally highly malignant.

Closely related to the sarcomata, and by Cornil and Ranvier placed among them, is the growth termed by Virchow '*Psammoma*,' which occurs solely in connection with the membranes of the brain and cord. It is vascular, soft, and friable, and chiefly characterised by an abundant development of concentric earthy concretions surrounded with

capsules of flattened cells or scales. The type of these tumours is furnished by the choroid plexus. They rarely attain a large size, and probably never cause mischief unless they be large.

j.—Carcinomata, or Cancers.

Cancerous tumours are considered by Virchow to be of a higher type than any which have hitherto been considered. He regards them, not as the more hyperplastic condition of a single structural element, but as consisting of a combination of tissues, so arranged as to present some of the distinctive characters of an organ; and he includes them, therefore, in his class of 'organoid tumours.' They are composed of a fibrous framework, or stroma, so arranged as to form a series of loculi, and of groups of cells which are contained in dense masses within them. The stroma consists for the most part of ordinary fibrous tissue and plasmatic cells, and carries and supports the arteries, veins, and capillaries, which are sometimes very abundant; it may be extremely dense or comparatively lax, and varies much in quantity relatively to the size and number of the spaces which it invests. The loculi differ in size, and on casual examination seem to be round or oval, and unconnected with one another; but as a rule they communicate freely, and form a series of branching channels. The cells are said by Virchow, and by many others, to be of an epithelial character; and they are so far epithelial, that they are developed from the surface of the loculi, are in absolute contact with one another, have no intervening cement, and are never traversed as granulation-tissue is by vessels. They vary greatly in size, and on the average are considerably larger than those of sarcomatous growths. They vary even more remarkably in form, and indeed their polymorphous character is often regarded as typical of their carcinomatous nature. They may be round or oval, or from mutual pressure polyhedral; but more frequently they are of very irregular form, presenting convexities or concavities upon their surface, and projecting here and there into flattened, pointed, bulbous, or nondescript processes. They consist of masses of protoplasm, more or less granular and often fatty, and containing within them one or more nuclei, which are for the most part round or oval, of comparatively large size, and exceedingly well defined. Moreover they not unfrequently become vacuolated, or hollowed out here and there into globular cavities, which are termed by Virchow 'physaliphores,' and are regarded by him as reproductive cavities. Cancer cells frequently have a close resemblance to the cells of the vesical epithelium. Cornil and Ranvier deny their truly epithelial character, mainly because as a rule they have no distinct cell-wall, and because, although in contact with one another, they generally do not cohere.

The origin of cancers, like that of all tumours in fact, is very

obscure. Rindfleisch, taking epithelial cancer as the type, considers that all forms of carcinoma originate in the hyperplasia of epithelial structures; which, as they grow, eat their way, as it were, into the subjacent tissues, hollowing them out into irregular cylindrical cavities, which then constitute the characteristic loculi of cancer. This mode of development calls to mind that of tubular glandular organs and hairs in the fœtus. Cornil and Ranvier, on the other hand, who expressly exclude epithelioma from true cancers, and consider cancer cells as being in no sense epithelial, conclude (mainly from their observations on the development of carcinoma in the bones and in the mammary gland) that the alveoli, within which the cells grow and multiply, begin in the plasmatic spaces or serous canaliculi, which are directly continuous with the lymphatic vessels, and that even when they attain their full size they maintain this connection: so that in a sense the alveoli of cancer may be regarded as the dilated origins of lymphatic vessels. To this connection, moreover, they attribute the peculiarly malignant character of all forms of carcinoma. Under any circumstances, however, the early stages of cancer are generally marked by the formation of embryonic tissue—of cells, therefore, differing little from those which are found in inflammatory processes and in sarcomatous growths. But soon differentiation takes place, and the specific character of the growth is revealed by the conversion of some of these cells into the fibrous tissue of the stroma, and of others of them into the epithelium-like cells of the loculi. There is good reason, nevertheless, for considering that the matrix in many cases, and in some perhaps almost exclusively, consists of the normal fibrous elements of the part affected, which have simply undergone some degree of thickening and overgrowth; just as in other cases, where glandular organs are involved, their follicles and ducts may be stimulated to unwonted development, and so form prominent objects in the field of the microscope, without necessarily constituting any essential part of the specific growth.

Like other adventitious growths, but in a greater degree than most of them, carcinoma is liable to undergo degenerative changes; these involve principally the cellular elements, and are sometimes so uniform in their occurrence as to give a special character to the case in which they prevail. Fatty degeneration of cells is the most common; but we meet also with caseous degeneration, calcareous deposit, and mucous softening; and not unfrequently extravasation of blood takes place, owing to the rupture of the morbid capillary vessels.

All kinds of carcinoma are malignant,—the most malignant being the soft or encephaloid form, with its pigmentary and other varieties; the least malignant being epithelial cancer, which speedily involves the neighbouring lymphatic glands, but is very rarely reproduced in other parts of the system.

The chief varieties of carcinoma are (i.) *Scirrhus* or hard cancer;

(ii.) *Encephaloid* or soft cancer; (iii.) *Colloid* or mucous cancer; (iv.) *Epithelioma* or epithelial cancer; and (v.) *Adenoid* or tubular cancer.

i. *Scirrhus*, in its typical form, is known especially by its hardness and slowness of growth. It creaks on section, and its cut surface presents a white or greyish, glistening, fibrous character, and yields a little milky juice on scraping. Its density and hardness are due to the great abundance and thickness of its fibrous matrix, and to the comparatively small size and number of its cell-containing loculi. The cells, however (which constitute the essential element of the milky juice), present the ordinary characters of cancer-cells. Scirrhous tumours rarely if ever undergo complete cure; yet it is certain, not only that they are of slow growth, but that their progress is specially apt to be attended with the degeneration (chiefly fatty or caseous) and the subsequent disintegration and removal of the cells of considerable tracts, and the consequent disappearance from such parts of everything except the fibrous stroma. Scirrhus is equally characterised by the slowness with which it obviously involves the neighbouring lymphatic glands, and becomes generalised. It invariably, however, sooner or later manifests the infectious qualities which belong to it.

ii. *Encephaloid cancer* is very soft in texture and rapid in growth, yields a very abundant milky juice, presents a tolerably uniform opaque white sectional surface, which, however, may be variously studded with patches of congestion or hemorrhage, of fatty or caseous degeneration, or even of pigmentary deposition. Its extreme softness is due to the fact, that the fibrous stroma forms a very small proportion of the whole mass, while the cells are relatively very abundant. The alveoli differ in size, but are generally comparatively large, and their walls exceedingly delicate; indeed, it is often difficult to recognise the latter at all, unless the cells be first removed by washing or pencilling. In encephaloid cancer, the secondary involvement of the nearest lymphatic glands, and of the general organism, takes place very speedily.

Several well-marked varieties of encephaloid cancer are met with, two or three of which may be here enumerated. These are,—first, *erectile* or *hæmatoid carcinoma*, in which the vessels (always abundant in encephaloid) are extraordinarily developed and tend to frequent rupture; second, the variety, which Cornil and Ranvier term '*pultaceous carcinoma*,' in which the alveoli are thicker-walled than in most forms of encephaloid, so large that they can easily be recognised by the naked eye, and from which the contents readily escape as a thick pulpy juice; third, *lipomatous carcinoma*, wherein the cancer-cells even from their infancy are loaded with oil, and in the adult state present so general and large an amount of it that, both to the naked eye and under the microscope, the tumour has (at first sight) a con-

siderable resemblance to ordinary fat; fourth, *melanotic carcinoma*, in which, as in the corresponding form of sarcoma, the cells are pigmented.

iii. *Colloid cancer* has a very close resemblance to myxoma. In both cases the tumours are more or less transparent, and gelatinous in consistence, and in both yield from the cut surface an abundant juice, which is transparent, glairy, and characterised by containing mucine. The fundamental anatomical distinction between them is this,—that, whereas in myxoma the framework of the tumour consists of plasmatic cells, the mucous fluid and vessels occupying the interstices between them; in colloid carcinoma, the mucus arises in the degeneration of the essential cells of the growth, the general solidity of the tumour being due to the fibrous stroma, which forms the walls of the alveoli. In colloid cancer the alveolar structure is extremely well-marked, and on this account colloid has often been termed 'alveolar cancer.' The alveoli are so large as to be easily visible to the naked eye; they are round or oval on section; and when the growth forms a projecting mass on a serous surface, their aggregation presents the appearance of an accumulation of small bubbles of air in a viscid fluid. They communicate freely with one another. Their walls are mostly extremely thin and delicate, displaying a fibrillated structure with an indistinct development of fusiform cells, which may themselves present indications of fatty or colloid degeneration. The glairy contents of the alveoli vary—from the consistence of white of egg up to that of pretty firm glue, from pure white to a more or less deep yellowish, brownish, or reddish hue, and from perfect transparency to tolerably complete opacity. Microscopically, cancer-cells can always be recognised. The smaller alveoli of the newly-developed parts are probably full of well-defined cells, of which some already contain globules of mucus. As, however, the growth gets older and the alveoli larger, the cells undergo more and more complete mucous degeneration, swell up, and presently disintegrate; and thus in many cases the alveoli get distended with mucus, presenting a certain amount of granular matter, mostly arranged in irregularly concentric circles, with here and there perhaps the ghost of a huge dropsical cell. In addition to the mucous conversion, which is the especial feature of colloid cancer, a certain amount of fatty degeneration is common. Calcareous deposition also is not unfrequent.

Although colloid cancer is certainly malignant, and affects lymphatic glands, and occasionally becomes generalised, it is specially characterised by a tendency to spread in area, and to implicate the tissues immediately subjacent to that area. Thus, when arising in the peritoneum, it soon diffuses itself over the greater part of that membrane, and also soon involves, in many situations, the whole thickness of the stomachal or intestinal walls. It shows also (though it is not peculiar

in this respect) an obvious proneness to spread along the lines of the lymphatic canals and capillaries.

iv. *Epithelioma*, or cancroid, is a very characteristic form of growth, originating, but not quite exclusively, in epithelial tissue, and characterised by a very abundant formation of epithelium in cavities or loculi of considerable size, which, as in other forms of carcinoma, communicate more or less freely with one another.

The commonest form of epithelioma is that which arises in the skin, and those mucous surfaces which are in relation with the external orifices, namely, those of the lips, tongue, œsophagus, anus, vagina, and uterus. It forms a tumour which varies in size, soon ulcerates, and on section presents (owing partly to the fact that the tissues which it invades are not yet wholly destroyed by it) a more or less variegated character; it is friable in texture, somewhat granular, and yields on pressure or scraping, not a juice, but rather an opaque, whitish, granular pulp. The stroma of the growth consists of fibrous or of embryonic tissue, including vessels, and more or less abundant traces of the original healthy structures. The pulp which exudes, and the contents of the loculi, consist solely of cells in different stages of development. These are distinctly nucleated, modified in shape by mutual pressure, and for the most part large and strikingly epithelial in character. The younger cells are in relation with the stroma, and occupy therefore while *in situ* the periphery of each cell-mass; the others are arranged in a more or less stratified or confused manner within. But we find additionally, in the latter situation, knots or nests or involucra of cells, the presence of which is almost conclusive as to the nature of the growth. These consist of large flat cells, arranged in concentric circles around a group of cells, or even a single cell, of smaller size, of plumper form, thick-walled, and containing a nucleus, together with perhaps some mucous or colloid material, or a few small fat globules. At first sight, these nests look not unlike transverse sections of cutaneous papillæ, but they obviously differ from them in the fact that their centres are made up simply of cells, and not of stroma containing vessels.

As regards the development of epithelioma, there is little doubt that when it occurs at epithelial surfaces, it commences with hyperplasia of the deeper-seated embryonic cells of the epithelium:—in the skin, therefore, with hyperplasia of the cells of the rete mucosum and of the sebaceous and sudoriparous glands; in the mucous surfaces, with hyperplasia of the corresponding cells of their epithelium, and of the glandular crypts. These multiply, become modified in form and arrangement, distend the cavities or depressions in which they lie, and send thence into the immediately surrounding tissues bud-like processes. The latter increase in number and size, and thus gradually invade and destroy the neighbouring textures. Rindfleisch quotes an

observation, and reproduces a drawing of Köster's, which seem to show that the extension of epithelioma is due to the involvement of the lymphatic networks—that the budding or sprouting epithelial processes above adverted to, instead of forming indiscriminately, penetrate the capillary lymphatics, run along them and distend them. There is probably some truth in this view; and, if so, it assimilates the local spread of epithelioma with that of colloid cancer, and especially with that of scirrhous and encephaloid cancer, as described by Cornil and Ranvier.

Epithelial cancer is undoubtedly the least malignant of all the varieties of carcinoma, for it is the only cancer which admits of being removed in its early stage with the tolerable certainty that it will not recur; and although it soon involves neighbouring lymphatic glands, it rarely manifests itself secondarily in other internal organs.

v. *Adenoid* or *tubular cancer*, otherwise termed 'columnar,' or 'cylindrical epithelioma,' is a rare affection, said generally to originate on some mucous surface, and to involve secondarily lymphatic glands and other organs. It occasionally, however, arises primarily in the liver or other parenchymatous organs. It forms tumours of various sizes, which have a close general resemblance to those of encephaloid cancer. They are highly vascular, soft, and yield an abundant milky juice. Microscopically, they are seen to consist of a system of tubules irregularly arranged, and separated only by a very small quantity of fibrous stroma; and bear a striking resemblance to sections of the cortical substance of the kidney deprived of malpighian bodies. The tubules are generally cylindrical, of tolerably uniform size, and lined with a layer (usually single) of spheroidal or columnar epithelium. They present, for the most part, a distinct central cavity or canal.[1] This form of carcinoma is highly malignant.

B. Atrophy, Degeneration, and Necrosis.

1. *Atrophy and Degeneration.*

The term 'atrophy' means strictly mere diminution in the bulk of tissues from deficient nourishment. The term 'degeneration,' on the other hand, implies degradation of tissue—in other words, a qualitative rather than a quantitative change. A part which suffers atrophy simply wastes, while one which undergoes degeneration often presents an actual increase in bulk. Yet, although atrophy and degeneration imply, so to speak, different lines of decay, these conditions are so constantly associated that, in a practical sense, they scarcely admit of separation.

[1] See Dr. Greenfield's account of a case of this disease in vol. xxv. of the 'Path. Trans.'

When degeneration is in progress, we find that the elementary constitution of the parts involved gradually becomes confused and destroyed, and that accompanying this process fat, pigment, or other matters, which normally have no visible existence in them, are deposited in a globular or granular form. Whence do these matters come? Are they simply due to the decomposition of the highly organised material which has undergone degeneration, and to the precipitation of its more insoluble constituents; or do the decaying tissues attract them to themselves from the blood or extra-vascular nutrient fluid? There can be no doubt that both of these processes take place; and that, although they are distinct and not unfrequently dissociated, they generally concur. In most cases, where degenerative products are visible, they are due partly to simple precipitation, partly to infiltration.

It will thus be understood that degeneration, in its widest sense, involves three processes which are essentially distinct from one another:—namely, first, simple atrophy or wasting of tissue; second, degeneration proper, or the decomposition of tissue; and, third, the deposition in the affected parts of insoluble matters derived from without; and that these processes are generally associated, although in very various proportions. It should be added, that the visible products of degeneration (according to the nature of which different names are given to the various degenerative processes) are only the more insoluble products of these processes; that other effete or degraded matters are produced simultaneously, which are probably just as important, although more difficult to recognise, partly on account of their solubility, partly because they assume no crystalline, molecular, or other visible form. We shall discuss the generally-recognised varieties of degeneration *seriatim*.

a. Cloudy Swelling.—When cells are exposed to the direct influence of certain poisonous substances, or when they soak in the dropsical or inflammatory fluids which escape from the blood, they often get distended from imbibition, and at the same time their protoplasm assumes a very finely granular condition. The same changes, according to Cornil and Ranvier, take place in the nuclei and nucleoli. Virchow regarded them as the result of nutritive irritation. But they are now generally admitted to be of a degenerative nature, or at all events passive, and in many cases a first step towards fatty degeneration. The granules, however, are not fatty but albuminous, and readily dissolve in acetic acid. Cloudy swelling is well shown by the hepatic cells, in cases of acute atrophy of the liver.

b. Mucous and Colloid Degeneration.—In many cases cells, and in some instances intercellular substances, undergo softening and conversion into matters which are known as 'mucus' and 'colloid.' These

may form a thin glairy fluid, or present all degrees of viscidity between this and a thick jelly; and may be transparent and colourless, or of different tints of yellow, brown, or red. They have, therefore, a very close resemblance to one another; and, indeed, are not always easy to distinguish. They differ chemically in the fact that mucus contains mucine in solution—a substance which is precipitated by acetic acid; while the specific element of colloid is an albuminous substance which is not affected by this reagent.

Mucous degeneration sometimes involves the intercellular parts of tissues, sometimes the cellular elements. Of the former case, we have examples in the mucous softening, which takes place in the matrix of enchondromata and of the cartilages of elderly persons, and perhaps also in myxomatous tumours. The latter case is exemplified in the formation of globules of mucus within the cells of mucous membranes, the consequent distension of the cells and their final deliquescence. Mucous degeneration is common in the cells of synovial and mucous surfaces; it is a characteristic feature of the progress of colloid cancer; and it is of not unfrequent occurrence in other morbid growths—leading to the formation of cysts.

Colloid matter is most frequently met with, in the cysts of the thyroid body, and in small renal cysts; and, like mucus, generally arises within cells, which it presently fills and destroys. In the cases just referred to, it forms rounded jelly- or glue-like masses filling the cavities, and containing imbedded in them the remains of the cells which gave them origin. It seems probable that the glassy transformation of voluntary muscles in typhoid fever, described by Zenker —and which is marked by a peculiar waxy lustre, the disappearance of the normal markings, and a tendency to crack transversely— is really an example of colloid degeneration. Further there is reason to believe that many so-called 'fibrinous' casts of the urinary tubules are rather colloid matter than fibrine.

c. *Lardaceous Degeneration*, known also as 'waxy,' 'bacony,' 'albuminoid,' 'amyloid,' and 'scrofulous' degeneration has (as the many names which have been applied to it testify) long been recognised, and presents many very remarkable characteristics. It occurs almost exclusively in cases of tertiary syphilis, chronic phthisis, and long-continued suppuration especially in connection with bone-disease; and indeed, since prolonged suppuration is constantly associated, with both the later stages of syphilis and chronic phthisis, there is some reason to regard the lardaceous change as the consequence essentially of suppuration. It affects mainly the liver, spleen and kidneys; which increase slowly to many times their original bulk, grow dense and homogeneous in texture and doughy in consistence, and present when cut a pale brownish tint, with a slight degree of translucency and a peculiar waxy lustre. Microscopically, the change is found to

be due to the infiltration of the walls of capillary and other small vessels, of the walls of ducts, and of the substance of cells, with a transparent, colourless, refractive material; which by its presence obliterates all their structural characteristics, gives them a jelly-like or vitreous aspect, and converts the vascular and duct walls into thick homogeneous hollow cylinders, and the cells, with their nuclei, into amorphous masses, with a tendency to irregular fracture. The microscopical appearances of parts which have undergone lardaceous infiltration are not altogether unlike those displayed by structures which are the seat of mucous or colloid change; but lardaceous matter is tougher and more consistent than mucous and colloid generally are; it invades structures which these latter never affect, and moreover never leads to the deliquescence and utter destruction of tissues. The chemical characteristics of lardaceous matter are of considerable interest. Virchow, some years ago, finding, as he thought, that on the addition of sulphuric acid and iodine it assumed a blue tint, concluded that it was identical with the cellulose of plants, which under the influence of the same reagents becomes first converted into starch and then blue; and hence he gave it the name of 'amyloid matter'—a name by which it is still largely known, although the theory which gave it origin is now only matter of history. It has been conclusively shown, indeed, that lardaceous matter has no sort of chemical relation with starch or cellulose, and that the addition of sulphuric acid and iodine produces a bluish tint only in consequence of the precipitation of the iodine in a molecular form. Lardaceous matter, in fact, is a modification of albumen, with a deficiency of potash and phosphoric acid, but with an excess of soda, hydrochloric acid, and especially cholesterine; and it is easily recognised by its rapid absorption of iodine, even when this is applied in the form of a very weak solution, and its consequent acquisition of a peculiar and very characteristic reddish brown or mahogany tint. Dr. Dickinson has pointed out, as an equally valuable test, the readiness with which it gets stained blue by solution of sulphate of indigo.

Lardaceous degeneration of organs, unless it becomes extreme, does not necessarily impair their functional activity. Its effects in this direction only show themselves late in the progress of the disease, and are then probably due in great measure to pressure and other simple mechanical causes.

Although occurring most frequently and obviously in the organs which have been named, lardaceous infiltration occurs also in other parts of the organism. Thus, it is not uncommon in the villi of the intestine and the mesenteric glands; and the so-called 'corpora amylacea' of the nervous centres not only have microscopic characters resembling those of some forms of starch, but present the same chemical reactions as lardaceous matter occurring in other organs.

d. Fatty Degeneration.—Three different conditions are not unfrequently included in this term:—namely, first, the overgrowth of fat-tissue; second, the superabundant storage of oil in cells (other than fat-cells) which are apt to contain normally a greater or lesser quantity of oil; and, third, the actual degeneration or decay of tissue attended with the appearance of molecules of oil in its substance. It is obvious that the first of these conditions is not a degeneration in any sense of the word. No doubt in many cases, as when it affects the heart, it impairs functional activity, but it impairs it by its mechanical influence only. Again, the second condition cannot properly be regarded as a degeneration. It is observed most characteristically in the so-called 'fatty degeneration' of the liver, where the organ increases in size, gets paler and softer than natural, and greasy, and is found under the microscope to have its constituent cells distended with accumulated oil-drops. But under normal circumstances the liver-cells contain a variable amount of fat. The presence of fat in them, therefore, is no sign that they are degenerate; and moreover the excessive accumulation of fat in them does not lead to their destruction, nor does it (according to our experience) affect injuriously the performance of the hepatic functions. The third of the above conditions is that which alone merits the name of fatty degeneration.

Fatty degeneration commences almost invariably in cells or other forms of protoplasm, which get studded with minute refractive molecules, and at the same time increase in bulk. These molecules are supposed to be derived mainly, if not exclusively, from the degradation or decomposition of the protoplasmic matter itself, and are at first few in number and small in size, and chiefly collected immediately around the nucleus. Gradually they increase in number and size, concealing the nucleus and distending the cell; which then acquires a round or oval figure, and appears by transmitted light as an opaque black granular mass, constituting what is generally known as a 'granule-cell,' and has been sometimes termed an 'inflammatory corpuscle.' The last stage is represented by the further enlargement of the cell, its rarefaction, and final deliquescence, with the setting free of the fat-granules which had been imbedded in it. In the later phases of fatty degeneration, when the cells are in great measure destroyed, and the oily matter is diffused throughout the tissues, cholesterine, which had doubtless been suspended in the oily molecules, separates from them, and appears amongst them in the characteristic form of incomplete rhomboidal plates; and the whole tissue gets confused, softened, and reduced to an opaque yellowish-white pulp, or 'detritus.'

The process above described is common in nearly all vital tissues. Pus globules, epithelial cells, connective-tissue corpuscles, are all apt, in the course of inflammatory processes, to become granule-cells.

Cartilage-cells undergo similar changes; and the stellate corpuscles of the cornea, and those of the inner coat of arteries are equally liable to be the seat of fatty deposition. Fatty degeneration of muscular tissue is of much interest. It occurs as a normal process in the involution of the muscular walls of the uterus after parturition; it may often be detected in the hypertrophied muscular fibres of the walls of the stomach and intestines, when carcinoma or other such growths affect these organs; and it is occasionally present in the voluntary muscles. But it is chiefly met with in the muscular fibres of the heart, and has indeed been principally studied in connection with this organ. It commences here with the appearance of fatty granules in the corpuscles which stud the substances of the fibres, and in the immediate vicinity of their poles; but gradually they get more general in their distribution, the fibres progressively losing their characteristic markings, and after a while becoming, like granule-cells, mere accumulations of granular matter. Fatty degeneration is often remarkably well seen in the cells of carcinoma, and many other kinds of morbid growths.

The term '*caseation*' is applied to that condition in which tubercles, syphilitic growths, carcinoma, and collections of pus, acquire the appearance and consistence of some forms of cheese. It is essentially fatty degeneration; but it is fatty degeneration in which there is a deficiency of moisture, in which the degenerate cells shrivel up instead of expanding and undergoing solution, and in which the diseased mass becomes dry and friable instead of pulpy or fluid. It was formerly supposed to be distinctive of tubercle.

e. Pigmentary Degeneration.—The deposition of pigmentary matter is not, any more than that of oil, necessarily a pathological process; nor, even when pathological, is it to be regarded as necessarily an evidence of degeneration. All pigment, originating within the body, appears to be derived from the hæmatine or red-colouring matter of the blood, or from the colouring matter of the bile, which is itself a derivative of hæmatine. In either case, it may be simply diffused in a fluid condition among the tissues, or it may be deposited in the form of granules or small solid masses, or it may assume a crystalline shape. And in either case, again, it may present various modifications of colour; of which red, yellow, brown, and black may be taken as the types. The various stages of pigmentation may be observed superficially in the progress of a subcutaneous bruise; but to follow them thoroughly, it is necessary to investigate the changes which clots, and the tissues in which they are imbedded, present at different periods after extravasation. The blood-corpuscles soon lose their colouring matter, which speedily diffuses itself through the surrounding tissues, staining them, and more especially their protoplasmic particles, of a more or less bright yellow colour. From this, ere long, granular pigment, of a yellow, brown, or black tint, is precipitated amongst the tissues, and in

the clot itself; and, probably, at the same time, small refractive nodulated masses of a deep orange or pale red hue make their appearance. Lastly, small thick rhomboidal crystals, of a deep ruby colour, are produced, which are generally termed 'hæmatoidin' crystals. The final colour which the granular form of pigment assumes is either brown or black; and this, together with hæmatoidin crystals, which are unalterable, is the permanent indication of the previous existence of extravasated blood. A nearly similar series of changes may be observed in the liver, in cases where the escape of bile is prevented;—namely, first, a general staining of the tissues, then a granular pigmentary deposit, and occasionally a more or less abundant formation of hæmatoidin crystals scarcely if at all different from those which are obtained from blood. It should be added that, according to Städeler, bili-rubin differs from hæmatoidin only in containing two more atoms of carbon; and that the various modifications of colour which bile undergoes by keeping are due to the development of substances which differ from bili-rubin only in possessing larger quantities of water, relatively to carbon and nitrogen.

The pathological precipitation of brown or black pigment in a granular form is well seen, in the cells of the rete mucosum in Addison's disease, and in the brown discoloration which often succeeds various forms of skin disease and cutaneous inflammations—the results of chemical or other irritant applications; in the cells of melanoid carcinoma and sarcoma; and we might perhaps add, in the cells of the testis and of the grey matter of the brain, during the later periods of life. The deposition of yellow, red, and brown pigmentary granules, and of hæmatoidin crystals, is, as before stated, a common result of the extravasation of blood; accordingly these matters are found in corpora lutea, in the neighbourhood of apoplectic effusions, and in the parietes, in the interior, and in the vicinity of small vessels obstructed by clots or otherwise diseased. In certain cases of malarial fever, in which the spleen is seriously affected, black pigment-masses are formed in that organ through decomposition of blood-corpuscles, and are carried thence by the circulating blood, and deposited in the capillary vessels of other parts of the system. Black pigment, also derived from the blood, is frequently met with in the tissues of the lungs and bronchial glands. In reference to this latter case, however, we must not forget that inhaled carbonaceous matter gets absorbed by the epithelial cells of the bronchial tubes; and that hence, just in the same way as pigments, artificially introduced by tattooing, find their way along the lymphatics, so carbonaceous matter may be absorbed at the mucous surface of the bronchial tubes, and thence conveyed to the bronchial glands. Nevertheless, the black pigment found in them is doubtless, in nearly all cases, chiefly of blood origin.

f. Uratic Degeneration.—This occurs only in gout. It is characterised by the appearance of needle-like crystals of urate of soda in the

substance of articular cartilages, in the periosteum, synovial membranes, and tendons. They are observed mainly in connection with the protoplasm of the cells, are often irregular in their arrangement, but are very apt to form opaque densely-arranged star-like clusters.

g. Calcareous Degeneration.—This consists in the deposition of a combination of carbonate and phosphate of lime in some previously existing albuminoid matrix, with which it combines to form minute granules and spherules. These increase in size by concentric additions to their surface, and presently coalesce into botryoidal masses—the general form and arrangement of which are determined by the peculiarities of the tissue in which the process is going on. The precipitation of calcareous matter takes place almost exclusively in the intercellular substance; which first appears dusted with minute granules, and then, as these multiply, becomes black and opaque to transmitted light. Later on, the enlarging granules run together, the blackness and opacity disappear, and the calcified tissue gets refractive and transparent. The cellular elements frequently remain intact, or nearly so, during this process; and if they be stellate, and numerous, the result is the formation of a mass having a close resemblance to true bone. Calcareous granules have a superficial likeness to globules of oil; but may be distinguished from them by their ready solubility (with the giving off of bubbles of carbonic acid gas) in hydrochloric and other acids, and if they be round by displaying a cross when examined by polarised light. Rindfleisch supposes, that the pathological deposition of calcareous matter, which in the blood is rendered soluble by the presence of carbonic acid, takes place primarily at the periphery of cell-districts; and that it is due to the difficulty of reabsorption of nutrient matters which have found their way thither. This difficulty favours the separation of their more diffusible from their less diffusible constituents, and thus the removal of the dissolved carbonic acid, and the precipitation of the calcareous matters which the carbonic acid had rendered soluble. This explanation accords very well with Mr. Rainey's views on the formation of shells and bone.

Calcareous precipitation is very common. It occurs in the internal coat of arteries, and in the whole thickness of the walls of minute vessels, in tendon and cartilage, and even in the substance of skin. It is especially apt to take place in inflammatory and other adventitious products. Thus, we find plates of calcareous matter (often assuming the characters of bone) in old false membranes of the pleuræ and pericardium, in the lining of cysts, and in the choroid coat of the eye. And, indeed, most degenerative products—such as cheesy tubercles, inspissated pus and old clots—when they have lost their moisture, and their more soluble or diffusible constituents, become its seat, and, first assuming a mortary condition, finally shrink into calcareous lumps. But, although earthy matter is deposited mainly in the tissues between cells,

it is occasionally found in the interior of cells, and especially in those of unstriped muscle. It is thus that the smaller arteries are sometimes converted into rigid cylinders, and large portions of uterine muscular tumours into calcareous masses.

2. *Necrosis, or Gangrene.*

Several of the degenerations just considered end, as we have pointed out, in the disintegration and death of the tissues which they affect. We do not intend to pursue this question further, or to speak of that form of death which results from the direct action of destructive agents; but we purpose discussing very briefly the subject of necrosis, or mortification. This often arises in the course of inflammation, and often affects rapidly-developing morbid growths; but whether occurring in these cases, or under other morbid conditions, it is always due immediately to obstruction of afferent vessels, or to weakness of the heart's action, and the consequent more or less complete arrest of the supply of nourishment to the affected parts.

When the death of any part of the organism takes place, the conservative influence of vitality ceases in it, its constituents fall under the unrestrained operation of chemical and other physical powers, and then undergo a series of destructive and often putrefactive changes, in virtue of which its complex organic constituents gradually get reduced to substances of much more simple elementary composition, and its various morphological elements lose in a greater or less degree their characteristic features. The rapidity, however, with which these processes take place, necessarily depends upon the degree in which conditions which favour them happen to be present. These are chiefly heat, moisture, and exposure to oxygen or air, and to the various microscopic organisms which air and water contain. Hence, it follows that gangrene is especially rapid, and its products especially fetid, when it occurs in superficial parts, or in the lungs, or in the course of the alimentary canal, where there is free exposure to oxygen in a more or less diluted form; or when it occurs in parts which are juicy and loaded with blood, as they are if they have been the seat of inflammation, or if there has been previous obstruction of veins, or if the arteries have continued for a time to pump blood into them—when in fact the gangrene is what is usually called 'moist.' When there is little moisture of tissue, and that moisture admits of ready removal by evaporation, or in other ways, and especially if there be at the same time entire protection from the influence of atmospheric air, the changes which ensue are very slow; the parts get inspissated, dried up, mummified; and even delicate structures retain for a great length of time their chemical and microscopical characters, in a very slightly modified condition. A good example is afforded by the changes which ensue in an extra-uterine fœtus long retained.

Bone, teeth, hair, horny matter, elastic fibres, and cartilage resist putrefactive processes in a remarkable degree. But all the softer albuminous or albuminoid tissues, and fat, rapidly change into a series of transitional compounds, the nature of which is very imperfectly known. Some, however—such as leucin, tyrosin, margarin, pigment, cholesterine and triple phosphate—are fixed; some are soluble but not volatile; and others again are volatile and offensive, and give to gangrenous parts their characteristic fetor. Amongst the last must be included sulphuretted hydrogen, sulphide of ammonium, and valerianic and butyric acids. Ultimately, most albuminous and fatty matters are reduced in large proportion into carbonic acid, ammonia, and water.

The visible changes which attend gangrene are not less remarkable than the chemical; but they closely correspond with those which characterise ordinary degeneration. The blood stagnates; and soon the colouring matter escapes from the red corpuscles, permeates the vessels, and infiltrates and stains all the tissues around. Thus, the course of the superficial veins gets indicated by broad livid lines. Soon the diffused pigment is deposited in the form of brown and black grains and even of hæmatoidin crystals, and its presence tends to give a characteristic hue to the parts. The red corpuscles themselves either melt away, or are converted into small angular pigmented bodies. The white corpuscles of the blood and other protoplasmic masses get opaque and granular, then the seat of deposition of molecules of proteinous matter and of oil, and finally after becoming caseous break up into fragments. The contents of fat-cells ooze through their membranous parietes, and diffuse themselves in globules of various sizes among all the tissues; and after a while the solid fats crystallise out, and plates of cholesterine make their appearance. Muscular tissue, whether striped or unstriped, suffers much the same changes as protoplasm; it first becomes opaque and granular, soon presents oil and pigment-granules in its substance, and presently breaks up (the striped fibres often splitting into transverse discs) and forms a viscid confused mass. Double-contoured nerves early present obvious changes:—the axis cylinder undergoes the same transformations as other forms of protoplasm; but the medullary sheath breaks up into globular, oval, and irregularly rounded, refractive masses of an oily character, and presenting the peculiar features of what is termed by Virchow 'myeline.' Ordinary connective tissue swells up, becomes opaque and granular, and then melts away. And bone, although it retains its characteristic form and appearance, loses its animal matrix.

Many lowly organisms make their appearance in putrefying tissue. But by far the most important of these are the minute omnipresent bodies which are known by the name of 'bacteria.' Indeed the evidence now seems to be conclusive, that actual putrefaction is determined by the growth and multiplication of these bodies, and that

the recognised effects of air and moisture in promoting the decomposition of dead and dying tissues are in reality due to the bacteria suspended in them.

We have adverted to the fact that the appearances and progress of gangrene vary according to the degree in which the dead parts are exposed to the conditions which promote putrefaction. But the nature of the organ involved also necessarily influences the nature of the result. Hence, we need not be surprised that gangrenous parts present great varieties of character. In internal organs, as the brain, the dead portion becomes soft and pulpy, and its colour opaque and yellowish, with perhaps a faint greenish tinge and a little red-mottling, and the cellular constituents get granular and fatty, and presently reduced to a mere detritus; but no putrefaction ensues, no offensive matters are developed, and the more soluble and diffusible products are at once removed from the part by absorption. When an inflamed or congested leg, or a strangulated bit of bowel, becomes gangrenous, the affected part contains an extraordinarily large quantity of blood which escapes into the tissues, and assuming there the characters of black pigment, blackens them; putrefaction takes place rapidly; a sanious fluid, charged with decomposing elements, and containing numerous globules of oil and much pigment, pervades the tissues and perhaps forms blebs at the surface; and bubbles of offensive gas probably appear in similar situations. When gangrene occurs in the lung, the tissue often looks anæmic (sometimes, however, it is black with congestion), and presents in the first instance a translucent greenish tinge, but soon breaks down into a turbid greenish pulp of horrible fetor. In other cases again, as for example in sloughing ulcers, or carcinomatous growths, the parts which are actually dead assume a dirty opaque white or greyish appearance, and are thrown off in masses. Lastly, when the affected parts have been supplied with little blood, or rapidly lose the fluid which is in them by evaporation, the condition termed 'dry gangrene' results. They then shrivel up, and gradually, by the retention of the blood-pigment within them, acquire a deep maroon or black colour; and, as was before pointed out, their decomposition proceeds very slowly, and they become dry and mummified.

C. Mechanical and Functional Derangements.

The various morbid processes of proliferation and degeneration, which have been described, bring with them a host of *mechanical* and *functional* disturbances, which form essential elements of disease, and are often far more important, at all events far more striking, elements than are those other lesions which give rise to them. As regards functional disturbances, indeed, it is obvious, that their presence

implies the coexistence of some nutritive or other material lesion of the part or organ whose functions are disturbed; and that their gravity must depend, far less on the amount or quality of this material lesion, than on the importance of the affected organ in relation to the well-being of our higher faculties, or to the maintenance of life. Thus, a fibroma, connected with superficial parts, may attain enormous dimensions without materially influencing the general health; whereas a very small growth of the same kind, involving the urethra or intestine, would probably soon cause mechanical obstruction, and induce the usual symptoms of strictured urethra or bowel. And thus, again, a tubercular mass or an hydatid may exist for some time imbedded in the substance of the brain, and yet give very little sign of its presence there; whereas those functional disturbances of the central nervous organ, which we know as acute mania and epilepsy, depend on such slight lesions that they even now, in great measure, elude detection.

1. *Mechanical Derangements.*

These consist mainly of (*a*) *displacements* of organs, (*b*) *compression, contraction*, and *impaction*, (*c*) *dilatation*, and (*d*) *rupture* and *extravasation*.

a. Displacement of parts is exemplified in the altered position which the heart assumes when it is subjected to the pressure of unilateral empyema, or of a mediastinal tumour, and which this organ, together with the lungs, acquires when there is extreme angular or lateral curvature of the dorsal spine; it is shown also in hernia, intussusception, and prolapse of the rectum, and in the various flexions and other displacements of the uterus.

b. Compression, contraction, and *impaction*.—These conditions scarcely need explanation; yet it may be well to illustrate them by their effects on tubular organs. Compression of a tube means that it is reduced in calibre and perhaps modified in shape, by forces acting upon it from without; contraction signifies that its bore is diminished by the inherent action of its own walls, or by morbid changes taking place in them—that there is in fact a 'stricture;' impaction implies that its channel is occupied by some concretion or other foreign body. It is obvious that any of these conditions may end in the complete obstruction, or closure, of the tube.

The effects of *compression* are manifested, when a large quantity of blood or serum is effused upon the surface, or into the ventricles, of the brain; when the lung shrinks under the pressure of accumulated pleural secretion; when the heart gets flattened, and incapable of dilating, under the influence of blood which has escaped into the pericardium from a ruptured aorta; when the trachea is squeezed by a goitre or aneurism; when the intestines are strangulated by bands, or the mouth of a hernial sac; when the rectum is flattened by the

pressure of a diseased uterus. They are shown also in many cases in which organs are the seats of interstitial growths:—in cirrhosis of the liver, the newly-formed fibrous tissue contracts upon the essential elements of the organ amongst which it is distributed, and leads to their more or less complete destruction; and the same thing happens, as regards the nervous centres, in the morbid condition now commonly termed 'sclerosis.'

Contraction may be due, either to spasmodic action of the part affected, or to some growth (inflammatory or other) involving it. As examples of the first condition we have temporary contractions—of the cerebral vessels, inciting epileptiform convulsions; of the muscular walls of the bronchial tubes, causing asthma; and of the sphincter ani and compressor urethræ, producing spasmodic stricture respectively of the bowel and of the urethra. As examples of contraction due to inflammatory or other such changes we may enumerate, obstructive disease affecting the several cardiac orifices; laryngeal œdema; malignant or other organic strictures of the œsophagus, the pyloric or cardiac orifice of the stomach, the ileo-cæcal opening, or the anus; and similar affections of any part of the genito-urinary apparatus. It need scarcely be added that smaller and even microscopic tubes and ducts, such as those of the breast, kidney, and sebaceous glands, may get similarly obstructed.

Impaction.—There are few tubular organs in which impediment from this cause does not occasionally take place. In the vascular system, especially in the systemic veins, thrombi or clots not unfrequently form, and cause obstruction. And in the same system, portions of such clots, or of inflammatory vegetations developed upon the cardiac valves, often get detached, and then carried onwards by the circulating fluid, until they reach some vessel too small to admit of their further progress, where consequently they get fixed, or impacted, and block it up. In the alimentary canal, and ducts which open upon its surface, concretions frequently form, and, becoming lodged, cause more or less serious consequences:—thus, the ducts of the salivary glands may be obstructed by salivary calculi, the common hepatic duct by gall-stones, and the intestine also by gall-stones of large size, or even by indurated fæces. In the intestinal canal, moreover, indigestible substances, purposely or accidentally introduced, such as masses of hair, or of vegetable fibres, and the like, occasionally form concretions. Calculi again are of extremely common occurrence in the urinary cavities and passages. In considering this subject we must not forget to advert to the impaction, or (what is equivalent to impaction) the accumulation, of abundant or tenacious secretions in cavities or canals, by which they are apt to become choked; as occurs in cases of severe bronchitis, when the bronchial tubes are overloaded with muco-purulent secretion, and in inflammation of the kidneys, or Bright's disease, when the renal tubules get blocked up by epithelial masses, or by blood or fibrinous casts.

c. Dilatation of cavities depends, for the most part, on some disproportion between the pressure which their contents exercise upon their parietes, and the force which these parietes are capable of exerting in opposition to that pressure; and hence may be caused, either by unwonted accumulation of contents, or by undue weakness of parietes, or by the concurrence of these two conditions. It should be added that even when dilatation does not originate in morbid weakness (however produced) of the walls of a cavity, it very soon causes it. But dilatation may occur in cavities of new formation, as well as in such as are of normal presence, and hence its discussion involves that of the growth, if not that of the origin, of cysts.

Cysts are very commonly classified as a subdivision of tumours. A little consideration, however, will suffice to show that they differ essentially from true tumours (that is to say, from neoplastic or proliferating growths), in the facts, that they are not themselves neoplasms; and that when they occur, as they often do, in association with such growths, that association is a mere accident, depending either upon some structural peculiarity or upon some special tendency, of the part affected, or of the neoplasm itself. Cysts may be divided generally, in accordance with their mode of development, into at least four different groups, namely: (i.) those formed by dilatation of natural cavities; (ii.) those resulting from distension of ducts; (iii.) those caused by extravasation of blood; and (iv.) those originating in the softening and destruction of tissue, or in the dilatation of natural alveolar spaces.

i. *Cysts by dilatation of natural cavities.*—Among these must be included the pleuræ, pericardium, peritoneum, tunica vaginalis, and synovial cavities, distended with dropsical or inflammatory exudation. They are exemplified also in the dilatations of the ventricles of the brain and cord, which constitute respectively the morbid conditions known as 'hydrocephalus' and 'hydrorrhachis,' and in the malformations of the same organs, termed 'encephalocele' and 'spina bifida.' Dilatations of the cavities of the heart, aneurismal tumours of arteries, varicose conditions of veins, ovarian cysts, and cysts of the broad ligament, thyroid body, and many other organs, fall more or less obviously into this group.

ii. *Cysts by distension of ducts, or 'by retention,'* are even more common and more important than the last. We meet with them in the lungs, when the bronchial tubes are dilated, or when emphysema is present. They occur in all parts of the alimentary canal:—in the œsophagus, when its walls are paralysed, or there is obstruction at the cardiac orifice; in the stomach itself, under analogous conditions; and in any part of the large or small intestines above the seat of an impediment, or when the parietes are weakened by inflammatory changes. When the hepatic, pancreatic, or salivary ducts are obstructed by concretions, the tubes behind get greatly dilated. Cysts from this cause are exceedingly common throughout the whole of the genito-urinary

apparatus:—as, for example, when the bladder is distended, secondarily to the presence of a urethral stricture; when, under similar circumstances, the ureters and cavities of the kidneys dilate; and when, owing to their obstruction, the tubules of the kidneys expand into rénal cysts. They occur also in the uterus and Fallopian tubes, and in the tubules of the testes, in consequence of stricture or other impediment to the escape of their contents; and so again in the breast, and in the sebaceous, and almost all other, glands.

A variety of this mode of formation of cysts has been observed, by Dr. Wilson Fox and others, in certain cases of multilocular cystic tumours of the ovary. They state, that papillary growths take place from the inner surface of a comparatively large cyst; that these, as they increase in length and bulk, get closely wedged together; and that they at length coalesce in numerous points, leaving irregular chinks between them—which chinks then, by the retention of the secretions of their parietes, gradually dilate, and ultimately form distinct cavities.

iii. *Cysts by extravasation.*—Blood effused either into cavities, or into the substance of organs, undergoes a series of degenerative changes. In some instances these result in the softening and breaking down of the central portion of the clot, and in the consequent formation of a cyst. The best examples of such cysts are furnished by the brain and the cavities of the heart—in the former case, as a result of the changes which take place in apoplectic effusions; in the latter, as a consequence of the softening of coagula which have formed some time anterior to death. Clots imbedded in the substance of the brain almost always undergo absorption, and leave behind them cysts filled with clear fluid, traversed by delicate filamentous bands, and bounded by tissue still coloured with blood-pigment; those occupying the cavities of the heart break down into an opaque milky fluid, charged with degenerate blood-elements. It not unfrequently happens that cysts are formed in the interior of sarcomatous and other soft and highly vascular tumours, by exactly the same process as that which produces apoplectic cysts. It may be added that extravasated blood, especially if it be extravasated in successive strata, in many cases forms solid masses, which may then become organised, and constitute, according to their position, the various forms of 'blood-tumour' or *hæmatoma*.

iv. *Cysts by softening of tissues.*—These are generally due to the occurrence of one or other of the degenerative processes, which have been described. We meet with them in abscesses, and in cases where tissues have undergone mucous, colloid, or fatty softening. Hence, putting abscesses on one side, they occur most frequently in proliferating growths; and indeed, in some cases of disseminated malignant tumours the tendency to become thus hollowed into cavities is general. Bursæ in unwonted situations must be included in this group.

It may be worth while to point out, that, as cysts dilate under the

influence of their accumulating contents, their parietes, which often increase at the same time in thickness, tend to tear or yield at points in their outer surface; that thus, pits which gradually increase in area and in depth are formed; and that these not unfrequently end in perforation or rupture, and, in the case of cysts separated by a party-wall, in the establishment of communications between them. We should also mention that the inner surface of cysts, contained within the substance of proliferating growths, may, however the cysts have been produced, get lined with epithelium, and the seat of new outgrowths; and that hence we not unfrequently see fungous, papular, villous, or cystic formations, springing from the inner surface of such cysts, just as they may spring from the diseased mucous, serous, or cutaneous surface.

d. Rupture and Extravasation.—The occurrence of rupture and extravasation, to which the distension of cavities and canals ultimately tends, is an event of great pathological importance, and often of the gravest danger. Such accidents are common. Sometimes the heart is torn, and the pericardium consequently gets distended with extravasated blood. The rupture of aneurisms and of varicose veins is, we need scarcely say, of extreme frequency. In the lungs, the progress of vesicular emphysema is largely dependent on rupture of air-cells; and in interlobular emphysema, and pneumothorax, we have not only laceration of tissue but extravasation of air. Laceration of the stomach in ulcer of that organ, or of the intestine in the course of typhoid fever, is attended with the escape of its contents into the peritoneal cavity. Again, abscesses and hydatid cysts often rupture, and discharge their contents; and, indeed (as we have already pointed out), cysts of all kinds are liable, in various degrees, and with various results, to similar accidents.

2. *Functional Derangements.*

To discuss these thoroughly would involve an analysis of nearly all the symptoms of all diseases. Morbid processes, indeed, are mainly recognised during life by the functional disturbances to which they give rise; and some diseases—so far, at least, as we know them—are nothing more than groups of such disturbances. Every organ of the body, every particle of the organism, has its proper duties to discharge; and, under the influence of morbid processes, these duties become increased or diminished, and in either case probably more or less profoundly modified. The function of the eye is to see, that of muscle to contract, that of the kidney to excrete urine; but the eye may be unduly sensitive to light, or its power of distinguishing objects may be impaired, or it may see things which have no real existence; muscle may contract with spasmodic violence, or it may be thrown into convulsive movements, or it may lose its power of action altogether; the kidney may cease to excrete urine, or it may separate from the blood a

portion only of the usual urinary solids, or it may discharge matters which are altogether foreign to the normal constitution of that fluid. In these, and in many other ways, the organs which have been named may present signs of functional disturbance; and it is clear that similar observations may be made in reference to every other organ. We do not purpose, however, to enter here upon the consideration of functional derangements generally, for most of them will best be discussed when we come to speak of local diseases. But some, which are connected more especially with the vascular and nervous systems, enter so largely into the complex phenomena of disease, and form such important elements in diseases which are fundamentally distinct from one another, that it will be convenient to discuss them separately, and at once. We refer mainly to *congestion, dropsy, fever, the typhoid condition, collapse,* and *death.*

a.—Congestion.

Accumulation of blood in the vessels of a part is necessarily associated with dilatation of these vessels; but, as we have pointed out in speaking of inflammation, this dilatation may be active, and the accumulation of blood therefore secondary to it, or it may be passive, the vascular walls yielding under the pressure of the blood within them.

i. *Active congestion* is due to active dilatation of vessels, or, at any rate, to that kind of dilatation which may be evoked by reflex irritation, and is effected under the influence of the nervous system. This dilatation commences for the most part in the small arteries, and presently involves the capillaries and small veins. Active congestion is constantly connected with inflammation, at least in its earlier stages, and generally with morbid proliferation. And as, in health, we recognise its temporary presence in the cheek which blushes with shame, and in the general surface after violent exercise; so, in disease, we recognise its temporary presence in the hectic flush of phthisis, and in the general redness which attends many forms of febrile disturbance.

ii. *Passive congestion* has been divided, unnecessarily as it seems to us, into two varieties, namely:—first, that which is dependent solely on loss of power in the walls of the dilated vessels; and, second, that in which the dilatation is traceable to some mechanical impediment to the passage of blood through the veins. There is doubtless a theoretical distinction between them; yet it is obvious that in both cases the dilatation is really passive, and due to the fact that the vessels yield under the internal pressure to which they are subjected. The first case is exemplified, by that dilatation of vessels which attends the later stages of inflammation; and by that permanent enlargement of them which is often seen in the vicinity of old ulcers, and of inflammatory and other formations, and is so common in the noses and cheeks of persons who are given to drink, or have been exposed to the influence

of weather, or suffer from acne rosacea. The second variety is observed generally in obstructive heart-disease, and under analogous circumstances in limited districts of the body. In disease of the mitral valve, in emphysema and some other affections of the lungs, and especially in disease of the valves of the right side of the heart, the blood gets delayed in the systemic veins—the trunk-veins, their tributary branches, and the capillary veins successively undergoing dilatation. We often see in such cases, groups of minute subcutaneous veins forming varicose tufts, persistent livid congestion of the nose and cheeks, of the hands and fingers, and of the feet and toes, due to general over-distension of their capillary veins and capillaries, and, more important than all, congestions of internal organs—especially of the liver, which assumes the 'nutmeg' condition, and of the kidneys, which get indurated and secrete albuminous urine. Again, whenever a vein gets obstructed by a thrombus, or by external pressure, the tributary veins undergo precisely the same changes which the veins undergo generally in heart-disease. Thus, if there be an aneurism, or other tumour, in the upper part of the chest, and the descending cava or one of its branches be compressed by it, the veins of the head and neck and upper extremities, or those of one side of this portion of the body, get distended; if the femoral vein be blocked up by a clot, the veins of the foot and leg suffer similarly; if the lateral sinus be obstructed, enlargement and congestion of the retinal veins, and of those of the conjunctivæ and eyelids, not unfrequently occur. So also when, owing to cirrhosis or other hepatic disease, the passage of blood through the portal vessels is impeded, the veins of the mucous membrane of the stomach and bowels become over-distended, and occasionally relieve themselves by actual hemorrhage. We may add that the mere statical pressure of a column of blood, which is competent to produce a varicose condition of the veins of the lower extremities, is competent also to produce dilatation of the smaller veins and capillaries. Nor should we omit to point out, that mere feebleness of the heart's action, in other words, incompetence to propel the blood efficiently, as occurs in the later periods of heart-disease, leads to stagnation of blood in the capillary and other small vessels, and hence to passive congestion; and that, on almost the same principle, obstruction of an artery, as we see in the lungs and kidneys, very often allows the territory to which it is distributed to become the seat of intense congestion and even of hemorrhage.

b.—Dropsy.

Dropsy is the accumulation of serous fluid within the cavities of the body, or in the areolar spaces of the connective tissue. It depends either, like passive congestion, upon mechanical obstruction to the flow of blood along the veins, or upon the presence of inflammatory or other analogous processes, or upon some morbid condition of the blood or blood-vessels, or, lastly, upon obstructive disease of the lymphatic tubes or

glands. Further, dropsy may be local or general, and dependent therefore on local circumstances or on causes which act universally.

i. The causes of *general dropsy*, or *anasarca*, are for the most part obstructive diseases of the heart, morbid conditions of the lungs impeding the circulation through the pulmonary vessels, affections involving the secreting structure of the kidneys, and certain morbid states of the blood or tissues. The general dropsy which attends heart or lung disease is, like the congestion which also attends these affections, purely mechanical, and indeed may be regarded as the sequel of that congestion. In the healthy condition the thin walls of the capillary vessels and small veins allow a constant escape of the serum of the blood into the tissues which are external to them—the quantity, which thus escapes in a given time, being largely dependent on the varying degrees of pressure within the vessels, and on the more or less facility with which the lymphatic vessels perform their proper absorbent functions. Now when a mechanical obstacle exists to the transit of blood through the heart or lungs, the systemic veins and capillaries soon get overloaded, and the pressure upon their inner surface rapidly rises. And we can readily see that, while there arises, on the one hand, a greatly increased tendency for the serum of the blood to transude at the peripheral distribution of the venous system; there is developed, on the other hand, a tendency at the opposite end of that system to impede the entrance of the contents of the thoracic duct; and that hence the fluid, which is effused into the tissues in abnormal quantity, is absorbed with difficulty, and dropsical accumulation necessarily ensues. It should be observed, that the effusion is not simply, although it is mainly, fluid, but that it always comprises a considerable proportion of leucocytes, and generally some red corpuscles. Cardiac and pulmonary dropsies are, as their mechanism would indicate, always associated with more or less obvious congestion, and almost invariably first show themselves in the parts which are most dependent. The explanation of renal dropsy is not so clear. It obviously does not depend on any obstacle to the circulation existing in the heart or lungs, or on over-distension of the venous system with blood, or, we may add, on any similar distension of the capillary vessels; for the patient usually presents an anæmic appearance, even when the blood itself is not abnormally pale. There is, however, in renal disease very unmistakable obstruction throughout the whole capillary arterial system; for, as Dr. Geo. Johnson has well shown, the small arteries generally become extremely thickened and their canals proportionately contracted; and we know that the left ventricle of the heart hypertrophies to overcome some impediment—doubtless the mechanical impediment which Dr. Johnson has discovered existing at the periphery of the vascular system; and that, associated with these conditions, there is, as we should expect, greatly increased blood-pressure in the arteries. It seems hardly likely, therefore, that in this case the escape of fluid into the tissues should take place

through the capillaries and capillary veins; but, on the other hand, it seems very probable indeed that it occurs through the thickened capillary arteries, in consequence of the extreme internal pressure of fluid to which they are subjected. It can scarcely be objected to this explanation, that the thickened walls of the small arteries would materially counteract the tendency for fluids to transude through them, in face of the fact that the hyaline thickening of the walls of the Malpighian vessels of the kidney, in lardaceous disease of that organ, is not incompatible with a profuse discharge of urine. In pure renal anasarca the skin is usually remarkably anæmic and waxy-looking, and the dropsy is often first detected, not in the lower extremities, but in the eyelids and the scrotum. General dropsy occasionally takes place in persons who, from whatever cause, are in a state of anæmia; it is especially common in chlorotic girls. We know that in these cases the blood is in a state of unnatural dilution; that the muscular tissue generally, including that of the heart and probably that of the blood-vessels, is enfeebled; and that the circulation, therefore, even though the heart acts quickly, is languid; and we are hence justified in assuming that the anasarca is due either to the fluidity of the blood, or to the languor of the circulation, or to a combination of these conditions. It need scarcely be remarked that the supervention of anæmia, in the course of disease of the heart or kidneys, is very often the determining cause of an attack of anasarca which otherwise would have been escaped; and that anæmia is, in many respects, a very serious complication of the diseases of these and other organs.

ii. *Local dropsy* depends either on mechanical obstruction of the principal vein or veins leading from the dropsical part, or on obstruction of the lymphatics, or on the presence of inflammatory or other like processes. When it depends on venous obstruction, we have, within a circumscribed space, very nearly the same conditions as those which, in cardiac disease, affect the whole body—a vein is impervious; its tributary branches down to the capillaries get distended with blood; the serum of which presently escapes into the tissues in larger quantities than the lymphatics are able readily to remove. The most important variety of local dropsy from venous impediment is that which takes place in the abdomen, when the passage of blood through the portal vein is impeded, as in cirrhosis, or by growths occupying the transverse fissure of the liver. But any vein may be obstructed, either by pressure from without or by a coagulum within it. Thus, by obstruction of the superior cava enormous anasarca, limited to the head and neck and arms, may be produced; from obstruction of the inferior cava (even from so slight an amount of it as results from the pressure of ascitic fluid) dropsy limited to the lower extremities may arise; and, in consequence of obliteration of the brachial or femoral vein anasarca of the corresponding arm or leg may ensue. It has already been pointed out that, whenever inflammation is in progress, a considerable excess of the

serum of the blood is poured out into the tissues; and that, especially when the parts involved are lax or present some suitable structural peculiarity, the effused serum accumulates in them, producing a more or less obvious dropsical condition. We see this in the œdema of the eyelids, which attends the formation of a common stye; in the dropsical condition of the tissues around the joints, in rheumatism and gout; in the œdematous state of the leg, when erythema nodosum or slight periosteal inflammation is present; but we see it especially in inflammation of the serous and synovial membranes—thus, inflammation of the pleura constantly causes hydrothorax, inflammation of the pericardium hydropericardium, inflammation of the peritoneum inflammatory ascites, and inflammation of the synovial membrane hydrops articuli. The effusion of serum in excess also attends the development of tubercle, and of carcinoma and other forms of malignant growths; and consequently we often find the serous cavities full of dropsical fluid, in connection with the growth of such tumours from their parietes. It may, perhaps, be a question in some of these cases, as to how far the dropsy is due to the mere excessive effusion naturally attending morbid proliferation, how far it may be attributed to obliteration of some of the veins leading from the great omentum and other parts. The remaining form of dropsy to which we have adverted is that which is due to lymphatic obstruction. We have already briefly considered this subject in connection with both fibroma and lymphoma; and need say no more about it now, than that the lymphatics of a limb or organ occasionally get obstructed, and that then (to take the case of the limb) the whole member becomes tense, elastic, pale, and infiltrated with fluid, having the chemical and microscopical characters of lymph; that the tissues thus soaked in nutrient fluid tend to become hypertrophied; and that here and there subcutaneous vesicles, which may be regarded as simply dilated lymphatic passages, make their appearance, and from time to time rupture, and discharge large quantities of lymph.

In cases of general dropsy, whether of cardiac or of renal origin, both the general connective tissue and the various serous cavities, as a rule get involved in pretty nearly equal proportion; but now and then, in association with slight anasarca, there may be extreme ascites, or extreme effusion into one of the pleuræ. In such cases the local excess is necessarily due to the co-operation of some local cause—the ascites, for example, to a nutmeg condition of the liver, or to some slight peritoneal inflammation; the pleuritic accumulation either to slight general pleuritis, or to the circumscribed inflammation of the pleura which is usually excited in the neighbourhood of pulmonary apoplectic clots.

c.—Fever.

By the term 'fever' is meant that abstract condition which is common to all so-called 'febrile disorders,' and the presence of which gives

them their claim to that designation. Essentially it means, undue elevation of temperature; the immediate or proximate causes of that elevation; and the consequences which these conditions entail.

i. The *normal temperature* of the body has been variously estimated; but, on the average, seems, in the adult, to range between 98·4° and 99·5°, in the infant, to stand at a somewhat higher figure. It presents however, within narrow limits, numerous variations. First. The most constant and important of these is the diurnal variation, which rarely exceeds 1·5°, but occasionally amounts to as much as 3·5°. The minimum temperature, according to Dr. Jürgensen, occurs from 1·30 A.M. to 7·30 A.M.; the maximum from 4 P.M. to 9 P.M.—the temperature between 7.30 A.M. and 4 P.M. rising with some fluctuation; that between 9 P.M. and 1.30 A.M. gradually falling. This daily variation corresponds pretty accurately to similar variations in the activity of respiration and circulation. Second. A slight but decided elevation of temperature usually follows the ingestion of food. Third. Muscular exercise has a similar influence; although, as Dr. Davy has shown, this elevation manifests itself, less by actual increase of the temperature of the internal organs, than by the general diffusion of temperature throughout the organism. Fourth. The external temperature, again, influences that of the body in a greater or less degree. But, under ordinary circumstances, its influence is much less than might be supposed; for variations of season in our own climate have a scarcely perceptible effect, and even tropical heat and arctic cold rarely disturb the temperature of the internal organs beyond a degree or two. The influence of external temperature depends, however, upon the conditions under which it is exerted; for, if these be favourable, the general heat of the body may be very largely and rapidly augmented or lowered, and even to a degree which is incompatible with the maintenance of life. Thus, whenever the medium (air or water), in which the body is immersed, is in rapid movement, it will, if of a higher or lower temperature than the body, elevate or depress its temperature in a much greater degree than if it were at rest; and again, whenever perspiration is impeded, as it necessarily is in a moist atmosphere, or in water, the effects of heat are exerted with special efficacy.

The conditions which determine the heat of the body, and which regulate it, have been investigated with considerable success. It is certain, in accordance with the laws of force, that no heat can be developed in the body save such as may be traced, directly or indirectly, to the latent heat of the substances which are ingested as food; that the total amount of heat, which the body is capable of evolving, is simply that which would be emitted in the course of its entire destruction by burning; and that, neither in its parts nor as a whole, has it any more power of creating heat than of creating matter. It is obvious, therefore, that the development of heat in the body is due simply to the setting free of latent heat by the destructive oxidation which is

constantly going on in it; and that the quantity of heat developed in any given time is an exact measure of the amount of oxidation which has taken place in that time. It is equally obvious, that the excreta furnished by the skin, lungs, kidneys, and alimentary canal (representing as they do the lowest degree of degradation to which, after various changes, the alimentary matters have become reduced), must furnish the means of determining exactly both the amount of oxidation which has been effected, and the amount of heat which has been evolved. Ranke, by comparing the daily quantity and quality of the food with the daily quantity and quality of the excreta, has arrived at the conclusion, that the healthy adult body evolves on the average enough heat in twenty-four hours to raise 44 lbs. of water from the freezing to the boiling point; and it has been estimated further, that of this heat 2·6 per cent. goes to the elevation of the temperature of the food ingested; 5·2 per cent. to the warming of the air breathed; 14·7 to the vaporisation of the water discharged by the lungs; and 77·5 to the radiation and evaporation from the skin.

The above statements, however, only represent the final result which is attained, after many transmutations within the body, during which heat becomes alternately latent and sensible. We know, for example, that heat is as essential for the maintenance of the corporeal functions, as it is for that of the functions of the steam-engine; that every act of growth and development, every nervous operation, every muscular contraction, is dependent on the heat developed by oxidation, and is attended with the temporary disappearance or absorption of a certain quantity of heat; while, on the other hand, everything which interferes with, or impedes, or arrests, the performance of these functions—the friction of the blood against the capillary and other vessels, and of the muscular fibres against one another, every opposed muscular effort, and possibly even the constant passage of nervous currents along the nerves—is attended with the reappearance of that heat in a sensible form.

It remains to consider on what conditions the regulation of the amount of heat developed, and the regulation of the temperature of the body, depend.

As regards the former question, there can be no doubt that that degradation of tissue and of material which results in the evolution of heat, although in itself a purely chemical process, is indirectly largely under the influence of the nervous system, and especially of its sympathetic portion; for it is to this that the varying rapidity and force of the heart's contractions, and the varying diameters of the vessels (which between them so powerfully affect the molecular changes which are going on in the body) are due; and it is possibly by its direct operation on the essential elements of glandular organs that the secretions of these organs are to a large extent regulated.

The maintenance of the body at a uniform temperature is due to

the existence of a remarkable power of adjustment between the amount of heat developed in the interior of the body, on the one hand, and the amount of cooling, on the other, which takes place during respiration by the admission of cold air and the exhalation of water, and at the cutaneous surface by radiation and evaporation—processes, however, which again are under the control of the nervous system. It scarcely need be added, perhaps, that the equalisation of the temperature of the body is dependent on the circulation of the blood: that the more active this is, the more does the temperature of the surface and extremities approximate to that of the internal organs, while at the same time the more rapidly is the general cooling of the body effected; but that, on the other hand, the more feeble the circulation, the cooler do the surface and extremities become, the wider grows the difference between the temperature of these parts and that of the interior of the body, and the more slowly does the internal temperature undergo reduction.

ii.[1] The presence of *abnormal or febrile temperature* is usually attended with various symptoms and phenomena more or less characteristic of the febrile state. The skin gets hot, the pulse and respirations accelerated, the gastro-intestinal functions impaired or modified, the urine and other secretions diminished; and headache and muscular pains are complained of. There is generally also a tendency for the febrile phenomena to assume a remittent character, for paroxysms to recur perhaps once or twice in the twenty-four hours, and for each paroxysm to comprise three more or less distinctly marked stages — namely, a cold, a hot, and a sweating stage. In the first of these the patient feels chilly or cold, shivers or has rigors; in the next his skin gets hot and dry; and in the third more or less abundant perspiration breaks out.

The increase of temperature may vary from the slightest rise above the normal up to 110° or 112°. If it do not exceed 101°, slight febrile action only is present; if it lie between 101° and 103° the febrile condition may be regarded as 'moderate,' if between 103° and 105°, the fever is considerable or 'high;' if it exceed 105° the febrile disturbance is excessive and there is usually considerable danger; from 106° upwards the temperature is frequently termed '*hyperpyretic*,' and (with one or two notable exceptions) if it surpass 107° or 108° death is almost certain to supervene. Febrile temperatures, like normal temperatures, undergo variations; and on the whole (excepting when interfered with by the influence of specific diseases) these correspond to the normal variations, but are exaggerations of them; thus, there is usually a matutinal fall, and an evening rise, and the difference between them generally amounts to 2 or 3 degrees; but it may be much more considerable.

[1] Several corrections and additions to this article have been derived from Dr. Burdon Sanderson's papers on 'The Process of Fever' in *The Practitioner* for 1876.

The skin is usually dry and hot; but it is liable to considerable changes. Thus, not unfrequently, during the early period of a febrile attack, or of a febrile paroxysm, while the internal parts of the organism are preternaturally hot, the vessels connected with the surface of the body, especially of the limbs and head and face, are so contracted as to allow comparatively little blood to reach the surface. This then looks shrunken and dusky, and in certain parts, especially the hands, feet, nose, and ears, may even be much colder than natural. But more or less general heat of skin is present even when the surface displays this appearance of chilliness; and before long the contracted vessels dilate, blood is admitted freely to the comparatively exsanguine parts, which then become plump, congested, dry, and often to the touch pungently hot. This latter condition is usually succeeded after a time by more or less copious perspiration.

The frequency of the heart's beats is always increased; and this increase has usually some relation to the temperature present. Thus, if the latter range from 100° to 101°, the pulse usually ranges from 80 to 90; if the temperature from 101° to 103°, the pulse from 90 to 110; if the temperature from 103° to 105°, the pulse from 120 to 130. With still higher temperatures, the pulse may rise to 140, 160, 180, or even over 200 beats in the minute. The rule, however, which is here laid down, is liable to frequent exceptions—especially in the case of irritable or nervous persons, in whom the pulse, in relation to temperature, is usually disproportionately frequent. The character of the pulse varies. In its typical condition it is more or less large, hard, and bounding, and its trace displays a sudden rise with an almost equally sudden fall, but no indication of dicrotism. This is its state during the height of fever. But during the cold stage it is small and hard; and in the sweating stage large and soft.

Although increase in the frequency of the respirations is undoubtedly one of the normal phenomena of fever, and we often observe the respiratory acts rising to 30 or 40, and in the case of children to 50 or 60, in the minute, the respiration-rate does not bear that close relation to the temperature which the pulse-rate does. It is not uncommon to find the respirations normal in frequency even when the temperature is considerably elevated; and, on the other hand, to find them greatly accelerated in febrile states of the mildest type. When the temperature is hyperpyretic, the respirations are usually very rapid and shallow, and the inspirations often attended with opening of the mouth and of the alæ nasi, and with a sniffing, sipping, or sucking sound. The amount of air respired in fever in a given time is always considerably greater than in health, and although the expired air contains a diminished percentage of carbonic acid, the total quantity of carbonic acid discharged appears to be largely increased.

Thirst is usually present, and often extreme; for the most part there is impairment or loss of appetite; the mouth feels dry and

clammy and acquires a bitter taste; and the tongue tends to be more or less thickly coated and dry. The bowels are generally constipated.

The urine is almost invariably modified in character; it is scanty, high-coloured, of high specific gravity, and deposits on cooling a more or less abundant sediment of urates and perhaps uric acid. But although the bulk of urine passed daily is generally below the healthy average, the quantity of solid matter which is passed with it is usually above the average. The chief increase here is in the urea, of which more than twice as much may be secreted as in health. Dr. Parkes has discovered 885 grains in the day's urine of a patient suffering from enteric fever, Alfred Vogel as much as 1,235 grains in that of one suffering from pyæmia, and Dr. Anstie over 1,600 in that from a case of pleuro-pneumonia. Uric acid also is increased, and may be increased twofold. Again the colouring matter may amount to three or four times the quantity discharged in health, and there is a more or less important rise in the quantities of hippuric, sulphuric, and phosphoric acids and of the salts of potash. On the other hand, chloride of sodium and other salts of soda are diminished. Febrile urine is usually more acid than healthy urine. But although the general fact of the increase of the solid constituents of the urine in fever has been well ascertained, it has also been well ascertained that the discharge of solid matters occasionally falls, sometimes suddenly, sometimes gradually, far below the normal, the urine becoming pale, limpid, and of low specific gravity. Such occurrences, however, are only of temporary duration, and sooner or later are always followed by an abundant discharge of effete matters which had been accumulating in the system.

Among the febrile phenomena referrible to the nervous system may be enumerated, headache, vertigo, delirium, a sense of weariness, soreness or aching in the loins and limbs, and alternations of subjective chilliness with flushes of heat. The sensation of chilliness is exceedingly common, and occurs most frequently at the beginning of a febrile paroxysm. It is often associated with rigors. These are violent tremulous movements of all parts of the body—legs, arms, trunk, head and neck—attended with chattering of the teeth and that pallor or duskiness of surface which has already been adverted to. The patient feels intensely cold, although the interior temperature of his body is probably far above the normal. Rigors appear to be due to the fact that, owing to undue contraction of its arteries, the skin receives less than its due share of blood, and less than its due proportion of the heat which is generated within the body. It is, therefore, either generally, or in certain parts, relatively cold. The feet, hands, nose and ears, indeed, are often livid, shrunken, and actually cold. Rigors may not unfrequently be re-induced by exposing portions of the surface to the influence of the air. In children, convulsions sometimes take their place.

There are several points of interest in relation to fever which may be briefly referred to. First. It is a remarkable fact that, notwithstanding the extreme thirst of most fever-patients, and the large quantities of fluid which they drink, but little water comparatively is discharged from the kidneys or bowels, and often little apparently from the skin. Dr. Parkes suggests that this may be due to the presence in the system of some intermediate waste-product which, like gelatine, is powerfully hygrometric. There is reason, however, to believe that the collective discharge of water is usually much greater than it seems to be, and than it is in health, and that in fact a considerable proportion of the loss of body-weight of fever-patients is due to this cause. If, then, the discharge of fluid from the bowels and kidneys be diminished, it is obvious that there must be augmented discharge from the lungs and skin. And, as regards the skin, it may be observed, that it is only during the cold stage and during rigors, that exhalation is in abeyance, that in the sweating stage the discharge of fluid is obviously excessive, and that even during the hot stage the escape of watery vapour by insensible perspiration is abundant. Second. The condition of the blood is a matter of much interest, yet little of importance is known about it. It seems, however, that after a time the red-corpuscles, albumen, and alkaline salts diminish in quantity—the blood consequently becoming impoverished. Third. That excessive waste of tissue goes on during fever is plainly shown by the condition of the urine, and by the gradual and often rapid emaciation of the patient, which takes place even if he be taking considerable quantities of nutriment. The tissues which especially suffer are the fat, which may almost entirely disappear, and the muscles, which dwindle away in a remarkable degree. But the more permanent tissues, such as the bones, also undergo some diminution. Recent observations show that although the amount of urinary solids discharged daily in fever is often not larger than that discharged in health, it is always much larger than that which would be discharged in health under fever diet. It has also been shown that the increase in the urea precedes the elevation of temperature, and that it is maintained even during defervescence. That this urea is due mainly to the disintegration of the tissues of the body and of the blood-cells, and not, as in health, mainly to the albuminous matters derived directly from the food, is proved by the fact that the salts of potash (which are normal constituents of the living tissues) become excessive in febrile urine, whereas the salts of soda (which abound in the plasma of the blood, and in food, and which in healthy urine exceed those of potash) diminish, sometimes almost to zero, to reappear in excess during convalescence, when the potash salts decrease. The undue disintegration of the red blood-cells is further proved by the excessive presence of colouring matter in the urine. Fourth. The supervention of convalescence is described as taking place in two different ways—either gradually by *lysis*, or suddenly by *crisis*. In the

former case, all the febrile phenomena gradually disappear, and the patient lapses gently into convalescence. In the latter case the progress of the attack is abruptly arrested with the appearance of a so-called 'critical' discharge—copious perspiration, profuse diarrhœa, or abundant secretion of urine loaded with effete matters—by means of which it is supposed that the morbid blood rapidly purifies itself. Fifth. But fever may also end in death. This event, however, can rarely be attributed to the influence of fever alone, inasmuch as fever is always secondary to some specific or other disease of which it is a mere epiphenomenon or symptom. Nevertheless it is obvious, if we consider the physiological and other recognised consequences of fever, that fever itself tends to the induction of death in some two or three different ways. The chief of these appear to be asthenia, blood-poisoning, and the direct influence of sustained high temperature. The continuous excessive waste of tissue, with the consequent emaciation, loss of strength, and impairment of the functions of various organs (which is an essential element of the febrile state), must clearly, if it be not arrested, involve sooner or later a fatal issue. The progress of the hectic fever of phthisis, and other chronic wasting disorders, furnishes a sufficiently apt illustration. This waste of tissue necessarily leads also to the passage through the blood of an excessive quantity of effete products, such as urea and other matters related to urea in composition, some or all of which are poisonous to the system in a greater or less degree. So long as these are freely eliminated by the emunctories, the blood may remain fairly pure, and but little mischief may ensue. We have shown, however, that this elimination is sometimes arrested temporarily. There is no doubt that it is often insufficient to effect the purification of the blood. Under such circumstances uræmic poisoning and typhoid symptoms are only too apt to usher in a fatal issue. Lastly, the persistence of a temperature above a certain elevation is incompatible with the maintenance of life. It has been shown by the experiments of MM. Delaroche and Berger that animals, placed in an atmosphere ranging from 122° to 201° until the heat had killed them, were found at the time of death to have an internal temperature of only 111° to 113° above their natural standard; whence it may be inferred that an elevation to this degree is necessarily fatal to them. We do not, of course, know with any degree of accuracy what is the upper limit of temperature which is compatible with the maintenance of life in the human being. We may say, however, with some degree of assurance, that a persistent temperature above 110° will certainly cause death, and that there is good reason to believe that a temperature of even 107° cannot be supported for any length of time. Death from high temperature is attributed by M. Bernard to a condition of the heart analogous to rigor mortis—the auricles are found full of blood, the ventricles contracted and empty. But the injurious influence of excessive heat is not exerted on the heart alone, but equally on all living tissues, and especially probably

upon simple protoplasm wherever it is distributed. In cases of hyperpyrexia, the symptoms referrible to the nervous system are particularly striking. They usually commence with more or less restlessness and confusion, and tendency to mental disturbance, and lead, through maniacal excitement, muttering delirium or convulsions, to coma and death. Nevertheless, it is by no means clear to what extent these symptoms are referrible to abnormal heat. It is a remarkable fact that frequently, when the advent of death is attended with rising temperature, the heat of the internal parts continues to increase for some hours after death.

It will be readily gathered from the foregoing discussion that the immediate cause of febrile temperature lies in the excessive degradation of the tissues of the organism, and the consequent evolution of their latent heat. The abnormal activity of circulation and respiration, which accompanies fever, alone implies unwonted activity in some at least of the processes which these functions subserve; and the progressive emaciation of the frame, and the continued over-discharge from the lungs of carbonic acid, and presence in the urine (notwithstanding, in many cases, almost total abstinence from food) of an excessively large quantity of those matters which are the result of the degradation of albuminous compounds, clearly demonstrate the character of these processes. This explanation obviously does not touch that further important question, 'What is the cause of the tendency, which is always present in fever, to that preternaturally rapid destruction and oxidation of tissue on which the febrile elevation of temperature depends?' This question, however, notwithstanding its importance, scarcely calls for discussion here.

The *thermometer* of late years has become to the physician almost as important as the stethoscope. It is in general use, and is certainly of extreme value, not only in the diagnosis, but in the prognosis of disease. It is desirable, therefore, to make a few observations in reference to it. A clinical thermometer should be accurate and sensitive, should have its degrees divided into fifths, and be so marked as to be easy of perusal. It should be furnished with an index, consisting of a single fragment of mercury, between one-fourth and one-third of an inch long, detached from the upper part of the mercurial column. For ordinary purposes an instrument, which may be carried in the waistcoat pocket in a case, and marked from 95° to 112°, is sufficient. It is well, however, to be provided, for special purposes, with a thermometer of greater range (say from about 80° to 112°), and probably, therefore, of greater length and comparatively cumbersome. The index should never be allowed to descend into the reservoir and so to mingle with the rest of the mercury; nor should supplementary indices be allowed to detach themselves from the mercurial column. The former accident may be prevented by never violently shaking the index into the

reservoir, and to some extent by the presence of an annular constriction in the channel of the thermometer a little above the reservoir; the second, by always carrying the thermometer horizontal or with the reservoir downwards, and by never permitting the mercury when it has risen into the tube to be too suddenly cooled. Prior to taking a temperature, the index should be brought into the lower part of the tube, at least below the mark indicating the lowest temperature we are likely to meet with. The bulb of the instrument should then be placed in the part selected—in the axilla, beneath the tongue, in the anus or vagina—and retained there sufficiently long to permit of the rise of the mercurial column, and the carriage of the index, to the position corresponding to the temperature of the part. It is important, especially as regards the axilla, that the bulb of the instrument should be tightly grasped, and entirely protected both from the influence of the air and from the contact of the clothes, and that it should be allowed to remain *in situ* from three to five minutes at least. A casual observation is of course often of considerable value; in many cases, however, and especially in fevers and inflammations, periodical observations should be made. Sometimes morning and evening determinations of the temperature are sufficient for all practical purposes. But not unfrequently, especially in very severe and acute diseases, in certain specific diseases, or when the effects of particular forms of treatment are under investigation, periodical observations of much greater frequency are called for.

Hectic fever.—The term 'hectic' is applied to those varieties of fever which attend various diseases of long duration, and more especially such affections as malignant disease, tuberculosis and chronic syphilis, which are characterised by the gradual development of proliferating growths in many organs, or such as caries of bones, disease of joints, and the like, in which purulent discharges are kept up for an indefinite period.

The phenomena of hectic fever are essentially those which have been described in the preceding account of fever. They are chiefly peculiar in their comparative mildness and long duration. The symptoms of hectic come on insidiously, and the febrile condition may be already far advanced before its presence is fully recognised. The patient probably finds himself gradually losing flesh and strength, and becoming disinclined for exertion. He observes that he is disposed to be chilly in the morning; that in the evening, and in a less degree after meals, his hands and feet are hot and dry, and his face flushed; and that he wakes towards the morning with a moist perspiring skin. But his tongue is clean, his appetite good, and, although he may be thirsty and his pulse quickened, his functions generally are properly discharged. At this time a careful thermometrical examination will probably show his temperature to be elevated by two or three

degrees; but also that, as in health, it is lowest in the morning and highest in the evening, and that his indistinctly developed cold, hot, and sweating stages correspond pretty accurately with the usual cycle of the healthy temperature-variations.

As the morbid condition on which the fever depends progresses, the symptoms (although of the same character as before) get more distinctly developed, the patient becomes pallid, his emaciation and debility more obvious, and the febrile character of his illness more striking. The temperature, even now, often does not exceed 103°; but it is liable to occasional higher degrees of elevation, and in its matutinal remissions may sink below the normal. The patient is apt to be chilly in the morning, with cold and livid feet, hands, and nose. In the evening exacerbation the skin gets hot and dry, the palms and soles burning hot, the lips dry and red, and the cheeks flushed with a circumscribed red flush; and towards the morning he wakes to find himself drenched in profuse (colliquative) perspirations. The chief exacerbation occurs almost invariably in the evening, and it is often the only one; but there is occasionally a second, earlier in the day; and generally the ingestion of food, and especially of an ample meal, is followed by more or less marked febrile reaction. With the progress of the other symptoms, the pulse becomes accelerated; and even if it feels sharp, as it may do during the febrile exacerbations, it is positively enfeebled, and undergoing progressive enfeeblement. Even now probably the tongue is clean, perhaps morbidly clean, and the appetite good. There is, however, more or less thirst, the bowels are probably constipated, and the urine (especially in the febrile paroxysms) is more or less scanty, high-coloured, and concentrated.

At a later stage the symptoms are modified, and other phenomena (not wholly referrible to the fever) superadded. The emaciation and debility get extreme, the pulse more and more feeble and rapid, the circulation imperfect; bed-sores form; the fingers grow livid and bulbous, and the skin harsh and scaly; diarrhœa not unfrequently supervenes; the tongue gets dry and fissured or aphthous; the appetite fails; and death from exhaustion presently ensues.

It is remarkable that the intellect is rarely affected, and that, in a large proportion of cases, the patient continues cheerful and hopeful even to the last.

d.—The Typhoid Condition.

The condition here referred to, like fever, is common to many different diseases. When erysipelas, carbuncle, pneumonia, or any other severe inflammation, is tending to a fatal issue; in the later stages of typhus, enteric fever, scarlatina, small-pox, and other specific fevers; towards the fatal close of acute atrophy of the liver, and of uræmia consequent on Bright's disease; and at the corresponding

period of many other affections; typhoid symptoms, or symptoms resembling those of the later stages of typhus fever, are apt to supervene.

The patient becomes excessively prostrate; he lies on his back in bed, with eyes closed, features shrunken and ghastly, and a dull stupid aspect, unconscious or nearly unconscious of everything that is going on about him. His skin is dusky, moist, and sometimes bathed in sweat, which often yields a fetid odour, and is for the most part, especially in the extremities or exposed situations, cold. His lips are dry, black, and probably fissured, his teeth loaded with sordes, his tongue dry, brown or black, and often contracted in all its dimensions. He has no inclination for food, and probably no material thirst, but he has a difficulty (partly due to the condition of his mouth) in swallowing and utterance. His bowels are sometimes constipated, but often relaxed, and the evacuations are apt to be offensive. His respirations are shallow, but for the most part not much accelerated—ranging probably between twenty and thirty in the minute. They may, however, be much more frequent, and are liable to variation. The pulse is rapid and feeble, and tends to get more and more rapid and feeble, and, towards the end, imperceptible at the wrist, and irregular. It may vary at first from 100 to 120, but often attains a frequency of 140 or more, and at the same time assumes an undulating dicrotous character. The first sound of the heart is liable to become inaudible. It may be added that, shortly before death, the superficial capillaries often dilate, the blood accumulates and stagnates within them, the surface acquires a rosy aspect, and a profuse flow of perspiration takes place. Bed-sores are apt to form upon the sacrum and other parts exposed to pressure. The condition of the urine presents considerable variety:—sometimes it is scanty, high-coloured, and loaded with urates; sometimes, on the other hand, it is abundant, pale and limpid, and of low specific gravity. Muscular debility is shown in the tendency which the patient has to lie upon his back, and to sink towards the bottom of the bed. His senses are blunted; often he is deaf; he takes little notice (even if his eyes be open) of surrounding objects; he rarely complains of pain or uneasiness, or acknowledges its presence, and is insensible to conditions which at other times would have caused much personal discomfort; his intelligence is impaired, especially his memory fails; his mind is full of delusions; and he is more or less constantly muttering—he is in a condition of 'low-muttering delirium' or '*typhomania*;' he can probably, however, be recalled to himself momentarily if addressed loudly, and will then half open his eyes, endeavour to do what he is told, and even give an intelligent response; but he soon lapses into the state from which he was aroused; he picks at the bed-clothes; his limbs are tremulous when he endeavours to move them; and his muscular fibres are in constant vibratile movement, giving rise to the condition known as '*subsultus tendinum*;' he passes

his evacuations unconsciously, or allows the urine to accumulate in his bladder. With the advance of the typhoid symptoms, the mind becomes more and more obtuse, and the patient gradually passes into stupor, and thence into profound coma. The temperature presents great variety, dependent in a considerable degree on the nature of the disease upon which the typhoid symptoms supervene:—sometimes, as in Bright's disease, it is a good deal below the normal standard; sometimes, as in the hyperpyrexia of acute rheumatism, it attains an elevation of 110° or more. The typhoid condition is always one of great gravity, and in a large proportion of cases terminates in death.

The collective phenomena of the typhoid state have generally been attributed to the presence of some poisonous matter in the blood. Formerly this was believed to be the specific virus of the disease in the course of which they were developed; or, in the case of local inflammations, some morbific elements generated at the diseased spot and thence thrown into the circulation. It is difficult, however, to understand how it can happen that numerous poisons, distinct from one another, and having different actions in other respects, should yet have the common property of inducing the complex phenomena of the condition under consideration. Another view is now commonly entertained, and has far higher claims to acceptance. It is to the effect that the poisonous matters which circulate in the blood are not the specific elements of diseases, but those products of the disintegration of nitrogenous tissues—urea and the like—which are known, when accumulated in the blood, to have poisonous effects; and which are apt to accumulate in the blood in all those diseases in the course of which typhoid symptoms supervene. The excessive production of these effete matters in various local inflammations, and in the infectious fevers, is an established fact; and as regards some of the latter diseases it has been distinctly proved, not only that the kidneys (even when healthy) often fail to eliminate them in normal quantity, but that even when these organs excrete them profusely, the blood still remains overloaded with them; and further that, in such patients, when they have died with typhoid symptoms, urea in excess has been discovered in the blood. In chronic Bright's disease there is the same accumulation of urea and such like matters in the system; and the typhoid symptoms which come on in its course have long been regarded as of uræmic origin. Indeed in this case it is impossible to suggest any other. The facts, of the presence of uræmia in all cases in which typhoid symptoms are present, and of the dependence of the typhoid symptoms in Bright's disease upon the condition of the blood, are almost conclusive in favour of the dependence of the typhoid condition generally upon uræmic poisoning. The circumstance that, in some cases the accumulation of effete matters is due to their over-production, in others to their retention, does not tend in any degree to invalidate this conclusion.

e.—Collapse. Syncope.

The states of collapse and syncope are in many respects the opposite of that of fever, and are attended with either general or partial loss of temperature. It is important, however, to observe, that a general depression of the temperature of the body may take place, without any of the other symptoms of collapse being present, especially during the remissions of various febrile disorders, or the periods of convalescence from them; and that, on the other hand, profound collapse may occur while the temperature of the internal organs is still many degrees above the normal.

The conditions under which collapse or syncope may supervene are very various. It may occur in the cases above mentioned—namely in the periods of remission of fevers, or during convalescence from them; it may come on in rigors, or when (as in cholera) a high internal temperature prevails; it may be consequent upon the presence of urea, or of extraneous poisons, in the blood. Other causes are mental emotions, more especially such as are of a depressing character; sudden and excessive pain; unwonted distension of tubes (the urethra, the ureters, and the bile-passages, to wit) by foreign bodies; rupture or perforation of internal organs; hemorrhage, and profuse discharges, especially from the bowels; vomiting; severe injuries of all kinds, including those due to the operation of irritant substances or poisons upon the stomach; mechanical obstacles to the cardiac circulation; and many others.

The symptoms of *collapse* are mainly the following:—coldness and pallor of surface, more especially of the extremities and face, which appear shrunken, pinched, and occasionally livid; perspiration, more or less profuse, sometimes limited to the extremities and face, and generally forming large drops in the latter situation; infrequency of the respiratory acts, which are shallow, sometimes scarcely perceptible, often irregular, and now and then sighing or gasping; feebleness of heart's action, indicated sometimes by increased frequency, sometimes by slowness, of the pulse, which often becomes irregular and often scarcely perceptible, or imperceptible, at the wrist; occasionally, hiccough and nausea, or even vomiting; extreme muscular debility; noises in the ears, indistinctness of vision, general soreness or sense of compression, want of breath, giddiness, depression or anxiety, and confusion of thought. In some cases there is restlessness, transient delirium or maniacal excitement, sometimes convulsions, sometimes complete insensibility; in some cases, on the other hand, the mental condition is wholly unimpaired from first to last. In severe cases the patient lies almost motionless, with eyelids half closed and perhaps slightly twitching—looking like a corpse. In true collapse there is probably always more or less marked fall of temperature; and that is the case even when, as in the collapse of cholera and other febrile

disorders, the internal temperature is still abnormally high. But in all cases the extremities and the head lose heat rapidly, and usually become positively cold. In cholera, the thermometer in the mouth or axilla may stand at 90° or less, while that in the rectum marks 105°; and in collapse, the result of severe injury, the temperature even in the rectum may fall (as is shown by Mr. Wagstaffe) as low as 82·15°. Much more commonly, however, collapse-temperatures range between 92° and 97°.

Syncope differs from collapse (of which indeed it is a mere variety) mainly in the suddenness of its access, and the rapidity of its progress, but generally also in the fact that the symptoms of syncope, during their continuance, are more severe than those of collapse. This latter distinction is, however, by no means essential; for, as is well known, syncope may present all degrees of intensity, from a simple sense of faintness to a prostration so profound as to simulate death. The short duration of syncope necessarily precludes the occurrence of any marked depression of the general temperature.

When recovery from collapse or syncope takes place there is always more or less reaction; the surface gets smooth, its colour returns, and a general glow supervenes, the circulation revives, the temperature rises, and other febrile phenomena manifest themselves. And if the collapse have been profound and of long continuance, the consecutive fever may assume serious proportions.

In considering the pathology of collapse there are three factors of that condition the importance of which is especially obvious. These are—depression of temperature, feebleness of circulation, and the condition of the nervous functions. First. The depression of temperature, so far as regards the limbs, face, and other exposed parts, can no doubt be traced mainly to the comparative failure of the circulation in them. But that this is not the sole cause of that depression is obvious from the fact that the internal temperature, instead of rising, as under such circumstances it should normally do, itself tends to diminish, and sometimes diminishes rapidly. It is clear indeed that there is throughout the organism a more or less complete arrest of those disintegrating processes upon which the maintenance of the temperature of the body depends, and presumably also a more or less complete arrest of those vital processes with which these latter are intimately interwoven. Second. The feebleness of the circulation is shown by the obvious weakness, and frequent irregularity, of the heart's action, by the failure, more or less complete, of the pulse at the wrist and in other peripheral situations, and by the concurrent disappearance of blood from the cutaneous surface and other textures. The details of the processes by which the failure of the circulation is induced doubtless differ in different cases. It may, however, be assumed that there is always cardiac debility, and in a large proportion of cases diminished supply of blood to the left side of the heart,

and hence to the vessels which it supplies. In collapse from hemorrhage the latter condition is of extreme importance. And, indeed, it is found, in a large proportion of cases of death from syncope or collapse, that the cavities of the right side of the heart are distended, while those of the left side, and more especially the ventricle, are contracted and empty. In other cases, however (especially if death has been sudden), the left cavities may be found overloaded. Third, and most important, is the condition of the nervous functions. We have pointed out the not unfrequent dependence of collapse or syncope on affections of the mind, and on many other conditions which can be operative only through the medium of the nervous system; and we have enumerated the various phenomena, referrible to the nervous system, which attend and characterise a large proportion of cases. These facts are sufficiently suggestive. But when we look a little more closely into the matter, and consider how many different causes, of different operation, equally produce the same collective phenomena of collapse; how rapidly these phenomena supervene, and how universally the organism is affected by them; how impossible it seems that a smash of the leg, a perforation of the bowel, or an agony of terror, should directly arrest the chemical changes going on throughout the organism, and so reduce the temperature of the body, or should directly influence the action of the heart and arteries, it is impossible to doubt (what many other considerations tend to prove) that all the phenomena of collapse are directly traceable to the operation of the nervous system —not however of the brain or cord, but of that department, namely the sympathetic, which presides over circulation, nutrition, and the functions of the various organs, including those of the brain itself.

f.—Death.

Death is one of the natural terminations of disease; and according to the nature of the disease, or the office, bulk, or position of the organ which may be its seat, the phenomena which usher in that event differ in a greater or less degree. Many of the specific fevers prove fatal with the supervention of typhoid symptoms; many exhausting diseases cause death by simple debility or asthenia, and other affections by the allied conditions of syncope or collapse; diseases of the air-passages or lungs prevent the due aeration of the blood, and are fatal by asphyxia; renal affections lead to the accumulation of urea in the blood, and death by uræmic poisoning; and diseases of the brain induce coma, from which death presently results. In a large proportion of cases, no doubt, various morbid processes concur in the induction of the fatal issue. Nevertheless, a careful consideration of the phenomena of death enables us to bring the different modes of dying, numerous as at first sight they may appear to be, into a comparatively small number of distinct groups.

Bichat, in his '*Recherches sur la vie et la mort*,' speaks of 'death beginning at the head,' 'death beginning at the heart,' and 'death beginning at the lungs.' It is obvious, however, that these are not the only organs from which death commences; and even those who follow Bichat most closely find it necessary to adopt his views with some modification or addition. To us it appears that the principal sources of somatic death are to be found: first, in failure of nutrition; second, in failure of the circulation of the blood; third, in failure of the emunctories to effect the elimination of effete and poisonous matters; and, fourth, in failure of the nervous system to perform its proper functions.

i. *Death from failure of nutrition.*—This may be due to many circumstances, and may arise in the course of many diseases. It may depend on actual deprivation of food, as in simple starvation, or in obstructive disease of the œsophagus or cardiac orifice of the stomach; or on persistent vomiting or diarrhœa, or any other affection (structural or functional) of the alimentary canal, which interferes with the due absorption of nutritious matters at the mucous surface; or on the presence of diabetes, or of rapidly-growing malignant tumours, in which there is a misappropriation of the nutriment received into the blood; or on the presence of inflammatory processes, or febrile disorders, in which excessive waste of tissue takes place without equivalent reconstruction; or, lastly, it may be referrible to the continuance of wasting discharges or losses of blood. The symptoms which precede death in these several cases depend largely upon the special conditions under which they arise, and are therefore liable to considerable variety. But such as are peculiarly referrible to innutrition are, more or less rapidly increasing emaciation and debility, mental languor, feebleness of circulation, and inability to resist the influence of external cold. The general emaciation is not always proportionate to the muscular debility, which, after a while, becomes extreme. The patient probably lies upon his back, motionless or almost motionless, with hands, feet, nose, and ears more or less cold and dusky; breathing feebly and at long intervals, with the pulse barely perceptible at the wrist; sensible, but dull and languid, taking little notice, and not even caring to restrain the escape of his evacuations. With possibly no addition to the symptoms, the general feebleness passes almost insensibly into death—the last indication of life being furnished by the barely perceptible movements of the heart. In simple starvation, there is a general lowering of temperature, which previous to death becomes considerable. Here life may sometimes be maintained for a while by the application of warmth. In disease, however, although loss of temperature is not unfrequent, rise of temperature, under certain circumstances, is of common occurrence.

ii. *Death from failure of the circulation.*—The failure may commence in various situations, may arise from many causes, and may come on

with different degrees of rapidity. It most commonly takes place at the heart; which ceases to propel the blood: either from actual inability or failure to contract upon its contents; or from spasmodic contraction which opposes the entrance of blood into it; or from the compression exerted upon it by accumulation of serum or blood in the pericardial cavity; or from the obstruction of one of its orifices by clot, or some other equivalent cause. It may also depend, upon obstruction of the pulmonary arteries by thrombosis or embolism; or upon general contraction of their smaller branches, as occurs in asphyxia; or upon similar contraction of the smaller systemic arteries, as probably happens in angina pectoris. Death from the causes here referred to may take place quite suddenly—the patient fainting and falling down insensible, and with a gasp or a convulsive tremor yielding up his breath. It may take place less suddenly, yet still rapidly —the victim getting pale, cold, bedewed with sweat, insensible or nearly so, and possibly convulsed, with slow and shallow or gasping respiration, extreme feebleness of the heart's action and imperceptible pulse. When the process of dying from failure of the circulation assumes a more chronic form, the phenomena of collapse are doubtless always present in a greater or less degree, and there is a more or less obvious disposition to depression of temperature; but, in addition, the blood tends to accumulate and to stagnate in the capillaries and veins; dropsy and congestion, with extravasation of blood, are apt to take place; and not unfrequently the parts furthest removed from the influence of the heart (nose, fingers, toes) become gangrenous. Certain differences in the details of dying depend, no doubt, on the situation in which obstruction occurs. It is stated that, if it take place suddenly on the right side of the heart or in the trunk of the pulmonary artery, extreme dyspnœa is one of the prominent symptoms. If, on the other hand, the sudden obstruction occur on the left side, insensibility and convulsions will probably be amongst the earliest of its consequences. Further, if gradual impediment arise on the right side of the heart or in the course of the pulmonary artery or its branches, more or less over-accumulation of blood will speedily ensue in the systemic veins and capillaries; if such impediment arise on the left side of the heart, the consequent congestion will first involve the pulmonary vessels.

iii. *Death from failure of the elimination of effete and poisonous matters.*—The poisonous matters, to which reference is here specially made, are those which accrue in the course of the disintegrating and secretory processes which are always going on, and are mainly, therefore—carbonic acid, which is evolved by the lungs; urea and other nitrogenous matters, which are discharged by the kidneys; and some of the constituents of the bile, which are formed in the liver and under certain circumstances absorbed into the circulation.

The retention of carbonic acid in the blood produces the condition which is commonly known as 'asphyxia,' but is more correctly termed

'apnœa,' and might perhaps still better be designated 'anthracæmia.' It may arise in various ways: from obstruction of the larynx or trachea; from bronchitis or other affections causing block of the bronchial tubes; from disease of the lungs; from mechanical impediment to respiration, due to accumulation of fluid in the pleural cavities; from paralysis or spasm of the respiratory muscles; or from deficient supply of atmospheric air. The symptoms of sudden asphyxia are manifested in their typical completeness in cases of drowning, or of choking from the intrusion of a solid mass into the upper part of the larynx. The sense of dyspnœa is extreme, and violent but futile respiratory efforts take place. But soon vertigo comes on, the respiratory agony diminishes, and the efforts at inspiration get less violent. Gradually unconsciousness supervenes, convulsive movements may occur, and in the course of a few minutes all muscular action ceases. The heart continues to beat, perhaps for a minute or two, after respiration has come to a standstill. During the progress of suffocation, the non-arterialised blood is impeded in its transit both through the small arteries and capillaries of the lungs, and through the corresponding systemic vessels, and the pressure of blood in the systemic vessels becomes augmented. But gradually, the obstruction getting more and more complete in the vessels of the lungs, less and less blood reaches the left cavities of the heart, and consequently less and less is propelled into the arteries; which also by gradually contracting on their contents drive them slowly onwards into the veins. Thus, while the pulmonary veins, left cavities of the heart, and systemic arteries, become comparatively empty, blood is gradually accumulating in the pulmonary arteries, right side of the heart and systemic veins and capillaries, and the general surface gets more and more livid and swollen, and the superficial veins more and more obviously distended. But poisoning by carbonic acid takes place much more gradually, in the course of many diseases, and may extend over a period of many months. The general phenomena, in such cases, are essentially the same as those which have just been detailed, but they are, as it were, more diluted and of less intensity. The surface gets dusky or livid and cool, the veins distended, the right side of the heart dilated, the pulse quick, feeble, intermittent; there is more or less distressing dyspnœa and anxiety; but gradually the struggle for breath grows less painful and violent, the patient gets drowsy, and rambles, and then, passing into a condition of coma and general debility, gradually sinks.

The accumulation in the blood of urea and other matters, which should be eliminated by the kidneys, leads to many important consequences. By their slow action they induce more or less marked anæmia, contraction of the smaller systemic arteries, hypertrophy of the heart, and dropsy, with, sooner or later, impairment of the nervous functions, and especially delirium, eclampsia and coma. It is to them also that are mainly due the collective phenomena to which the name

of 'typhoid condition' has been given, and which (as has been already pointed out) are apt to come on in the course of various febrile disorders and in structural diseases of the kidneys.

iv. *Death from failure of the nervous system to perform its proper functions.*—Diseases of the nervous system are fruitful sources of death. Coma is not only a frequent precursor of death in cases in which the brain is not primarily involved, but it is a common symptom of grave cerebral lesions. In coma there is profound unconsciousness, the patient breathes slowly, irregularly, and stertorously, the saliva and other secretions from his mouth, throat, and air-tubes accumulate in these several passages, and are not expelled; and gradually, partly from this cause, partly from failure of the respiratory muscles, he dies of asphyxia or apnœa. Again spasm, or motor paralysis, may equally produce death by apnœa: in epilepsy spasm of the glottis, in tetanus spasm of the muscles of respiration, may stop the breath and asphyxiate the patient; and the like result may ensue from paralysis of the muscles of the throat and larynx, or of those that govern the movements of the chest. But in these cases it is obvious that, although death may be said to begin from the brain and cord, the patient dies in reality of carbonic acid poisoning. In truth, however, it is not the brain and cord, but the sympathetic system of nerves, which has the direct control over the functions the sum of which constitutes life. It is this which has within its grasp, so to speak, the whole of the circulatory system, the excretory, secretory, and nutritive processes, and even the functions of the brain itself; and it is to this system, therefore, that we should especially refer when we speak of death commencing from the nervous centres. It is to the influence of this system that both paralysis and spasmodic contraction of the heart and blood-vessels are due; it is to the influence of this system alone that the phenomena of shock or collapse (which have been previously described) are directly referrible.

Now, although, in the foregoing paragraphs, we have distinguished several modes of dying, or groups of processes by which death is induced, it is obvious, if we come to compare them among themselves, that they have much in common, and tend to shade the one into the other. Thus, death from coma, or tetanic spasm, resolves itself eventually into death from asphyxia, and death from asphyxia into death from arrest of the circulation of the blood, and this arrest of the circulation of the blood into spasmodic and insuperable contraction of the pulmonary arterioles, which in its turn is referrible to the influence of the vaso-motor nerves. And, indeed (excepting probably those cases in which death is induced by the sudden cessation of the heart's contractions under the influence of shock) the last obvious efforts of life are those of the heart; the patient becomes unconscious, the respiratory efforts cease; yet still we listen for the sounds of the heart, and only when these finally disappear consider life extinct.

But in neither shock nor asphyxia does the heart (at all events as a rule) cease to act because its muscular parietes have wholly lost their aptitude for contracting. In the former case the heart is, as it were, stunned, and may yet, under the influence of artificial respiration, have its movements re-established; and in the latter case, where the heart seems to cease from sheer debility, this debility is rather in the ganglionic centres and nerves, which fail to supply the accustomed stimulus, than in the muscular tissue itself, which may still be made to contract under the influence of artificial stimulation. Hence it would seem that while, as a rule, the cessation of the heart's beats may be regarded as the last observable phenomenon of life, this cessation, as well as that of many other phenomena of organic life, may in their turn be referred to the sympathetic system.

V. THE TREATMENT OF DISEASE.

Details of treatment are discussed, with more or less fulness, under the heads of the various maladies which are described later on in this volume. There are, however, some general principles involved in the treatment of disease which it will be convenient to touch upon briefly here. They come mainly under the heads of 'Hygiene,' 'Prophylaxis,' and 'Remedial Treatment.'

A.—*Hygienic Treatment.*

By the term '*Hygiene*' is meant the science of health, or the study of those conditions on which the maintenance of health depends. Hygiene, therefore, takes cognisance—of the sanitary influences of the atmospheric and telluric circumstances among which we dwell; of the conditions, in relation to density of population, ventilation, drainage, cleanliness and the like, in which we live; of the quality of the water and food which we swallow; and also of our dress and personal habits. The immense importance of attention to this department of medicine is beyond dispute; yet the subject is so vast, and the details which it involves are so numerous, that it would be out of place to engage in their discussion in such a work as the present.

But attention to the laws of hygiene is not less important for the welfare of the sick and convalescent, than it is for the welfare of those who are as yet in the enjoyment of good health; and, indeed, it not unfrequently happens that it is to hygienic measures, rather than to drugs, that we must look for the cure of our patients. Even in this restricted sense, the subject of hygiene is too extensive to admit of satisfactory discussion within the limits of space at our disposal. It must be sufficient (by way of example) to refer—to the important

beneficial influence which a mild balmy air exerts upon those who are suffering from inflammatory affections of the respiratory organs, or from pulmonary phthisis, and upon convalescents from many different diseases; to the injury which cold winds or variable weather inflicts on rheumatic patients; to the essential importance of treating the sick in airy, well-ventilated apartments, and of yet securing an equable genial temperature, of maintaining perfect cleanliness of the patient's person and of everything around him, of removing at once from his chamber all evacuations and other offensive matters, and of taking care that the water which he drinks is free from unwholesome impurity, and the food which he takes is of good quality; and as regards those who are suffering from illnesses which do not necessitate confinement to the house, or those who are recovering, to the need for seeing that their dress is sufficiently protective against the weather, that they are not intemperate in meat or drink, and that they do not keep bad hours, or indulge in any other habits which are or may be hurtful.

But different diseases are obnoxious to different injurious influences, and call for more or less important modifications in the employment of hygienic measures. But these are points which, so far as is necessary, will be dealt with subsequently.

B.—*Prophylactic treatment.*

By '*Prophylaxis*' is signified the preventive treatment of disease. In some respects this subject may be regarded as a part of hygiene, in some as a part of ordinary remedial treatment. We prefer, however (mainly for convenience of discussion), to look upon it as distinct from both. We understand by it the adoption of special measures to prevent the outbreak of special diseases which threaten, or the supervention of anticipated dangers in the course of diseases, and shall briefly consider it under the following heads:—

1. *Prophylaxis in relation to the tendency, inherited or acquired, to disease.*—We know that many persons derive from their parents proclivities towards certain diseases, such as phthisis, gout, epilepsy, and insanity. We know also that many of these affections may be induced, in those who are free from taint of inheritance, by circumstances which tend to impair the general health. We know, further, that exposure to similar conditions is peculiarly apt to act injuriously on those in whom such tendencies already exist. And hence the importance, which is fully recognised, of adopting precautionary measures in reference to such persons: of sending the patient, in whom phthisis threatens, to an equable climate; of restricting the diet, and especially of curtailing the alcoholic drink, of him who has reason to anticipate gout; and similarly with reference to many other affections. Again, there are many diseases of which one attack imparts a liability to subsequent attacks: such are rheumatism, erysipelas and other

inflammations, ague, and intermittent hæmaturia. It is obvious here, again, that it is of the utmost importance, for the welfare of the patient, that he should be protected from those injurious influences which he knows by experience to be the sources of his malady.

2. *Prophylaxis in relation to parasitic, endemic, and infectious diseases.* —Many parasitic diseases are developed under circumstances which are well understood. Tape-worms are derived mainly from the use of the insufficiently cooked flesh of oxen and pigs, and the trichina spiralis from the ingestion of that of the latter animal; the Guinea-worm and the Bilharzia both prevail in certain regions. It is needless to dwell on the importance which the knowledge of such facts has in reference to the prevention of maladies of the kind. Endemic diseases are due to the operation of local causes, a knowledge of the behaviour of which, or of their distribution, clearly furnishes an important clue to their prevention. Thus ague prevails in certain regions, goitre and cretinism in others; and in both instances the occurrence of disease may be prevented by removal to some more salubrious district. In the former case, indeed, the malarious poison may be eliminated or destroyed by effectual drainage. Amongst endemic affections may be included ergotism from the use of spurred rye as food, and lead-poisoning from drinking lead-infected water, the suitable prophylactic measures against which are sufficiently obvious. Epidemic diseases are probably always directly or indirectly contagious; but the several poisonous matters or contagia to which their spread is due, are thrown off from different parts of the organism, gain entrance into the system by different portals, and present in other respects essential differences of habit. The knowledge that the contagium of typhus becomes especially virulent in the presence of over-crowding, and that that of relapsing fever has some peculiar relation with starvation, is of great importance in reference to the measures which should be adopted in order to prevent the development, or arrest the spread, of these diseases; the knowledge also that measles is in the highest degree contagious previous to the occurrence of the rash, and that scarlet fever is comparatively little contagious during the corresponding period, or even for a few days subsequently to the appearance of the rash, is of importance also in reference to the management of these affections; again, the knowledge which we now possess that, while most of the exanthemata are propagated through the atmosphere by the breath or cutaneous emanations, cholera and typhoid fever are only infectious through the intestinal excreta, and their poisons received into the system mainly by means of contaminated drinking-water, supplies us with practical data of the highest value, as to the methods by which their outbreaks should be dealt with. The fact that in most of the diseases coming within the epidemic class, one attack is protective in a greater or less degree against future attacks, is also of great importance in relation to prophylactic medicine.

3. *Prophylaxis in relation to the complications or sequelæ of disease.*—Most diseases bring in their train liabilities to specific incidents of more or less gravity—a fact, the appreciation of which enables us in many cases to take early measures for their prevention or alleviation. The knowledge, that rheumatism is apt to involve the pericardium or valves of the heart; that, in scarlet fever, renal inflammation, albuminuria, and anasarca are liable to supervene; that, in enteric fever, perforation of the bowel may take place at certain stages of the disease; that, in gonorrhœa, the eyes may get infected and destroyed, enables us, in dealing with these affections, to take precautions which are often successful against the supervention of the mischances which have been enumerated.

C. *The remedial and therapeutical treatment of disease.*

The great aim of medical art is the cure of disease. Unfortunately, however, a direct cure—at all events a direct cure by means of drugs—in the great majority of cases is totally impossible. In some parasitic affections, and more especially in such as involve the surface of the body, we may kill or expel the parasites, and so restore the patient to health; by surgical operation or other mechanical measures, we may get rid of foreign bodies or concretions from internal cavities or canals, remove diseased parts, discharge the accumulated contents of normal or abnormal cavities, reinstate displaced organs, dilate contracted channels, or, failing this, make new openings above the seat of obstruction, and so provide passages for the habitual escape of matters that need evacuation; and we may, in a small number of cases, by the use of specific medicines or diet materially alleviate, and even cure absolutely, certain diseases: by arsenic or quinine ague, by mercury syphilis, by colchicum gout, by iron chlorosis, by fresh vegetables scurvy, and by suitable food, possibly rickets and some other affections. But neither by mechanical measures, nor by specific drugs, nor by the restoration to the dietary of matters in which it has been wanting, can we cure the infectious fevers, internal inflammations, carcinoma, degenerative changes, or many of the functional and other disturbances to which the organism is liable. Most of these affections, indeed, take a course peculiar to themselves, tending in some cases to ultimate recovery, in some to chronic ill-health, in some to speedy death. We can do little, often nothing, to arrest them in their progress, or to put limits to their duration. And frequently all that remains to us is, by maintaining the patient's strength, by relieving symptoms, and by taking precautions against the supervention of complications or accidents, to enable him to pass with comparative safety or comfort through his malady—hastening convalescence if the disease be one that does not necessarily end fatally, postponing the final issue if the disease be in the nature of things mortal. The chief general indications under such circumstances seem to be:—1st, to promote the patient's general comfort;

2nd, to support the patient's strength by appropriate nourishment; 3rd, to maintain or to restore the healthy tone of his nutritive functions; 4th, to promote the free action of his emunctories; 5th, to relieve the secondary phenomena or symptoms of his disease; and 6th, to obviate the tendency to death.

1. The rendering the patient's condition as comfortable as circumstances permit, involves of course careful and judicious nursing, and the closest attention to all hygienic and other details of management. The latter will necessarily differ in different cases; but, in illustration of our meaning, we may signalise the following points:—keeping the room dark in eye-diseases, or where it is important to promote sleep; maintaining quiet where in brain diseases and other affections there is acoustic hyperæsthesia; soothing the patient when he is irritable or excited; raising his hopes and spirits when he is depressed or desponding; and when he is in a condition to enjoy such pleasures, to gratify, without worrying, his mind with pleasant surroundings and diversions. It need scarcely be added that patients should always be kept as clean, dry, and free from undue pressure or friction as possible, and should not be allowed to soak in their own discharges; for in a large number of cases, and particularly in those of chronic wasting diseases, of inflammatory and febrile disorders in the typhoid stage, and of paralytic affections of the central nervous organs, there is a peculiar aptitude, especially under such circumstances, for the speedy production of bed-sores.

2. The maintenance of the patient's strength by the judicious administration of food is an essential element in the successful treatment of disease. In most diseases, the tissues of the body disintegrate with unwonted rapidity, and emaciation and debility tend to supervene in a proportionate degree; and in most, this over-rapidity of disintegration is accompanied with loss of appetite, loathing of food, impairment of the nutritive functions, or some other condition, which renders it difficult or impossible to supply to the organism the alimentary matters necessary for its renovation and maintenance. If the obstacle lie in the patient's determination not to take food, as is the case with some lunatics, food must be administered by means of the stomach-pump; if it depend on some mechanical impediment in the œsophagus, stomach, or elsewhere, the food must be administered in such a form (for the most part fluid), and in such quantity, as will permit of its comparatively easy transmission through the constricted, compressed or paralysed part; failing such measures, operative procedure of some kind or other may under certain circumstances become advisable. If the patient's inability to take food depend upon irritability of the stomach, this condition must be remedied by suitable treatment, and all food administered meanwhile must be nutritious, unirritating, easy of digestion, and given in small quantities, and, if possible, frequently. Milk, barley-water, gruel, and the like are

generally best adapted for such cases. Occasionally, however, small quantities of solid but well comminuted food are preferable. If the patient be suffering from inflammation, or fever, or other constitutional conditions, in which utter abeyance of all desire for food exists (associated as such abeyance often is with irritability of the stomach, and even difficulty of swallowing), it is generally advisable, in order to insure the due administration of nutriment, to draw up some scheme for the guidance of the nurse or other attendants,—to determine how much food it is desirable to administer in the twenty-four hours, the intervals at which it should be supplied, and the quantity which should be given on each occasion. A teacupful, a wineglassful, or a tablespoonful of fluid nourishment may, according to the nature of the case and the circumstances which arise, be directed to be administered every two hours, or hour, or half-hour. The quantity given at one time should never (if it can be avoided) be so large as to cause sickness; and the frequency of administration must be regulated in some measure by the quantity which is given at each meal; but we must not be disheartened if we find (as is too often the case) that the patient is unable to take the whole amount of nourishment which we have determined upon as his minimum allowance. In cases of this kind nothing, as a rule, can be better than milk; and generally even those with whom it habitually disagrees can now take it with little difficulty; but it is often necessary to alternate its use with that of other nutritious fluids, such as gruel, barley-water, rice-water, arrowroot, corn-flour or biscuit-powder properly prepared with water or milk, or beef-tea, mutton-broth, chicken-broth or soups, or to replace it by them. Alcohol, in some form or other, is frequently necessary, and must then take its place in the rota. In all cases, whether of inflammation, fever, gastro-intestinal affection, or mechanical obstacle to the entrance of food into the stomach, if the amount administrable by the stomach be insufficient to maintain life, nutritious enemata must be systematically used; and, indeed, this mode of giving food may sometimes be employed temporarily with great benefit, to the total exclusion of that by the mouth, in cases of extreme irritability of the stomach. In many chronic diseases, such as pulmonary phthisis, the appetite often remains good, though perhaps variable and capricious, and hence it is a comparatively easy task to insure the due administration of nourishment. The appetite is generally good, also, during convalescence from wasting disorders, and for the most part may be taken as an indication that the patient needs to be well fed. Although the rules above laid down are generally true, there are occasional exceptions to them; and moreover special diseases in some cases need special modifications of diet. A day or two of abstinence or of starvation is often beneficial, sometimes imperative; and, again, the importance is obvious of the avoidance of amylaceous matters by diabetic patients, and of excess of nitrogenous food by

those who are suffering from Bright's disease. It may be added that persons frequently come under our care who are suffering not only from disease, but from starvation, which may have commenced prior to the commencement of their disease or supervened upon it; and that here especially the good effects of careful attention to the nutritive functions are often strikingly exemplified.

3. It has already been hinted, in the foregoing paragraph, that in many cases it is essential for the successful exhibition of nourishment that the stomach and bowels should be first rendered capable of retaining and acting upon the alimentary matters which are introduced into them. It is, in fact, always important, in the presence of disease, to maintain, or as far as possible to improve, the general welfare of the nutritive functions. To some, and indeed to no inconsiderable, extent this end may be attained, as we have pointed out, by the judicious administration of food. But in a large proportion of cases tonic medicines of various kinds are of extreme efficacy in this respect. It is needless to indicate the numerous cases in which iron, cinchona, cod-liver oil, and the like, act almost as specifics in the cure of disease. We wish, however, particularly to insist on their value in the treatment of many morbid conditions, in reference to which they do not possess obviously specific powers. Among these we may name the various forms of dropsy, and many other consequences or secondary phenomena of organic lesions of the heart, lungs, liver, kidneys, and other organs. In such cases, it is generally necessary to adapt the form of tonic to the condition of the alimentary canal, or it may be to associate with it medicines which tend to soothe or stimulate the mucous membrane, or to act otherwise beneficially on it.

4. The notion of getting rid of the poisonous elements of disease, by eliminating them by the various emunctories or other routes, is an old one. It happens unfortunately, however, that as a rule we have little or no power in thus discharging the proximate causes of disease. It is entirely beyond our competence to promote the separation from the system of the material factors of the various forms of inflammation, of the living elements of malignant growths, or of the contagia of the infectious fevers. Neither can we, by the use of drugs taken into the stomach, cause the elimination or death of parasites imbedded in the organism, or even of such as infest the surface of the body. It is very different, however, with regard to the effete matters which are so abundantly produced in many diseases, which so frequently tend in them to accumulate within the blood, and which so often by their presence therein cause toxæmic symptoms and thus add seriously to the dangers which the patient incurs. For this reason it is generally advisable to maintain, as far as possible, free action of the various secretory organs—the skin, kidneys, alimentary canal, and lungs. In febrile disorders not only is there usually a large over-production of urea and of matters related to urea, but the urine, by which alone

they can be efficiently removed, is usually scanty. It is obviously desirable therefore, in these cases, to promote the flow of urine—a result which may generally be best attained by allowing the patient to drink freely. In gout, a somewhat similar accumulation of effete matters, and especially of urate of soda, takes place in the blood, and consequently here again eliminative treatment is indicated. But it not unfrequently happens that poisonous matters accumulate in the blood in consequence of structural disease of the organs by which they should be separated. In disease of the kidney, urea and other waste nitrogenous matters are retained in the blood, in disease of the liver the elements of bile, in disease of the lungs carbonic acid. Under these circumstances unconquerable obstacles frequently exist to the purification of the blood. Still, good may often be effected, if not by promoting the eliminative action of the implicated organ, at any rate by encouraging the vicarious action of other organs. In renal disease much benefit is generally obtained by the regulated use of drastic purgatives, and by promoting profuse perspiration; and in liver disease with jaundice, by encouraging diuresis. Again many substances, poisonous and other, which occasionally gain entrance into the organism, tend, like urea, and other effete matters, to be thrown off—sometimes by the kidneys, sometimes by the lungs, sometimes into the parenchyma of certain organs. Their discharge may often be hastened by appropriate measures. It is an important statement that lead and mercury, which have an aptitude to be deposited in certain of the tissues, can be removed thence by means of iodide of potassium; with which they are said to unite in the organism, and in company with which then to escape with the urine. But eliminative treatment is by no means called for in all diseases; and, even where it is indicated, it must not be assumed that the emunctories must be powerfully stimulated into action, still less that we should act violently upon all at the same time. Here, as in other cases, we must be guided in our efforts by the nature of the case with which we have to deal, and by the phenomena which manifest themselves during its progress.

5, No inconsiderable part of the duties which a medical practitioner is called upon to perform consists in the treatment of the secondary phenomena or symptoms of disease—in relieving pain or uneasiness, in giving sleep, in soothing irritability or anxiety of mind, in promoting or checking the action of certain organs, in removing or dissipating matters which, from their position or quantity, interfere with the due performance of functions that are important to life or health. And it is certain that, if we do not by such measures actually cure the primary disease, we often make life tolerable, we are often successful in prolonging life, and not unfrequently succeed in prolonging it until the disease, which would otherwise have carried the patient off, itself subsides, and by its subsidence leaves him convalescent. The importance of relieving pain in acute inflammation of the peritoneum or pleura, or

in enteritis, and in various forms of neuralgia, is fully admitted by everyone. The necessity of giving sleep in traumatic delirium, in the wakefulness which sometimes precedes the outbreak of acute mania, and in many febrile and organic diseases, is equally recognised. The relief of spasmodic action of the voluntary muscles in tetanus, or of the involuntary muscles in spasmodic stricture of the urethra and various other tubular organs, is often a matter of urgent need; as also, on the other hand, is the stimulation of an inactive organ—of the heart under certain conditions, or of the flaccid uterus after parturition when profuse hemorrhage is taking place. The last examples which we shall adduce are supplied by the removal, whether by tapping or by medicinal means, of dropsical accumulations in serous cavities; and the dissipation of effusions, tumours, or foreign bodies, which by their position compress or interfere with passages—such as the larynx, or bowel—the patency of which is necessary for the maintenance of life.

6. To obviate the tendency to death is to a great extent implied in the foregoing discussion. In a sense it is the principal aim of all medical treatment. The expression, however, is generally employed in reference to the duty which devolves upon us at the time when death appears to be imminent, and when the exact nature of the process by which death will be brought about becomes more or less clearly indicated. On a former page we have discussed the various modes of dying; and we must refer to what was there said for the special indications for treatment furnished in the several cases there enumerated.

PART II.

SPECIAL PATHOLOGY.

SPECIAL PATHOLOGY.

CHAP. I.—SPECIFIC FEBRILE DISEASES.

I. INTRODUCTORY REMARKS IN REFERENCE MAINLY TO THE INFECTIOUS FEVERS.

A. *Specific Origin and Spread of Epidemic and Endemic Diseases.*

THE diseases, to which the following remarks are intended to be introductory, are for the most part linked together by the possession of certain striking characteristics. They originate severally in definite specific causes, the prevail endemically or epidemically, and are in large proportion infectious or contagious.

1. *They originate in specific causes.*—To this subject we shall presently recur; meanwhile, the truth of the statement here made is proved by the fact, that the several diseases of this group never pass the one into the other, or (notwithstanding that, within certain limits, they may present variations of character) lose their specific identity—that while malarious poison never causes small-pox, typhus, or scarlet fever, so the specific poison of either of these latter affections never gives origin to ague, or to any other disease than that from which it was derived. Small-pox produces small-pox, typhus typhus, scarlatina scarlatina; and ague arises under special conditions which are productive of ague and of ague alone.

2. *They prevail endemically or epidemically.*—The term 'endemic,' as applied to disease, signifies the prevalence of disease among a people. For the most part, also, it implies its limitation within certain restricted areæ, its dependence on local or localised causes, and a tendency to persist in the district which it affects. The term 'epidemic,' on the other hand, implies that the disease of which it is used falls as it were suddenly upon a people, and generally implies, further, that it spreads widely and rapidly, and that its prevalence is of limited duration. Goitre is the very type of an endemic disease, influenza perhaps the most characteristically epidemic of all epidemic diseases.

It is important, however, to observe that epidemic diseases comport themselves in many different ways, and that the epidemic and endemic conditions not unfrequently pass the one into the other. Influenza, and it may be added small-pox, scarlet fever, measles, and other like affections, when occurring for the first time in an unprotected community, diffuse themselves generally with marvellous rapidity. Typhus and relapsing fever, virulent though they be, limit their spread mainly to those who are under certain defective sanitary conditions. Cholera, though distinctly epidemic, diffuses itself mainly by irregularly scattered local outbreaks — a peculiarity still more markedly belonging to enteric fever and to diphtheria, which, moreover, are apt to persist in an endemic form, in the localities into which they have been introduced. Further, many affections, which are now more or less characteristically endemic, or epidemic within restricted areæ, have been, or are liable to become, epidemic in the wider sense of the word, under certain ill-understood conditions; among these may be enumerated leprosy, syphilis, plague, and yellow fever.

3. *They are in large proportion infectious or contagious.*—It was formerly largely believed, that epidemic disease was the result of the operation of some mysterious influence, diffusing itself like a vapour over the surface of infected regions, involving equally the whole population, modifying the general health, tincturing the already prevalent diseases, and causing among those who were predisposed to it the specific epidemic attack. This view was once held with regard to syphilis itself — a disease which is now known, like hydrophobia and glanders, to be imparted only by direct inoculation. It is even now held by many in respect of influenza—a malady which is one of the most eminently contagious of maladies, and in this respect allied with small-pox, scarlet fever, and measles. That the origins of cholera and enteric fever were long enshrouded in mystery is not surprising; yet even in the case of these diseases there is now scarcely room to doubt their diffusion by means of specific contagia. And, indeed, (though it has not yet been distinctly proved of every epidemic affection) the progress of pathological science leaves little room for doubt, that all truly epidemic diseases are communicable directly or indirectly from the sick to the healthy, and that their spread is due solely to the operation of a specific virus which the former yield and the latter absorb. Endemic affections, on the other hand, are not necessarily infectious; and some — such as ague and goitre — seem clearly to originate in certain poisonous matters, developed, or existing, in the soil of the localities which they affect.

4. *Behaviour of contagia within the organism.*—The virus or contagium of an infectious fever, having gained entrance into a susceptible body, apparently remains dormant in it for a time, which varies according to the nature of the fever, and is termed the period of '*latency*' or '*incubation.*' To this succeeds the period of '*invasion,*'

during which the first symptoms of the disease manifest themselves. And on this soon supervenes, in its turn, the period during which the specific symptoms become declared. This, in the case of the exanthemata, is termed the '*eruptive*' period. In other varieties of infectious fevers, the period of invasion, and that which corresponds to the eruptive period, are for the most part indistinctly divided. In most cases, after the symptoms have endured for some definite time, they begin to abate—the period of *decline*, or *defervescence* comes on. On this *convalescence* ensues and the patient is presently restored to health.

In order to impart disease, the contagium must enter into the system. But the mode of its entrance, and the route by which it enters, differ in different cases. Some contagia—such as those of syphilis, glanders, hydrophobia, and vaccinia—can be introduced only by direct inoculation, effected by placing them in substance on some delicate mucous surface, or by inserting them beneath the epidermis; some are carried by the atmosphere, are inhaled, and enter through the respiratory mucous membrane; some are introduced mainly with the food, and act primarily on the gastro-intestinal tract. Many of the diseases which are ordinarily conveyed by the air, or by food, have been found to be also communicable by inoculation; and it seems not improbable that, under favourable conditions, all such diseases might be thus imparted.

In some of the inoculable diseases—such as syphilis and small-pox—a specific pimple gradually rises at the point of inoculation; specific affection of the lymphatic glands next above speedily ensues; and, at or about the time when these have attained their full development, febrile symptoms supervene, to be followed in a short time by the characteristic rash. In vaccinia the same sequence of events takes place, with the exception that the febrile symptoms are not succeeded by any specific cutaneous eruption. In these cases, the period of the development of the primary pimple or pock, and of the affection of the neighbouring lymphatic glands, corresponds accurately to the period of incubation of natural small-pox, or of other infectious fevers not acquired by inoculation. It is reasonable to believe that, what occurs in these particular affections, during the period of incubation, occurs during the same period, with some modifications of detail, in others—in other words, that specific local processes (followed by specific affection of the next lymphatic glands) take place in all of them, during the period of incubation and preliminary to the general diffusion of the poison, at the spot or spots at which the virus enters the organism. It is not improbable that the specific lesions of diphtheria, cholera, and enteric fever, are to be regarded as the immediate consequence of the local action of the specific poisons of these diseases, and as corresponding therefore to the syphilitic chancre, or the primary pock of inoculated variola, and not to the eruption of the generalised disease.

The period of general diffusion follows; the infected lymphatic glands shed specific elements into the blood, with which they are distributed throughout the organism, to sow themselves in, or to infect, those parts of it which offer a suitable soil for their further development or growth. Various constitutional phenomena, due to the effects of the poison upon the blood and tissues, attend their diffusion; but, in addition to these, various specific lesions of particular parts ensue, which are more or less characteristic for each form of disease. In many cases (the exanthemata) a rash appears upon the skin; in some the tonsils, in some the salivary glands, in some the respiratory tract, in some the alimentary canal, in some certain other internal organs, are mainly involved. It is obvious, from the above account, that the contagious matters of the contagious diseases must at some time or other be contained within the blood. The blood, indeed, in some cases and under certain conditions, is undoubtedly infectious. But, for the most part, this fluid rapidly purifies itself of the poisonous elements which enter it, discharging them mainly into those organs or tissues, or at those surfaces, which are the seats of the specific lesions of the diseases to which they belong, and which consequently become surcharged with infectious matter.

During the progress of a contagious disease, the contagium which gave it origin undergoes enormous development within the organism. An inconceivably minute quantity of the variolous poison, placed beneath the skin, results in the formation of a pock, which itself contains an infinitely larger amount of poison than was introduced in the first instance, and subsequently in the formation of thousands of pocks scattered over the general surface, each one of which is as fully charged with contagium as was the first. There can be no doubt that, in other diseases besides small-pox, this development of contagium goes on during the whole period of ingravescence—beginning at the seat of its introduction, continuing in the lymphatics and probably in the blood, but taking place with especial energy in the cutis in exanthematic diseases, and in connection generally with specific lesions.

In the majority of cases the poison, which is thus manufactured within the organism, is discharged from it in greater or less abundance, and serves to propagate the disease of which it is the specific cause. This discharge, which occurs mainly in connection with the seats of specific lesion, takes place at different periods in different diseases, and necessarily also from different surfaces. Thus, the contagia of cholera and enteric fever are discharged with the alvine evacuations; those of measles, hooping cough, and influenza are yielded from the respiratory surface; that of scarlet fever escapes probably from the throat and skin, that of hydrophobia with the saliva or oral mucus, that of glanders mainly with the nasal secretion, and that of syphilis with the discharges from its specific sores.

It is very remarkable, that the majority of contagious fevers end

in recovery—that the poisonous matters which they engender, either die out, or escape from the body by one or other of the routes which have been enumerated. This latter process has been compared to the discharge of urea, or other effete matters, by the emunctories. But it is obviously of quite a different character; for—to take small-pox again as an example—there is not simply a discharge from the diseased surface of matters which had accumulated in the blood, but there is an actual manufacture of poison going on at each spot of disease. There arises, further, a remarkable condition of the organism, by which its susceptibility of the specific poison is destroyed; for not only does the poison within it die out, but the system refuses to reabsorb any of the abundant poison which it manufactures, and remains for many years, it may be for life, free from liability to become again affected.

5. *Behaviour of contagia external to the body.*—There is a time during which contagia exist external to the body. How do they then comport themselves? It is clear that, in this respect, they present as important differences among themselves as they do in their influence over the body. The contagium of influenza is remarkable for its amazing diffusibility; that of typhus clings as it were around the patient and is readily destroyed by atmospheric dilution; that of scarlet fever remains dormant for months in articles of clothing; that of small-pox, or of vaccinia, may be preserved for years between two pieces of glass, or concreted upon an ivory point. But the most remarkable peculiarities are presented by the contagia of enteric fever and cholera. In both cases, the specific poison is yielded by the bowel, and escapes with the fæces; and in both, probably, the poison is innocuous at the moment of escape, and only acquires virulent properties after the lapse of some time—in the case of cholera, after the lapse of four or five days.

6. *Nature of contagia.*—Having briefly considered the dependence of epidemic diseases on specific contagia, and the modes by which these poisons enter the body, act upon it, and finally get discharged from it, together with some of their peculiarities of behaviour outside the organism, it remains to discuss the question of their nature. In reference to this subject, we must not lose sight of some of the important facts with regard to contagion which have been adduced; we must bear distinctly in mind, that the virus of one disease produces that disease only and never any other; that a virus received into the body multiplies indefinitely within it; that it leaves the body, not by the organs provided for the separation of effete matters, but by a process of efflorescence or multiplication, taking place in certain situations and modes, which are characteristic for each disease; and that external to the body it comports itself in various manners, of which some (as in cholera and enteric fever) evidently imply progressive developmental changes. It seems impossible that these conditions can be fulfilled by any element, or any combination of elements unendowed with life. No

inorganic solid, still less any inorganic fluid or gas, no dead organic compound, could thus multiply itself either within or without the body, or thus affect the body in its progress through it. It is impossible to conceive of a bubble of sulphuretted hydrogen, a drop of gin, a fragment of marble, or a grain of morphia, multiplying itself a thousandfold within the system, making for its discharge some special route, and leaving the system henceforth incapable of its further production. Nothing analogous to this has been shown to exist in the whole range of inorganic or organic chemistry. The facts, however, are all compatible with what we know of the development and behaviour of organised beings, and especially of such as are lowest in the scale of life. We know how, when the spores of fungi get deposited in a suitable soil, they grow and multiply and rapidly pervade it until they have exhausted it; how, each fungus fructifies according to its specific character, and yields innumerable spores, which become widely diffused, and though retaining their specific characters and their vitality under apparently the most adverse circumstances, remain dormant until the opportunity for their development offers itself. The above, however, is not the only argument in favour of the dependence of infectious fevers on living organisms. Others of still greater value remain to be adduced. (*a.*) We know that many diseases, among which may be mentioned tinea tonsurans, tinea favosa, tinea versicolor, scabies, and those in which trichinæ and hydatids are present, are actually due to the presence of animal or vegetable parasites; and that the behaviour of the living contagia in these cases manifests at least as great variety as does that of the virus of the infectious fevers. (*b.*) The important experiments, first made by Chauveau, with regard to the infectious fluids of cow-pox, sheep-pox, and glanders, and since repeated in the case of cow-pox by Dr. Burdon Sanderson, show clearly, that the contagious element is not uniformly diffused throughout these fluids, that it does not reside either in the inflammatory corpuscles which they contain, or in the dissolved constituents, but in certain minute protoplasmic particles or living bodies which, at the period of their chief infectiveness, they contain in great abundance. (*c.*)[1] Specific parasitic growths have actually been detected in connection with several of the diseases in question, under circumstances which leave little doubt that they are the actual contagia, or specific elements, of these diseases. The most important observations relate to relapsing fever, and anthrax or the splenic fever of cattle. In relapsing fever, a form of bacterium named 'spirillum' was first detected in the blood by Dr. Obermeyer in 1872. Spirilla are moving spiral filaments of extreme tenuity, and measuring from $\frac{1}{1500}$ to $\frac{1}{500}$ inch in length. They are found in the blood in connection with the febrile paroxysms only—

[1] See, in relation to what follows, Dr. W. Roberts's lecture, published in the *British Medical Journal* of Aug. 11, 1877; and Dr. Sanderson's lectures on the 'Infective Processes of Diseases' in the same journal of Dec. 1877 and Jan. and Feb. 1878.

making their appearance in it shortly before the rise of temperature commences, and disappearing from it just before the occurrence of the crisis. They vary in number from day to day during the persistence of fever; and disappear absolutely during the remission. The above facts have been confirmed by many subsequent observers; and recently by Dr. Heydenreich of St. Petersburg, who also shows experimentally that spirilla are very short-lived at febrile temperatures, and even at the normal temperature of the blood, and that there is good reason therefore to believe that their variable prevalence in the same attack is connected with the development and disappearance of successive generations. Recent experiments in regard to the inoculability of relapsing fever tend to substantiate the belief that spirilla constitute its contagious element; for the disease can be readily imparted by the blood of a patient in the febrile paroxysm, but not by that of the same patient during the apyrexial period, nor by his secretions at any time of his illness. In splenic fever, which is communicable only by direct contagion, and occasionally spreads in this way to man, peculiar organisms are always to be found in the blood, lymphatic glands, and spleen during the height of the disease. These are motionless, rod-like, bacteria about the $\frac{1}{3000}$ of an inch in length, and have been named 'bacilli anthracis.' Quite recently Dr. Koch of Woolstein has performed a series of experiments which throw a very important light on the character and behaviour of these organisms. He has cultivated them externally to the body, and finds, that under suitable conditions they grow into branching filaments of considerable length; that the filaments, which are at first structureless and transparent, after a time become studded with small dots; that these gradually increase in size until they form oval spores, which presently, on the breaking down of the filaments, get detached. He also finds that these spores, like all spores, are bodies of robust vitality and comparatively indestructible; that under favourable circumstances they elongate into rods; and that under the continuance of such circumstances the rods themselves are capable of indefinite multiplication by fission. He further finds that mice are highly susceptible of the disease; and that while disease is not imparted to them by the bacillus in its filamentous form, it is readily given by either spores or rods. Further, the spores appear always to become rods in the organism, and the mycelial stage is never attained there; so that the rod-like form is the only one under which they seem to live parasitically.

The arguments in favour of the dependence of the specific contagious diseases on living organisms, apart even from the remarkable series of observations which have just been adduced, seem almost conclusive. It might still, however, have remained a question whether these living organisms were animacules, as some have supposed, vegetables as others believe, or particles of the living tissues of the patient, as Dr. Beale thinks, endowed with specific properties. It need

scarcely be said that these recent discoveries go far to give a positive solution to this question, and at the same time to confirm the belief of those who maintain that the specific fevers—in other words, their specific causes—never originate *de novo*. If contagia be lowly vegetable organisms, it is easy to understand how it is that they present so many characteristic differences of behaviour, how it is that they are infectious at different periods of disease and under different circumstances, and how (if like the bacillus anthracis they pass through different phases of living, of which some are parasitic, some non-parasitic, some infective, some innocuous) they may from time to time pass an innocent or dormant existence externally to the body until favouring conditions bring them again to active infective life—how in fact (as in cholera, plague, typhus, and relapsing and enteric fevers) they may, from time to time and under special combinations of circumstances, appear to undergo spontaneous development.

7. *Septicæmia.*—In connection with the subject which has just been discussed, as well as on account of their intrinsic importance, it seems desirable to call attention, however briefly, to the observations in regard to septicæmia which have been made during the last few years abroad, and by Dr. Sanderson, Prof. Lister and others in our own country. It seems now to be well ascertained that septic bacteria, or their invisible spores, are largely diffused throughout nature, but mainly in connection with water and watery vapour; that they rapidly attack all organic infusions and all dead or dying animal or vegetable matters which are not specially protected from them; and that they are in fact the essential agents in all putrefactive processes. It has also been ascertained that these organisms tend to breed in our bowels, and on those other mucous surfaces to which the air has ready access, and especially to attack external wounds or ulcers. And it has further been ascertained, beyond the possibility of doubt, that when such parts become foul and unhealthy, and their secretions fetid, these conditions are always associated with an enormous development of septic bacteria, which are then found mainly in the diseased tissues, but also in the adjoining lymphatics, and generally to some extent in the circulating blood. It was a natural inference, that under these circumstances the bacteria were not only important (if not essential) agents in the local unhealthy processes, but that they were largely (if not solely) instrumental in causing the constitutional disturbance which was associated with them, and in the propagation of unhealthy inflammation from patient to patient—that they were, in fact, contagia in the sense in which we have hitherto employed that word. Further investigations have partly confirmed, partly corrected this inference. They have confirmed it, by demonstrating that the development of bacteria is essential to the production of the local putrefactive changes and general febrile symptoms, and to the communicability of the morbid process. They have corrected it, by proving that the bacteria (unlike

true contagia) do not multiply within the organism, and that their injurious influence over the system, when they exert any, is due not to themselves, as such, but to a poison which they generate under certain circumstances.

This septic poison is soluble, and can be obtained in solution, entirely free from bacterial or other organisms, and from putrefactive taint or tendency. It is of extreme virulence (its effects being proportionate to its dose), and it has exactly the same effect on the system as the material containing bacteria from which it is obtained. When injected into the tissues of the dog it gives rise to the following phenomena:—the animal first shudders and then moves about restlessly from place to place; its gait becomes unsteady, and in a short time it staggers and falls on its side; in the meanwhile, vomiting and violent tenesmus, followed by the discharge first of fæcal and subsequently of mucous dejecta, take place; and then, if death do not ensue, the symptoms quickly subside and the animal soon recovers its normal appetite and liveliness. During the attack the temperature rises gradually to about a couple of degrees above the normal, and then, whether recovery or death ensues, gradually falls. Immediately before death the fall is rapid. In fatal cases, small extravasations of blood are found beneath the endocardium (mainly of the left ventricle), pericardium and pleuræ; the abdominal organs generally are congested; but the mucous membrane of the stomach and small intestines is hyperæmic to an extreme degree, and the spleen is large and infiltrated with blood. The blood is darker than natural, owing mainly to the fact that the red-corpuscles are partially dissolved, and that their colouring matter is diffused to some extent through the plasma. Extreme anæmia is generally observed after recovery.

The application of the above facts in explanation of the phenomena of septicæmia in the human being is sufficiently obvious. The symptoms of human septicæmia, which however is rarely uncomplicated, will be considered further on. Meanwhile the fact that the septic poison is of local manufacture, and that its continued presence or its increase in the system is due, not to self-development in the system, but to repeated or continuous dosage, is of such supreme importance, both on scientific and on practical grounds, that it can scarcely be impressed too strongly on the mind.

In the above account of septic poisoning, we have insisted on the distinction between septic bacteria and the true contagia—namely, that in the former the organisms undergo development in some limited area where they evolve a material, unliving, poison, which is thrown into the system and thus acts injuriously upon it, while in the latter the organisms themselves enter the system, undergo development within it, and thus produce their characteristic effects. But while admitting the reality of the distinction, is it, we may ask, a fundamental distinction? It can scarcely be supposed that the true contagia act

otherwise on the body than by some poison which they yield or produce; and if this be the case it can only be a matter of subordinate scientific importance, whether the contagia which evolve poison multiply within the blood as in relapsing fever, in the skin (mainly) as in small-pox, or in ready-formed ulcerated surfaces as in septicæmia. These remarks not merely have reference to the organisms of septicæmia on the one hand and the contagia of the exanthemata and continued fevers on the other, but they bear upon the question of the nature of the contagious element, in diphtheria, enteric fever, and some few other diseases, in which lowly organisms (micrococci or bacteria) have been detected in connection with the characteristic local lesions, but in which the relations between these organisms and the disease have not yet been satisfactorily determined.

B. *General Rules to be observed in the Management of Epidemic or Contagious Diseases.*

We can, as a rule, do little or nothing medicinally for the direct cure of the infectious fevers. So far as the patient is concerned, we can only treat symptoms as they arise, support his strength by suitable nourishment, promote the action of his excretory organs, and take precautions against the supervention of complications. It is, however, a most important duty of the medical man to prevent the spread of these diseases. The measures to be adopted for this end will differ to some extent, according to the character of the disease he has to deal with, and according to the properties and peculiarities of the contagium on which it depends. The following general rules, partly derived from 'Suggestions by the Society of Medical Officers of Health,' partly from other sources, may be laid down as generally applicable:—

1. The patient should be at once separated, as efficiently as circumstances permit, from the other inmates of the house, and if possible placed in a top room, and have that floor devoted to him and his attendant.

2. All bed-curtains and other hangings and carpets, and all articles of dress and the like in wardrobes and cupboards, and all unnecessary articles of furniture, should be removed thence.

3. The room should be well ventilated; windows should be kept partly open, communication with the chimney free, and if the weather or size of the room permit, the fire burning. The floor should be sprinkled daily with disinfectant fluid and cleansed.

4. The door should be kept closed, and a sheet kept wet with a solution of carbolic acid, chloride of lime, or Condy's fluid, hung outside it so as to cover every crevice.

5. Everything that passes from the patient (spit, vomit, urine, fæces) should be received into vessels containing either of the above solutions; and an additional quantity of solution should be added to

the vessel, before removing it from the room and emptying it into the closet. All superabundant food or drink, and all scraps, should be similarly treated, and under no circumstances partaken of by other persons.

6. Pieces of rag should be used for wiping discharges from the nose or mouth, and burnt immediately after use.

7. All cups, glasses, spoons, and such-like articles, used in the sick-room, should be placed in some disinfectant solution before leaving it, and subsequently washed in hot water.

8. All bed and body linen should at once, and before leaving the room, be put into a disinfectant solution. After remaining in this, for at least an hour, they should be boiled in water.

9. The patient's person and bed should be kept scrupulously clean; and when, during the progress of the disease, scales or crusts form upon the skin, their diffusion should be prevented by smearing the surface daily with oil.

10. Nurses in attendance should, if possible, be such as have already had their patient's disease; their dresses should be of cotton or of some other washable material; they should keep their hands clean, using carbolic acid soap, or adding Condy's fluid to the water in which they wash, and should as far as possible avoid inhaling the patient's breath, or other emanations from his person or discharges. They should remain with the patient; or if compelled to leave the room, leave it under proper precautions; and under no circumstances mix with other members of the household.

11. Visitors should not be allowed, or if allowed, should conform, as closely as circumstances permit, to the conditions required of the ordinary attendant.

12. The medical attendant should remain no longer than necessary in the sick room, and expose himself as little as possible to contamination; should wash his hands before leaving; hold as little subsequent communication as possible with the inmates of the house; and never go direct, or without proper precautions, from the infectious to other patients.

13. The patient must not be allowed to mix with the rest of his family, until all peeling of the skin has ceased, or until all specific phenomena of disease have disappeared, and until he has been well purified by the use of warm baths and carbolic acid soap or Condy's fluid. Clothes used during the time of illness, or in any way exposed to infection, must not be worn again until they have been properly disinfected.

14. When the sickness has terminated, the sick-room and its contents should be disinfected and cleansed. This should be done in the following manner:—Spread out, and hang upon lines, all articles of clothing or bedding; well close the fire-place, windows, and all openings; then take from a quarter to half a pound of brimstone, broken into small pieces; put it into an iron dish, supported over a

pail of water, and set fire to it by putting some live coals upon it; then close the door, stopping all crevices, and allow the room to remain shut up for twenty-four hours. At the end of this time the room should be freely ventilated by opening doors, windows, and fire-place; the ceiling should be whitewashed, the paper stripped from the walls and burnt, and the furniture and all wood and painted work washed with soap and water containing a little chloride of lime. Beds, matresses, and other articles which cannot well be washed, should, if possible, be submitted to a heat of from 210 to 250 degrees for two hours or more, in a disinfecting chamber.

15. The house in which the patient, suffering from infectious disease, resides, should, during his illness, be well ventilated and kept very clean; all sinks and water-closets should be in good order, and have solution of sulphate of iron, carbolic acid, or chloride of lime, poured into them daily; dustbins should be regularly emptied, all offensive accumulations removed or disinfected by the free use of chloride of lime; and all water-butts and cisterns kept clean and well covered. Indeed, the greatest possible care should be taken to prevent any kind of contamination of drinking-water.

For the purposes of direct disinfection, many different substances may be employed. The following are among the more commonly useful:—Sulphate of iron, one pound to the gallon of water; chloride of lime, one pound to the gallon; carbolic acid (No. 4), a quarter of a pint to the gallon; Condy's red fluid diluted with fifty times its bulk of water; the green fluid with thirty times its bulk of water. Chloride of lime, carbolic acid, and Condy's fluid are, on the whole, preferable for disinfection in connection with the infectious fevers. For the disinfection of linen and other wearing apparel, chloride of lime should be avoided on account of its corrosive quality. Solution of carbolic acid, or of Condy's fluid, is preferable.

II. INFLUENZA. (*Epidemic Catarrh.*)

Definition.—A contagious, catarrhal affection of the respiratory tract, of short duration, but attended with much prostration, and occurring, for the most part, in widespread epidemics.

Causation and history.—Influenza is one of the most mysterious, and at the same time one of the most interesting, diseases with which we are acquainted. The obscurity of its origin; the swiftness with which it spreads throughout a district into which it has been introduced, and passes from one city to another city, from one country to another country, thus involving entire continents within very brief limits of time; the shortness of its stay in any locality, which rarely exceeds six weeks or two months; the suddenness and completeness of

its disappearance; and the irregularity of its epidemic visitations; all combine to render it the most typical of all epidemic diseases. Its origin and diffusion, therefore, have not unnaturally been sought for in some occult telluric, atmospheric, or electrical condition, some widespread morbific influence external to, and independent of, the frames which it affects. On the other hand, experience has shown that its prevalence is altogether independent of climate and season, and has no relation to defective drainage or other local sources of sanitary evils. But its conveyance has frequently been traced from locality to locality by the direct agency of those who are suffering from it, and its diffusion in fresh localities from these infected immigrants as centres. It is certain, therefore, that it is infectious in a very high degree, and that it may be imparted by a contagium, which, like other contagia, is specific, multiplies indefinitely in the body into which it has gained access, and is thence evolved in marvellous abundance. Under these circumstances it seems most philosophical, at all events most consonant with the present state of our knowledge, to reject the vague theories first adverted to, and to assume that the contagious influence, which certainly causes it to spread in large numbers of cases from man to man, affords the true explanation of its epidemic diffusion. The virus is doubtless given off with the breath. The disease has never been imparted by inoculation; its attacks are in no degree determined by age or sex; and it is quite uncertain whether, or to what degree, one attack is protective for the future. It has been held by some that epidemics of influenza have a tendency to precede, or to follow, or to be associated with, other epidemic diseases, such as cholera. This relation is doubtless accidental.

Symptoms and progress.—The duration of the latent period of influenza has not been accurately ascertained. According to Dr. Squire it is very short, namely, three or four days, or at the outside a week. Its invasion is for the most part sudden, and marked by elevation of temperature; chills, especially along the spine, sometimes amounting to rigors, and alternating with flushes of heat; pain, uneasiness, or a sensation of burning in the back and limbs; and sometimes vomiting. With these phenomena are associated, occasionally from the beginning, but more commonly after the lapse of some hours, severe catarrhal symptoms, indicated by dryness, redness, and swelling of the mucous membrane of the nose, sneezing, and, in consequence of involvement of the frontal sinuses, intense frontal headache; affection of the conjunctivæ and pain in the eyeballs; inflammation of the fauces, larynx, trachæa, and bronchial tubes to their smallest ramifications, with soreness of throat, hoarseness, constant hacking, often croupy, cough, rapidity and difficulty of breathing, and a sense of tightness or constriction of the chest. At this time the skin is generally dry, the tongue covered with a moist fur, the appetite lost, the pulse quickened and moderately full, the bowels confined, and the urine febrile; but,

above all, there is extreme prostration, with muscular weakness, depression of spirits, and præcordial oppression. In the subsequent progress of the disease, general prostration, and inflammation of the bronchial tubes, constitute its most striking features. The heat of skin now probably subsides somewhat; but the patient is still apt to have alternate chills and flushes; the fever assumes a remittent character; perspirations, which are sometimes very copious, break out; not unfrequently sudamina appear, and occasionally an herpetic eruption about the lips; the mucous membrane of the nose and respiratory passages begins to secrete a more or less abundant, thin, colourless mucus, which before long assumes a muco-purulent character; the soreness of the throat and hoarseness probably continue; the difficulty of breathing and the cough increase; and on auscultation the breath-sounds are found to be feeble, or masked by sibilant and sonorous rhonchi and sub-crepitation; the face gets congested or livid; the pulse increases in rapidity, and loses in fulness and strength; the tongue becomes more thickly coated, except perhaps at the tip and edges, and sometimes dry and brown; the sickness possibly continues, and diarrhœa may come on; debility grows extreme, and muscular tremors and subsultus may appear; the intelligence becomes markedly dull and impaired, and delirium is apt to supervene. Epistaxis is of common occurrence, and otitis and jaundice are neither of them unfrequent.

In mild cases, the disease is at its height on the second or third day, and then declines gradually; but in more severe cases—cases in which there is much pulmonary affection—convalescence does not commence until as late as the tenth or twelfth day. The patient is always much reduced in strength at this time; and convalescence is protracted in consequence, partly of persistent debility, partly of the continuance of catarrhal affections, or of a proclivity to catch cold.

The most important of the complications of influenza are those arising out of the characteristic lesions of the air-passages:—namely, laryngeal inflammation, bronchitis (especially bronchitis of the smaller tubes), and lobular and lobar pneumonia, often associated with pleurisy. These affections creep on insidiously during the progress of the case, and reveal themselves only by aggravation of the ordinary symptoms, or by the blending of their proper symptoms with those due to the influenza itself. Gastro-intestinal complications also are described, and occur; but there is no doubt that the accidental concurrence (which is so common) of influenza with other diseases explains a large proportion of the cases in which it is found associated with these gastro-intestinal, and other less frequent, complications.

Single cases of influenza may readily be confounded with severe catarrhal affections of the nose, throat, and bronchial tubes. But the high fever, extreme prostration, and short duration of the graver symptoms, are, all, important characteristics pointing to the specific

nature of the disease. If to these peculiarities be added the fact of epidemic prevalence mistake is no longer possible.

The percentage of deaths from influenza is very small, and indeed the uncomplicated disease is rarely fatal. Still, it attacks so large a proportion of a population (in some cases between a quarter and a half of the total number), that that small percentage does very largely augment the mortuary rate. Indeed, the prevalence of influenza has been found to swell the death-rate much more than the prevalence of cholera. The disease is chiefly fatal among the old, and such as are already suffering from pulmonary or cardiac affections.

Morbid anatomy.—There is nothing distinctive in the morbid anatomy of influenza. Patients die chiefly of pulmonary mischief; and the evidences of this may be detected in the form of inflammation of the bronchial membrane, secretion into the tubes, emphysema or collapse of tissue, or both, or pneumonia, combined or not with pleurisy.

Treatment.—In treating influenza, it is important to adopt the hygienic measures which are generally useful in the treatment of infectious febrile affections. Medicinal treatment is not generally very efficacious. Small doses of nitre, alone, or combined with a few drops of laudanum, have been highly recommended. But probably nothing is better than a few drops of ipecacuanha wine combined with a little laudanum, or ammonia associated with solution of acetate of ammonia, administered every two or three hours. If the bowels are confined, they may be moved either by mild aperients or by enemata. The inhalation of steam may relieve the laryngeal and bronchial affection, as also may the diffusion of moisture through the atmosphere of the room. The removal of blood, even by leeches, is rarely admissible; still, in cases in which the congestion of the lungs is extreme, and death by asphyxia impending, they may be justifiably employed. Blisters again are of doubtful efficacy. Flannel or cotton wool, bran-poultices or hot fomentations to the chest, on the other hand, are often beneficial, as also are mustard plaisters. But little food will probably be taken, or needed, during the earlier days of the disease; and such as is swallowed should consist mainly or exclusively of milk, and the various farinacea, suspended or dissolved in milk or water. Thirst may be relieved by these means, or by the administration of water, tea, lemonade, soda-water, or other such drinks. Owing to the remarkable prostration which generally is present, stimulants are for the most part soon required. The nature of the stimulants to be employed must depend on circumstances. When the patient begins to amend, tonics are indicated, and the diet must be gradually modified, until it combines the ordinary proportions of solid and fluid, and of animal and vegetable, matters which constitute the diet of healthy persons. The presence of complications will necessarily, in many cases, make some modification of treatment desirable. It need only be said, however, in reference to this point, that, as in the uncomplicated disease, so here, depletory measures generally are attended with risk, and very rarely called for.

III. HOOPING COUGH. (*Pertussis.*)

Definition.—An infectious disorder, for the most part of long duration, characterised by inflammation of the respiratory tract and a peculiar paroxysmal cough.

Causation.—Hooping cough is met with both sporadically and in an epidemic form, mainly attacking children, but not altogether sparing adults or persons of advanced age. It is said to be more common in spring and autumn than in other seasons; it is probably not more common at these times, but attended then with a specially high mortality. Neither climate, nor other hygienic conditions, have any notable influence in promoting its spread; but epidemics of it are frequently associated with epidemics of scarlet fever or measles; and it is held by many that there is some kind of mysterious relation or attraction between them. It is contagious in a very high degree, especially during the earlier period of the disease, and before the whoop is established. Its contagium is given off with the breath, and conveyed mainly by the atmosphere; but it is readily carried by clothes, and preserved in fomites. One attack confers almost complete immunity against subsequent attacks.

Symptoms and progress.—As in all similar diseases, a period of latency intervenes between the inception of the virus and the occurrence of symptoms. The duration of this period has not been accurately ascertained: it was exactly a fortnight in some cases which we had the opportunity of investigating in reference to this point.

The invasion of hooping cough closely resembles that of an ordinary catarrh, and is often undistinguishable from it. There is more or less fever, with irritability or inflammation of the mucous membrane of the respiratory tract, and frequent cough, attended with much tickling in the throat and some expectoration of mucus. Some sonorous and sibilant rhonchus may be detected on listening to the chest, but in other respects the respiratory sounds are healthy. There may be injection of the conjunctivæ, photophobia, and nasal catarrh with sneezing. The main points in which the disease differs thus early from ordinary catarrh are—that the fever is commonly higher; that the cough is much more troublesome, sometimes occurring incessantly night and day, several times in the minute; and that these symptoms are all much more persistent, often lasting for a week or fortnight without undergoing any change.

About the end of this time the symptoms become modified; the fever abates, and probably soon disappears, and gradually the irritative cough of the period of invasion subsides, to be replaced more or less completely by the peculiar paroxysmal cough which characterises the

disease. Single paroxysms of this cough may be almost exactly simulated, especially in children, by the effects of the application of pepper or other irritants, to the laryngeal mucous membrane. But in its best-developed form, and by its recurrence, it is quite pathognomonic. The paroxysm is preceded by tickling in the throat, and perhaps pain beneath the sternum; and at the same time a little rhonchus is probably audible on applying the ear to the chest. The child seems to know what is impending, becomes quiet and anxious, and for a short time seems to struggle against it. If lying down, it rises to the sitting or standing posture, and when up, clutches any firm object which is near, or rushes to its nurse or mother. The actual attack usually begins with a deep inspiration. This is at once followed by a rapid succession of short coughs, with no intervening inspirations, which, gradually becoming feebler and feebler, are continued until the cavity of the chest is contracted to the utmost, the veins of the head and neck turgid, the face congested and livid, the eyes watery and starting from their sockets, the whole surface bathed in sweat, and asphyxia seems imminent. Then succeeds a long, whistling, crowing, or whooping inspiration, which is prolonged until the chest is once more distended with air. But the patient is not yet relieved; for the cough immediately recurs, and may be repeated two or three times in continuous succession until the child is utterly exhausted. During the paroxysm, which often lasts for two or three minutes, and more especially at its close, a considerable quantity of viscid transparent mucus is discharged, and very often the contents of the stomach are vomited. In the attack the child may faint or become insensible; the urine and even the fæces may be voided; punctiform extravasations of blood may occur beneath the conjunctivæ, and in the skin of the eyelids and other parts of the face; and there may be hemorrhage from the nose, and even from the air-passages and ears. The membranæ tympani have occasionally been ruptured. The attack does not invariably begin in the manner above described; for occasionally the paroxysmal cough precedes the long-drawn noisy inspiration; and occasionally complete spasmodic closure of the glottis, followed perhaps by insensibility, replaces it altogether. After the paroxysm is over, the child remains more or less exhausted for a time; but for the most part soon resumes his amusements, and appears to have little or nothing the matter with him. The paroxysms recur at irregular intervals, and vary in number from twenty to two hundred (according to the severity of the case) in the course of the day and night, but are almost always more numerous, as well as more severe, at night time. In the interparoxysmal period, auscultation of the chest reveals only slight indications of catarrh; but when the patient is making the crowing inspiration no breath sounds whatever are audible within the chest.

After the above symptoms have lasted with little change for several weeks (usually from three or four to eight or ten), the period of con-

valescence commences. This is of very various duration, and is especially apt to be prolonged if the weather be inclement, if the patient be neglected, or if complications have supervened. During its continuance, the attacks of cough gradually decrease in number and severity, and lose their paroxysmal character; the expectoration becomes thicker and opaque, and then ceases; and the patient more or less rapidly regains health and strength.

Attacks of hooping cough vary very much in their severity and duration; and just as scarlet fever, or measles, may occur without the development of its characteristic rash, so hooping cough may pass through all its stages and yet its cough never be attended with the characteristic whoop. This is especially the case in attacks of exceptional mildness, or when it affects the adult. Trousseau records a case in which an attack of hooping cough lasted three days only. Its entire duration may certainly be as short as a week or two; but much more frequently ranges between six and twelve weeks. Occasionally the disease does not wholly disappear for six, or even twelve, months. If, in the fully developed disease, the paroxysms of cough do not exceed twenty in the four-and-twenty hours, the case may be regarded as a mild one. If they exceed forty or fifty, the case is certainly severe; and the child is probably ill, and feverish, and has signs of pulmonary congestion or bronchitis in the intervals between them. If they are still more numerous, the danger of complications, and to life, is serious. Hooping cough (although one of the most common causes of death in children) is rarely fatal in the absence of complications. These are apt to come on in the second period of the disease, and especially in cases of great severity. They are mainly vomiting, bronchial inflammation, pulmonary collapse, lobular pneumonia, and emphysema, together with epileptiform convulsions and other forms of head-mischief. Vomiting chiefly attends the paroxysms of cough, and if these be frequent, innutrition, emaciation, and debility will necessarily result. The pulmonary complications reveal themselves by difficulty of breathing, lividity of face, crepitation and sibilant rhonchus (without any necessary dulness on percussion of the chest), increased frequency of pulse, and rapid impairment of strength. The emphysema of the lungs, which is the result of laceration of the air-cells, is in children often interlobular, and occasionally spreads through the root of the lung to the connective tissue of the neck, face, and chest. Convulsions occur, chiefly in infants who are teething, and may be either ordinary attacks of eclampsia, or attacks resembling those of laryngismus stridulus in which respiration is arrested by spasmodic closure of the glottis—insensibility supervening, attended with convulsive movements of the muscles of the face and eyes. These complications are, no doubt, serious, and the latter especially may be suddenly fatal, yet the great majority of children who experience them recover perfectly. Dr. E. Smith has shown, that

hooping cough is the most fatal of all the diseases of children under one year of age, that sixty-eight per cent. of all the deaths from it occur under two years of age, and only six per cent. above the age of five years.

Morbid anatomy.—The lesions observed after death from hooping cough are always those of its complications:—namely, congestion of the mucous membrane of the larynx and other air-passages with secretion into the bronchial tubes, collapse of lung-tissue in patches, lobular pneumonia, emphysema, and in children interlobular emphysema. Post-mortem examination, indeed, throws no light whatever on the nature of the disease. Congestions of the medulla oblongata, and of the pneumogastric nerves, which have been described as occurring in hooping cough, are probably purely accidental conditions, if not the result of mere post-mortem changes. So again, enlargement of the bronchial glands, which has been frequently observed, has no necessary connection with it. It has been much discussed, whether the disease is essentially nervous, or a mere inflammatory condition of the respiratory mucous membrane. It seems probable, however, that it is not exactly either one or the other; but that, like other infectious fevers, it is the result of a virus, which affects more or less the whole system, but has a special tendency to involve the respiratory mucous membrane, producing in it a slight but specific inflammatory change, to the effect of which on the peripheral ends of the pneumogastric nerves the cough, with its peculiar characteristics, is due. This view is confirmed by the fact that it is evidently from the implicated mucous surface, that the contagium of the disease is chiefly, if not exclusively, emitted.

Treatment.—As is the case with all diseases of uncertain duration and of intractability, many specifics have been vaunted for the successful treatment of hooping cough. Among the more important of these are hydrocyanic acid and belladonna. With respect to the latter remedy, Trousseau strongly urges that it should be given in one dose daily—and *that* in the morning on an empty stomach; and that, if an increase be necessary, it should be by augmentation of the morning's dose. For infants under four he recommends, to begin with, a pill made with $\frac{1}{10}$ gr. of the extract and $\frac{1}{10}$ gr. of the powdered leaves, or $\frac{1}{200}$ gr. of the neutral sulphate of atropia. If hydrocyanic acid be preferred, from one to two minims of the dilute preparation may (according to Dr. Roe) be given to young children every three or four hours. Strychnia, hyoscyamus, conium, arsenic, iron, bromide of potassium, and bromide of ammonium, have also been strongly recommended, as also have alum, tannin, and the mineral acids. But it is almost certain that no drug has any direct influence over the course of disease; and that hence our efforts must be directed to the relief of distressing symptoms, to the prevention of complications, and to the maintenance of the patient's strength. To these ends it is important that he be kept to his room, which, though well ventilated, should be maintained

of uniform temperature; that, if not confined to bed, he be clothed in flannel; and, generally, that he be not exposed to draughts, or conditions liable to cause pulmonary inflammations. For medicine, there is probably nothing better than a combination of a few drops of ipecacuanha wine with a minute proportion of laudanum or belladonna, to be administered every two, three, or four hours. Counter-irritants are sometimes useful; and the application of a strong solution of nitrate of silver to the larynx has been much recommended, especially by Bouchut and Eben Watson. The patient's diet must be regulated according to circumstances; but generally it should be plain, wholesome, and nutritious. In the period of convalescence, tonics, and change of air, or, failing this, daily exercise in the open air are advisable. When complications arise they must of course be treated specially. But they need no treatment distinct from that of the same affections occurring under other circumstances.

IV. MUMPS. (*Parotitis.*)

Definition.—A contagious fever, of which the chief characteristic phenomenon is inflammation of the salivary glands.

Causation.—Mumps, like scarlet fever, measles, and hooping cough, is a malady which is generally present among us, in a greater or less degree, and every now and then assumes an epidemic character. Like them, moreover, it is extremely infectious, infects, as a rule, but once in a lifetime, and may be regarded as mainly a disease of childhood. It is not, however, confined to childhood; and unprotected adults, and even persons of advanced age, may suffer from it. It is probably not influenced by sex; and there is no reason to believe that its prevalence depends, in any degree, on season, weather, or climate. The virus of mumps seems to be contained principally, if not solely, in the breath.

Symptoms and progress.—The incubative period of mumps doubtless varies; many cases, however, have been recorded in which it seems to have been fourteen days; and this may be taken as probably its average duration. The invasion of the disease is sometimes indicated by febrile symptoms and headache, on which, after a few hours or a day or two, parotid inflammation supervenes; but, in many cases, the affection of the parotid gland precedes the febrile phenomena, or accompanies them from the first. The patient usually complains of aching and tenderness behind one of the ascending rami of the lower jaw; and, in a short time, a little fulness is perceived there, completely obliterating the groove normally existing in that situation. But occasionally the inflammation begins in that part of the parotid which lies upon the masseter muscle. The aching, tenderness, and swelling gradually increase for three or four days, until

the whole of the parotid region is occupied by a dense elastic tumour, which extends forwards over the masseter muscle, and downwards below the angle of the jaw, and over which the skin may assume a rosy hue. Sometimes, the inflammation remains limited to one parotid gland; but more frequently, it involves both parotids, and both submaxillary glands as well, attacking them successively at short intervals, so that all become implicated in the course of two or three days. The inflammation spreads also to surrounding parts, and especially to the fauces and tonsils. When the affection embraces all the glands, and is fully developed, the swelling (which then involves the parotidean and inferior maxillary regions of both sides) marvellously alters the character of the face, giving to its sides great fulness and breadth, and adding beneath a large double chin. The glandular affection generally reaches its full development in from three to six days, and remains stationary for a day or two longer. During the whole of this period the swollen parts are firm and tense, very tender on pressure, and attended with much aching, which becomes exceedingly severe when the jaw is moved, and even when the act of deglutition is performed. Hence the patient cannot masticate, and has much difficulty in swallowing, and the saliva tends to accumulate in his mouth. The febrile symptoms moreover continue—the temperature sometimes attaining a height of 103° or 104°; and there is more or less thirst and anorexia. The character of the saliva, and its quantity, are not usually altered, at all events not altered materially. After a time, which varies according to the severity of the case, and rarely exceeds a week, the swelling of the glands begins to subside, and therewith all the general symptoms. The whole duration of the illness may be a week, but more frequently extends to ten or twelve days or a fortnight; but even at the end of that time, the shrunken submaxillary glands may often still be felt of almost stony hardness. Occasionally the skin over the swollen regions desquamates.

It sometimes happens in the course of mumps (generally in the period of its decline, and occasionally after it has apparently disappeared), that in the male one or both of the testicles get enlarged and painful, and in the female inflammatory swelling of the mammæ or labia comes on. These complications supervene generally without warning, but at times are preceded by apparently unaccountable symptoms of the most alarming kind—sometimes severe collapse, sometimes high fever with delirium. They subside in the course of a few days. Atrophy of the testicle occasionally follows.

Mumps is a disease of little gravity, and rarely, if ever, terminates in death. But it is apt to leave behind it a good deal of feebleness of health. It is most likely to be confounded with non-specific inflammation of the parotid, and inflammatory enlargement of the cervical lymphatic glands; but, under any circumstances, the confusion can only be temporary.

Morbid anatomy.—So little opportunity is afforded of investigating the morbid anatomy of mumps that little can be said positively on the subject. The salivary-gland inflammation probably differs anatomically in no respect from that arising from other causes, but it never proceeds to suppuration. There is doubtless considerable infiltration of the connective tissue of the glands; and indeed the infiltration extends beyond the limits of these organs, involving more or less of the subcutaneous connective tissue on the one hand, and of that of the fauces on the other.

Treatment.—Persons suffering from mumps should be kept out of draughts, and, if not confined to the bed or sofa, at least debarred from making active exertion. The swollen parts may be relieved by fomentations, or the application of flannel or cotton-wool. The bowels may be kept slightly open. The patient should be fed, during the ingravescence of the disease, on milk, bread and milk, eggs, and other like foods, which need no mastication. When alarming symptoms show themselves, ammonia and other stimulants are indicated.

V. MEASLES. (*Rubeola. Morbilli.*)

Definition.—A contagious exanthem, characterised by the presence of catarrh of the respiratory mucous membrane, and a peculiar eruption, coming out on the fourth day. The disease usually lasts between one and two weeks.

Causation.—Measles is one of the most virulently contagious of diseases; and, although its virus can probably not be so long preserved in an active form by fomites, or in other ways, as the contagia of scarlet fever and small-pox, the presence of a case of measles amongst a number of unprotected persons will, as a rule, induce a more certain and widespread outbreak of disease than either of the other exanthems would do under similar circumstances. This peculiarity is due, in some measure, to the fact, that its contagiousness is fully developed at a very early stage—being at its height on the second, if not the first, day of invasion, and consequently before the specific nature of the attack is revealed. Hence the great difficulty, if not impossibility, of effectually preventing its spread in households and schools. Measles is generally present in a sporadic form, but at irregular intervals assumes an epidemic character, spreading rapidly amongst those who have not yet suffered from it, and subsiding when its pabulum gets exhausted. It is mainly a disease of childhood; not, however, so much because adults are naturally indisposed to take it, as because, from its constant presence amongst us and its extreme contagiousness, almost all persons have it early in life, and are thus protected from subsequent attacks. In exceptional cases,

the same individual takes it a second and even a third time; and occasionally the second attack follows so quickly on the first that it constitutes a relapse. This proclivity to repeated seizures occasionally runs in families. In the great majority of cases, however, one attack is permanently protective.

Symptoms and progress.—The latent period of measles varies like that of all other similar diseases; its extreme limits are probably seven and twenty-one days. When the disease has been given by inoculation with the nasal mucus, the first symptoms are said to have manifested themselves on the seventh or eighth day. But when it is caught, in the usual way, by inhalation of the virus, the incubative period is generally from twelve to fourteen days.

During this time the patient, with rare exceptions, is apparently in good health; but occasionally he suffers from lassitude, debility, and slight febrile disturbance. The invasion of the disease is marked by catarrhal symptoms, in association with slight fever. Chills or slight rigors occur, with elevation of temperature and acceleration of pulse; and, at the same time, the mucous membrane of the nose gets injected and irritable, and secretes a thin mucus, and there is frequent sneezing and sometimes epistaxis. The catarrhal affection speedily extends—to the frontal sinuses, causing frontal headache; to the eyes, causing congestion of the conjunctivæ, watering, and intolerance of light; to the fauces and mouth, inducing patchy redness; and to the larynx, trachea, and bronchial tubes, causing soreness, hoarseness, and a hacking cough. Occasionally, in children, the disease is ushered in with an epileptiform convulsion, or several such convulsions; and, on the other hand, not unfrequently the initiatory symptoms are so slight as to escape observation. During the period of invasion, the skin is mostly dry, though sweating may come on from time to time, especially after the rigors; the tongue remains natural or becomes somewhat furred; there is loss of appetite, sometimes sickness and thirst, swimming in the head, and occasionally on the third day some remission of symptoms.

On the fourth day (inclusive) after invasion—sometimes a little earlier, sometimes later—the catarrhal symptoms and fever become aggravated, the temperature rises, the pulse quickens, the patient gets dull and perhaps a little confused, diarrhœa sometimes comes on, and the characteristic eruption begins to appear. This first shows itself on the forehead and temples, near their junction with the hairy scalp, on the cheeks, chin, and back of the neck, whence it gradually diffuses itself over the general surface from above downwards, invading first the chest and arms, then the abdomen and legs. Hands and feet are both affected. It usually becomes most developed on the back of the trunk, and probably least on the generative organs and neighbouring portions of the abdomen. The rash generally attains its height in a couple of days (on the sixth day of the disease), sometimes in three or four, and then declines in the order of its appearance. Its subsidence

is followed, in ten days or a fortnight, by a very fine scurfy desquamation, which is chiefly observable about the forehead and cheeks. The severity of the symptoms continues to increase so long as the rash itself increases; and, with the height of the eruption, the temperature attains its highest point, which rarely exceeds 103° or 104°. When, however, the eruption begins to fade (namely on the sixth day, or it may be a little earlier or later) the temperature almost suddenly falls several degrees, the severe symptoms subside, and convalescence commences. The temperature in some cases at once sinks to the normal, but more frequently it descends to 101° or 100°, at which elevation it remains for a day or two, and then reaches the normal limit, or even sinks below it.

The catarrh of measles is very characteristic and important. It usually commences in the nose, and extends as has been already described. In favourable cases, it involves simply the discomforts of ordinary catarrh. But not unfrequently it assumes a more serious character:—sometimes it induces inflammation of the eyes, which may terminate in chronic or in purulent ophthalmia, and even in their destruction; not unfrequently it reaches the tympanum, through the Eustachian tube, causing more or less intense ear-ache and deafness, upon which suppuration of the middle ear or permanent deafness may supervene. Very often croupy symptoms manifest themselves, or acute bronchial catarrh, or capillary bronchitis.

The tongue is sometimes clean, sometimes covered with a whitey-brown fur, but does not usually get dry. Occasionally, however, when typhoid symptoms manifest themselves, it becomes both dry and black, and sordes appear on the teeth and lips. There may generally be seen, early in the disease and before the appearance of the cutaneous eruption, spotty redness of the palate and fauces, of the inner surface of the cheeks and lips, and of the gums. This often gets uniform and intense, especially on the gums and at the back of the mouth, and is sometimes attended later on in the disease with aphthæ or excoriation, and ulceration of the gums. Gangrene of the mouth is met with in rare cases. Sickness is by no means a constant symptom, and seldom lasts beyond the period of invasion. Diarrhœa frequently comes on with the eruption, and is often very troublesome. Sometimes late in the disease it assumes a dysenteric character.

The urine is scanty and somewhat high-coloured, and often deposits a sediment of urates. Albumen is occasionally present in it during the height of the fever.

The eruption on its first appearance has a dusky pink colour; it consists of small slightly-elevated papules, which gradually increase in area until they attain a line or even two lines in diameter. They are darkest at the centre and fade towards the periphery, and are momentarily effaced by pressure. They are at first discrete, although arranged in groups which have a tendency to form irregular crescents or circles. When they have attained their full size, however, neighbouring spots

often run together; and sometimes, where the rash is very thick, an extensive area of nearly uniform redness results. Whilst the eruption is well-marked, there is always more or less subcutaneous infiltration, and the face appears swollen, and the hands and feet feel tight and uncomfortable. The spots fade very quickly; but, for the most part, there remains some pigmentary discoloration, and perhaps, too, some slight tendency in the vessels of the affected spots to dilate under excitement, which collectively render indications of the rash visible long after the actual rash has disappeared. The skin is generally hot and dry. Gangrene of the vulva occasionally occurs in young children.

The presence of frontal headache has already been adverted to. The chief other pains to which patients are liable are those connected with the occurrence of diarrhœa or dysentery, and otitis. If young children seem to be in severe and continuous pain, the latter complication may be suspected. Patients, especially children, are somewhat dull and irritable, and occasionally, during the early period of the eruptive stage, slightly delirious. Marked delirium is unusual, except in severe cases, and cases assuming a typhoid character. In the latter, coma sometimes supervenes, and sometimes convulsions. Convulsions in the eruptive stage are far more serious than those occurring during the period of invasion.

Measles, if unattended with any serious complication, is commonly a mild disorder, convalescence from which commences about the sixth day, and is completed by about the tenth. Sometimes it is so slightly developed, that its presence is only indicated by slight feverishness and fretfulness, and an inconspicuous rash about the cheeks and back of the neck, associated or not with catarrhal symptoms. In such cases the patient may be well within three or four days from the first manifestation of symptoms; and it may be impossible by these alone to recognise his disease. Sometimes the attack of measles is inherently very severe; and such severity of attack occasionally characterises epidemics. In this case, the patient manifests obvious prostration from the beginning; the pulse is rapid and feeble; the eruption is scanty and of a dusky hue, sometimes almost black, or petechial; the lungs get congested; typhoid symptoms, characterised by black tongue, tremulousness, and delirium, soon come on; and the patient dies collapsed, perhaps comatose, at an early period. When the crisis is delayed beyond the sixth or eighth day, the cause of the delay is generally the supervention, or aggravation, of one of the ordinary complications of the disease, especially laryngitis, bronchitis, lobular pneumonia, or pneumonia. These in fact constitute the main causes of the unfavourable results of measles. Death, however, may ensue from any of the other complications which have been enumerated—diarrhœa, dysentery, epistaxis, gangrene of the mouth or other parts, or the results of otorrhœa. Pulmonary phthisis appears to be a not unfrequent sequela of measles, following upon the more common pulmonary or bronchial inflammation.

Diarrhœa of a very persistent and troublesome character often comes on after measles in children.

Morbid anatomy.—Internal organs manifest no post-mortem appearance peculiar to measles. If the patient die early, or of the malignant form of the disease, the blood is dark-coloured and coagulates imperfectly, and there may be hypostatic congestion of the lungs and congestion of other organs. Later on, we necessarily detect the lesions which have been instrumental in causing death—lesions chiefly of the air-passages and lungs, or bowels.

Treatment.—The patient, for the sake partly of counteracting spread, partly of preventing aggravation of the various mucous inflammations by exposure to cold, should be confined to his room, and, if possible, kept in bed until febrile symptoms have entirely subsided. His room should be airy and well-ventilated, but of an agreeable temperature; and he should be carefully protected from draughts or chills. It is not generally necessary that medicines should be given; but, partly to promote the excretions, and partly to relieve the irritation of the respiratory mucous surface, a mixture, containing a small quantity of ammonia with the acetate of ammonia, to which may be added ipecacuanha wine and minute doses of laudanum (very minute in cases of young children), may be frequently administered; for the soreness of the throat, a little black currant jelly may be used, and the patient may gargle with warm milk. In consequence of the tendency to dysenteric diarrhœa, purgatives should be avoided, or employed with great caution. The diet should be mainly bread and milk, beef-tea, and other such fluid, bland, nutritious articles of diet. When convalescence is in progress, vegetable tonics are useful, and a substantial diet must be gradually adopted. The various complications of the disease, and its sequelæ, will require each its appropriate treatment, which need not differ materially from that of the same affection occurring independently; only it is important to recollect that depletory measures are in this case specially injurious. When the eruption is dusky, or comes out imperfectly, and the patient at the same time appears to be very ill, a warm bath is often of great service. It may also prove beneficial when, late in the disease, convulsions come on. When the patient shows signs of exhaustion, and especially therefore in the malignant form of the disease, and when typhoid symptoms are present, stimulants are imperative. In most cases of measles they are quite unnecessary.

VI. EPIDEMIC ROSEOLA. (*Rötheln. Rubeola.*)

Definition.—A contagious disorder, having a close resemblance to measles, with which it is often confounded.

Causation.—This disease is said to occur chiefly in hot seasons, and to affect children much more readily than adults; it is doubtful, however, whether season or age exerts any special influence over it. It certainly spreads by contagion, and doubtless, therefore, depends on a specific virus. Its contagiousness is apparently much less active than that of measles.

Symptoms and progress.—The incubative period of epidemic roseola is probably about a week. Its invasion, in a considerable number of cases, is coincident with the appearance of the rash. In some cases, however, the eruptive period is preceded by a day or two of poorliness; the patient has a headache or is feverish, and may even have rigors; or he complains of cold or catarrh, and, according to Trousseau, may, if a child, have diarrhœa and convulsions. The latter occurrences, however, must be very rare; and, indeed, among the chief distinctions between this affection and measles are the slightness, the want of character, and the uncertain but always short duration, of its stage of premonitory fever.

The rash generally appears first on the sides of the nose and adjoining parts of the cheeks, the lower region of the forehead, and the lateral aspects of the inferior maxilla; but it shows itself almost, if not quite, as early on the forearms and hands, and corresponding parts of the lower extremities, and then rapidly diffuses itself over the whole cutaneous surface It usually attains its height on the second day, and, in the course of the next two, three, or four days, rapidly disappears. The rash has much resemblance, in tint and general appearance, to that of measles; but it does not assume the crescentic grouping which is characteristic of that affection. The spots, which fade on pressure, are of a dusky red or purplish hue, of irregular shape and often clustered—sometimes running together over considerable tracts—and vary in size from mere points up to a line or more in diameter. They are, for the most part, scarcely elevated above the general level of the skin; but occasionally, and more especially on the face, form considerable papular or tabular elevations. The rash is generally most abundant on the face, where it is often confluent, and on the forearms and legs (especially about the ankles and wrists), where also there is often a similar tendency to confluence. It is less thickly developed elsewhere; but no part is free; and generally, abundant discrete spots may be observed on both the palmar and the dorsal aspects of the hands and fingers, and on the corresponding parts of the feet and toes. It is attended with considerable itching, and is often followed by branny desquamation. The patient does not generally complain much, or at all, of soreness of the eyes, or lachrymation; nevertheless there is nearly always marked congestion of the conjunctivæ. There is frequently a little sore throat; and sometimes red puncta, or more or less diffused redness, may be recognised on the soft palate and fauces. There is not, as a rule, defluxion from the nose or

sneezing, or, if these symptoms are present at all, they are by no means prominent. There is often a little cough. During the first day or two after the appearance of the rash, the patient may be somewhat feverish, with slightly elevated temperature, headache or swimming in the head, and other slight symptoms referrible to fever; but not unfrequently he feels and expresses himself as being perfectly well. The affection is unattended with complications, subsides ordinarily within a week, and has no sequelæ.

Epidemic roseola has been described as a hybrid of scarlet fever and measles; and some have regarded it literally as such. There is little likeness, however, between it and scarlet fever. Its resemblance to measles, on the other hand, is very close. It differs from measles, chiefly in the slight development of its initiatory fever, in the almost complete absence of coryza, in the arrangement of its eruption, and in the general mildness of its symptoms; but these differences are chiefly of degree, and only such as might be observed between very slight and severe cases of true measles. The main distinctions are these—that roseola and measles are mutually unprotective; that roseola is of frequent occurrence in those who have had measles only a short time previously; and that when it breaks out in a family or school of children, of whom some have had measles and some not, it attacks them indiscriminately and with equal mildness, and never gets developed into true measles.

No special *treatment* is needed.

VII. SCARLET FEVER. (*Scarlatina. Febris Rubra.*)

Definition.—A contagious malady, characterised mainly by a general punctiform scarlet eruption, usually appearing on the second day, and by inflammation of the fauces, tonsils and kidneys.

Causation and history.—Down to the sixteenth or seventeenth century scarlet fever was confounded with measles. Yet they are two perfectly distinct diseases, and are now fully recognised as distinct. Whatever its original source, or however it may formerly have been limited in area, it is now general throughout the world, occurring in most parts sporadically, but frequently breaking out into epidemics of greater or less severity. Its prevalence seems independent of season or climate, but, as with other infectious epidemic disorders, is largely promoted by overcrowding and poverty. Children suffer from it in much larger proportion than adults; not, however, because there is any special proclivity to it in childhood; but because, from its frequent prevalence and highly infectious nature, the great majority of children are exposed to its influence during the first few years of life, contract

it, and thus acquire protection. Scarlet fever rarely occurs a second time; yet second and even third attacks have been noticed. It is a common observation, however, that protected attendants on scarlatinal patients frequently suffer from sore throat during the period of their attendance; and the question naturally arises, whether such attacks should not be regarded as abortive attacks of scarlet fever. They probably are so. The contagion of scarlet fever is very powerful and diffusive. It may be carried considerable distances by the atmosphere—certainly through the whole dimensions of a large ward; and it clings to clothes and other fomites with great tenacity, and may thus lie latent yet capable of action for an indefinite period. Scarlet fever occurs only as the result of contagion, usually conveyed by the means which have been already indicated. It seems that it may also be transmitted by direct inoculation. For there is reason to believe that it can be imparted by inserting the fluid of the scarlatinal vesicles beneath the cuticle of persons who have not yet had it; and it is certain that women, at the time of parturition, are specially liable to take it, receiving it then, in some cases, apparently direct from the fingers of the accoucheur. The time at which a scarlatinal patient begins to be infectious is uncertain. We know, however, that his infectiousness is not very well marked during the first two or three days. It probably increases with the development of the rash and sore throat, and pretty certainly does not cease until desquamation has been completed.

Symptoms and progress.—The incubation of scarlet fever is shorter than that of most diseases of the same class. It usually varies between six and eight days, but is occasionally longer, and very often less. Many cases, indeed, of undoubted authenticity have been recorded, in which it certainly did not exceed twenty-four hours. Especially in puerperal women, and probably also in persons suffering from large wounds, the period of latency seems generally to be of very short duration. Scarlet fever varies, perhaps more than any other like disease, both in the degree of severity of its attacks, in the symptoms which it presents, and (in fatal cases) in the cause and period of death. In a typical case, the invasion is sudden, and usually marked by chills, vomiting, and sore throat; with which are associated, or on which soon supervene, great rise of temperature, general dryness of skin, much acceleration of pulse, languor, drowsiness, frontal headache, giddiness, aching in the limbs, slight coating of tongue, thirst, anorexia, and sometimes diarrhœa. The most characteristic of these symptoms are—the sore throat and vomiting; the remarkable rise in the frequency of the pulse, which may attain 120 in the adult or 160 in the child; and the rapid augmentation of temperature, which may reach very nearly 105° during the first day. The disease is sometimes ushered in with rigors, and not unfrequently there is some delirium or even tendency to coma.

On the second day, the rash makes its appearance, first on the

chest, and simultaneously or very soon afterwards on the forearms, lower part of the abdomen, and upper part of the thighs. It becomes general in the course of four-and-twenty hours, more or less, and attains its full development on the third or fourth day. It consists, in the first instance, of very minute, rosy papules, due for the most part (as those of so-called 'goose's skin') to the conical elevation of the cutis around the points of emergence of the hairs; hence they are closely and pretty uniformly arranged, but discrete and separated from one another by healthy skin. But they soon increase in size and intensity of redness, and presently, blending with one another by their congested margins, give to the surface a uniformly scarlet hue. The papular character, however, of the rash is still, for the most part, distinguishable on close inspection. Not unfrequently the papulæ on the chest and sides of the neck become vesicular; and generally the rash is attended with more or less infiltration and thickening of the cutis. The vivid redness of the skin disappears readily on pressure, as by drawing the point of the nail firmly along the surface; and the line thus formed remains anæmic for a second or two. The scarlatinal rash varies much in its intensity and in its diffusion. It is sometimes very pale and almost imperceptible; and it may be strictly limited to the parts in which it usually first appears. When general, it is most vivid on the neck, chest, abdomen, and inner aspects of the thighs and arms. It is rarely distinct upon the face, which, however, often presents irregular patches of redness. The feet and hands are not unfrequently stiff with it, and its attendant œdema.

While the rash is attaining its full development, the other symptoms are all undergoing aggravation:—The heat rises; the pulse increases in frequency; the respirations grow more rapid; the tongue, which was at first covered (excepting at the tip and edges) with a thickish whitey-brown fur, soon cleans, and towards the end (that is in four or five days from the invasion) becomes morbidly red, with swollen papillæ, and presents the remarkable strawberry-like appearance so characteristic of this disease. At this time too it is apt to get dry. The soreness of the throat increases; and, on inspection, more or less vivid or dusky redness of the pillars of the fauces, soft palate, uvula, and tonsils, is apparent. These parts, moreover, swell; and the tonsils often enlarge as in common quinsy, and present here and there on their surface imbedded, or adherent, spots of inspissated secretion. With the faucial swelling and inflammation are usually associated pain and difficulty in swallowing, fulness and tenderness behind the angles of the jaw, and some enlargement of the neighbouring lymphatic glands. The patient's muscular weakness increases, and his limbs get tremulous; he becomes and looks dull and stupid, or restless, is forgetful and slow to answer; delirium probably increases; vomiting is now not common, but thirst and anorexia continue; and the bowels, though variable, are generally constipated.

From the fourth to the sixth day of the disease, the rash begins to fade; and it disappears, according to its intensity and the date at which it attained its maximum, between the sixth and twelfth day of the disease, or between the fifth and tenth day from the commencement of the rash. It is frequently about this time that, if the case be going on badly, the patient passes into a typhoid condition, or throat complications become serious—the tonsils suppurating, ulcerating or sloughing—or the urine gets albuminous, and anasarca and uræmia supervene. If, however, the case be going on favourably, all the symptoms now gradually subside:—the temperature, with slight daily remissions, ere long becomes normal or even sub-normal; the pulse by degrees sinks to its healthy rate or below it; the soreness and inflammation of the throat subside; the tongue gets clean and moist; thirst abates; appetite returns; and delirium, with other symptoms referrible to the nervous system, vanishes.

With the disappearance of the rash, desquamation commences. It may be observed, indeed, on the chest before the rash has quite left other parts of the surface. It usually begins on the neck and chest; whence it spreads to the rest of the trunk, and then to the limbs, involving lastly the palms of the hands and soles of the feet. Desquamation always takes place in considerable flakes, the size of which is greater according as the epidermis is thicker. Hence, they are small and delicate on the chest and abdomen, large on the limbs; and from the hands and feet the epidermis occasionally separates in the form of a glove. The period of desquamation is of very various duration; it is sometimes completed in one or two days, not unfrequently extends over a week or two, and occasionally is prolonged for several weeks. It is a period of some danger; for it is chiefly then that albuminuria arises, that dropsy and uræmia threaten, and that rheumatism and other serious sequelæ are liable to come on; moreover, there is good reason to believe that the desquamating particles of skin are charged with the contagium of the disease, and are highly infectious.

We will now pass briefly in review some of the more important phenomena of scarlet fever. Acceleration of the pulse, especially in children, is a notable feature of the disease; it probably rises on the first day to between 100 and 120—in children still higher; and it generally continues to increase up to the time of full development of the rash, sometimes attaining a rate of from 120 to 160, or more; after which, if the case go on favourably, it gradually falls. This great acceleration of pulse is not necessarily an indication of danger. Nevertheless, unusual rapidity with marked weakness of pulse, especially when associated with other unfavourable symptoms, is of grave import.

Respiration is always more or less hurried, but there is not necessarily any cough or difficulty of breathing. Sometimes, however, in cases of great intensity (as also in pyæmia and other forms of so-called

'blood poisoning') the respirations become very rapid and shallow, and the inspirations attended with dilatation of the nostrils, and a sniffing or sucking sound—conditions which, unassociated with distinct pulmonary lesion, indicate very great danger. During the latter part of the eruptive stage, or subsequent periods of the disease, inflammation may extend to the larynx and trachea, and produce the usual symptoms of laryngitis; or coryza, bronchitis, or lobular or lobar pneumonia, with their several groups of symptoms, may supervene.

Thirst and loss of appetite are always present in a greater or less degree. Vomiting is for the most part a characteristic feature of the invasion, and few children fail to suffer from it; but it does not usually persist. Diarrhœa is not uncommon at the commencement; after which, the bowels are generally, but by no means necessarily, constipated. The tongue varies in character:—in very mild cases, it is only slightly furred, and soon cleans, without ever displaying the strawberry-like appearance; sometimes, it very early becomes thickly coated, dry, and even black—sordes appearing at the same time on the teeth and lips; but more frequently, as has been pointed out, it is coated at the beginning, and on the fourth or fifth day gets clean and unnaturally red, with prominent and swollen papillæ; after which, it may either gradually acquire the normal characters, or become dry and mahogany-like. The soreness of the throat causes difficulty and pain in swallowing, and a nasal quality of voice. It involves all the parts at the back of the mouth, the fauces, and the upper part of the pharynx, but does not usually include the larynx. The tonsils chiefly suffer; and, as has been pointed out, they generally get enlarged, and present on the surface opaque patches, which have been secreted by the glandular follicles. In mild cases, the soreness may be very slight, and may speedily subside. Very often, however—sometimes at the beginning, more frequently in the second or third week—the tonsils suppurate, ulcerate, or slough; or abscesses and buboes form in their neighbourhood; or a false membrane appears upon the surface and extends to other neighbouring parts.

The urine, during the febrile stage of the disease, is scanty and high-coloured, contains a diminished quantity of chlorides, and not necessarily, according to Dr. Gee, any increase of urea. Subsequently it becomes more abundant and of lower specific gravity. Albuminuria is frequently present, and its presence is a matter of importance. It appears to have no particular connection with the degree of severity of the attack. Indeed, many of the severest cases escape it altogether, and many of the mildest suffer severely. The time of the first appearance of albumen varies. It has been detected on the second or third day of the disease, but commences far more commonly in the course of the second or third week, or during the period of desquamation. Its amount varies, as also does the period during which it persists. The

urine is not unfrequently smoky. Under the microscope are found hyaline and epithelial casts of the renal tubules, and usually also blood-corpuscles, or casts containing altered blood.

The characters of the rash have already been fully described; it must be added that, during the height of the disease, the skin is generally dry and feels pungently hot, and that in 'malignant cases' petechiæ often make their appearance.

The temperature of the body attains a greater height in scarlet fever than in any other disease of the same class; it frequently reaches 104° or 105° when the eruption is fully developed, and occasionally rises to 110° or even 112°. It differs in its course from that of small-pox, in the fact that it rises, instead of falling, when the rash appears; and from that of measles, by subsiding slowly after the rash has reached its acme, instead of undergoing a sudden fall.

The patient complains of soreness of throat, and has some headache and giddiness, with general aching of his limbs; but the pains are not so severe as in many other febrile disorders. In the beginning of the disease he is generally restless and sleepless, and often a little delirious. When the eruption comes out, and during its persistence, he may still be restless and excited, or dull and inclined to coma, or he may have more or less delirium. In grave cases, violent delirium is sometimes one of the earliest symptoms. Occasionally, in children, convulsions come on early in the disease; they are rarer, however, than at the commencement of measles or small-pox, and are far more serious—indeed are generally followed by a fatal result. Coma, delirium, or convulsions not unfrequently usher in death. Tremors of the muscles, subsultus, and picking at the bed-clothes occur in serious cases.

No known disease is more unequal in its attacks than scarlet fever. In individual cases, it often proves one of the mildest and most trivial of ailments, often one of the most terrible and rapidly fatal of plagues. In one household all the members may have it so slightly that they scarcely acknowledge to themselves that they have been ill; and in another not one that is attacked survives. And varieties of this kind characterise epidemics. Thus, in many cases, the disease spreads rapidly through a village or town, or over a large extent of country, and its attacks are so mild that scarcely a death results; while in other cases, the epidemic is characterised by great malignancy and terrible mortality.

The mildest form has been termed '*latent scarlet fever.*' In this the cases are so slightly developed that they would probably not be recognised as scarlet fever at all, were it not for the fact, either that they occur while scarlet fever is prevailing, or that they impart scarlet fever, or that desquamation, or albuminuria with anasarca, or both, supervene. The patient may suffer from slight febrile symptoms only, lasting for a day or two, with which may, or may not, be associated evanescent traces of a rash, or some degree of roughness of the throat. It is a

question, which has already been raised, whether the sore throat, which protected attendants on scarlatinal cases so frequently experience, is not the visible sign of latent scarlatina, or rather perhaps of the disease in a modified form.

The more ordinary forms of scarlet fever are those to which the previous detailed description applies. The symptoms of invasion are well-marked, the rash is abundantly developed, the throat and tongue are typically affected, and the rash disappears between the sixth and the twelfth day of the disease, to be followed by desquamation. But cases of medium severity may present considerable varieties among themselves. Thus in some, while every other characteristic symptom is present, the throat may escape; in some, while the throat suffers severely, the eruption may be imperfectly developed. The former cases are often distinguished by the epithet of '*scarlatina simplex*'; the latter by that of '*scarlatina anginosa*.'

The name '*malignant*' is commonly given to those cases of scarlet fever in which the symptoms are unusually severe, and death tends to come on rapidly. It is somewhat loosely applied, however, and embraces cases of widely different characters. The most terrible of such cases are probably those in which the patient seems to be struck down by the severity of his attack, and dies collapsed during the first three days of the disease—sometimes on the first day, often before the rash has had time to appear or to develop, or before the affection of the throat has become a special cause of complaint. The symptoms of invasion are severe; the vomiting probably is distressing; the chills or rigors are unusually well-marked; the temperature attains an extraordinary elevation; the pulse becomes extremely rapid and weak, the respirations quick, shallow, and suspirious; prostration and muscular debility are extreme—there is tremulousness of the muscles and jactitation; the face is dusky and the expression anxious. The patient is sometimes sensible, almost to the last; sometimes there is from the beginning fierce or muttering delirium, which lapses before death into coma, occasionally preceded by an attack of convulsions. Another variety of malignant scarlet fever is that in which the throat is gravely implicated. The throat-affection may be serious from the first; but more frequently, in a case which presents no very unusual features at the beginning, it undergoes aggravation either at the acme of the fever, or during the subsidence of the rash, or even on its disappearance. The nature of the affection has been already adverted to. There may be abscess of the tonsil, or ulceration or gangrene, with œdema of the surrounding tissues; and supervening thereon, the glands in the neck may inflame and suppurate, and sinuses form. Under these circumstances the patient is apt to fall rapidly into a typhoid condition, and so die; or he may be carried off by œdema of the glottis, perforation of an artery, or pyæmia. Scarlet fever occurring at or just subsequently to parturition is excessively fatal, and constitutes one of the gravest forms of so-called

puerperal fever.' It does not appear, however, to be specially dangerous during pregnancy, or to lead to abortion.

The sequelæ of scarlet fever are numerous and important. It is difficult, however, to make any clear distinction between the complications which form an essential part of the disease, and have already been described, and the phenomena which are simply secondary. It is needless to repeat what has been said about bronchitis, pneumonia, and ulceration of the throat, all of which are apt to complicate the disease in its later stages. We will briefly consider the more important of those sequelæ which have not yet been referred to. First. The conjunctivæ not unfrequently inflame in the course of scarlet fever; and occasionally in the second or third week of the disease the ophthalmia becomes intense and purulent, and sloughing of the corneæ may result. Second. Inflammation sometimes extends along the Eustachian tube to the tympanic cavity, producing ear-ache or otitis, with disease, may be, of the petrous bone, and, possibly, sooner or later, abscess of the brain, pyæmia or some other fatal lesion. Inflammation may extend also to the nose, and produce chronic catarrh of its mucous surface. Third. Inflammation of the pericardium or of the pleuræ (the latter often purulent) is not uncommon. Fourth. During the decline of the fever, or even during the period of convalescence, rheumatism is very apt to supervene. This differs in no respect from ordinary rheumatism, involves successive joints and in many cases the pericardium or the cardiac valves, and adds seriously to the fever and distress of the patient. To scarlatinal rheumatism, as to other varieties of rheumatism, chorea or embolism occasionally succeeds. Fifth. The most important sequelæ of all are, undoubtedly, anasarca and uræmic poisoning. We have pointed out that in a large proportion of cases (and for the most part in the second or third week) the urine becomes albuminous. Now this condition generally passes off without any ill result. But not unfrequently, and more frequently after mild than after severe cases, anasarca and uræmia supervene, which may presently be attended with severe headache, and followed by epileptiform convulsions and death. Under judicious treatment the albuminuria and the dropsy may subside; but sometimes the urine remains permanently albuminous, and the kidneys undergo slow disorganisation. It may be observed that anasarca sometimes survives the disappearance of the albuminuria; and that it is sometimes developed in those who have never had albumen in the urine. Uræmic convulsions generally involve a fatal issue.

Morbid anatomy.—On post-mortem examination of scarlatinal patients most internal organs appear to the naked eye fairly healthy. The liver and kidneys may be somewhat softer than natural, and the blood imperfectly coagulated. Yet, well-formed fibrinous clots are not uncommon in the right ventricle. In so-called 'malignant' cases, there may be collapse and hypostatic congestion of the lungs, and hemorrhage into and at the surface of internal organs. The throat gene-

rally presents distinct traces of inflammation and ulceration. The solitary intestinal glands and Peyer's patches are somewhat enlarged. The only other morbid appearances (and they are sufficiently important) are such as are connected with the sequelæ and complications of the disease. These, however, though common in scarlet fever, are not peculiar to it, and will be considered with the special diseases of the various organs to which they belong, or under other appropriate heads. The microscopic morbid anatomy of scarlet fever has recently been investigated with minute care by Dr. Klein,[1] who shows that even at the earliest stages of the disease there is a marked tendency to inflammatory hyperæmia and proliferation, not only in the skin, mouth, throat, and kidneys, but throughout the alimentary canal, and in the salivary glands, pancreas, liver, lymphatic glands, and spleen. Generally in all these parts there are observed germination of the endothelium of the small blood-vessels, hyaline thickening of the intima, germination of the nuclei in the muscular coat, and accumulation of lymphoid cells in the tissues around; besides which—in the epidermis, swelling and proliferation of the cells of the rete mucosum, with serous effusion and migration of leucocytes between them, and tendency to detachment of the horny-layer; in the various epithelia (including those of the renal tubules) changes resembling those in the skin; and in the interior of lymphatic glands, especially those of the neck, disappearance of the lymphoid cells, and development in their stead of many-nucleated giant cells, which ultimately become fibrous. A more minute description of the changes which take place in the kidneys will be given hereafter.

Treatment.—Whenever scarlet fever breaks out among a number of susceptible persons, the sick should be at once separated from the sound. The patient should be placed in a suitable room, at the top of the house if possible, and if possible should have a floor to himself. All the usual measures should be taken as regards nursing, ventilation, disinfection, cleanliness, and removal of surplus furniture. He should be kept strictly in bed, with only so much covering as is absolutely necessary. His diet should consist of milk, beef-tea, eggs, and other such articles. And for medicine, acetate of ammonia or nitrate or chlorate of potash in solution may be serviceable. Some strongly recommend ammonia in large and frequent doses; and some dilute hydrochloric acid, or the perchloride of iron. Ice is often useful to allay vomiting. To relieve the soreness of the throat, ice, or the inhalation of steam, or warm milk slowly swallowed, or astringent or antiseptic gargles may be employed. The patient is generally benefited also by tepid sponging, or the tepid douche bath. If the bowels are much constipated they should be relieved by laxatives; if there is diarrhœa they should be restrained by opium or other astringents.

[1] See Report of the Medical Officer of the Privy Council. New Series, No. viii. p. 23 *et seq.*

When convalescence is taking place, it is recommended to keep the body well greased in order to prevent the dissemination of the flakes of cuticle. The practice is a good one, and may be associated with the daily use of warm baths. Tonics must now be had recourse to, and the diet should be nutritious and include a fair proportion of solid food. It is during this period that the dangers of rheumatism and of dropsy are greatest. It is important, therefore, that the patient should be kept warm, that he should not be exposed to draughts, that he should keep his room—either confined to bed or encased in flannel—and that the excretory functions should be carefully attended to, until the period of desquamation has come to an end.

In most cases stimulants are not needed; but in malignant cases, and all cases where the muscular debility is great, and there is a tendency to collapse, or to the coming on of typhoid symptoms, they are imperatively demanded.

In the severest cases of the disease, however, all treatment is futile; and in the milder cases, the care of the physician must be directed, not so much to the cure of the disease, as to the relieving of discomfort, and to the obviation by precautionary measures of complications and sequelæ.

If there be nasal catarrh with discharge, it is well to syringe the nostrils with warm water, or water containing chlorate of potash, nitrate of silver, or some antiseptic. If the throat be ulcerated or gangrenous, solution of perchloride of iron or of nitrate of silver, or even the latter in a solid form, or hydrochloric or nitric acid, may, according to circumstances, be applied. Warm fomentations or poultices should be employed externally; and if there be suppuration in the glands or connective tissue behind and below the jaw, a puncture or incision should be made. Otorrhœa, rheumatism, renal dropsy, and uræmic convulsions must be treated as these affections are treated when they arise under other circumstances. And so with regard to other complications. Only, it must not be forgotten that these affections, occurring as complications, bear depletion less and need stimulation more, than do the same affections when they are of spontaneous or idiopathic origin.

VIII. SMALL-POX. (*Variola.*)

Definition.—A specific fever, spreading by contagion, and especially characterised by the appearance on the third day of a papular eruption, which gradually becomes pustular, and attains its full development on or about the eleventh day of the disease. The eruption shows itself also in the mucous membrane of the mouth, fauces, and larynx.

Causation and history.—As with many other of the infectious fevers, the history of small-pox cannot be traced further back than the Christian era. The first recorded epidemics, indeed, seem to have occurred in the sixth century. Since when it has never disappeared from among us, has been carried from Europe and Asia over all parts of the world, and, down to within a recent period, has formed one of the most formidable and fatal of pestilences. The disease was robbed of many of its terrors by the practice of inoculation, introduced first into this country, early in the eighteenth century, by Lady Mary Wortley Montague, who had witnessed the efficacy of the procedure in Constantinople, whither it had been imported from Persia and China. It was yet more marvellously controlled by the application of Jenner's discovery, made at the end of the same century, of the protective influence of vaccination; since the general adoption of which small-pox has become a comparatively rare and unimportant affection. But it still maintains all its old virulence when it attacks those who are not protected by vaccination or by a previous attack of the disease, and all its old epidemic violence when it is introduced among susceptible communities. Small-pox has no special predilection for age or sex; but it is said that dark-skinned races, and especially negroes, suffer more severely from it than the denizens of temperate climates. All persons, indeed, are liable to take it, unless protected in one or other of the ways which have just been adverted to, or, (as rarely happens) by some peculiar constitutional insusceptibility. Instances, however, are, on the whole, not uncommon in which persons have a second and even a third attack—such attacks being for the most part mild; and it is a curious circumstance that those who, in spite of constant exposure, have enjoyed immunity from the disease for many years not unfrequently end by contracting it, and have it in a severe form. Whatever the source of small-pox may originally have been, there is no doubt whatever that it now comes solely by contagion, and that this may be conveyed either through the atmosphere or by fomites, or by direct inoculation with the contents of the variolous pustules. Few diseases, indeed, are more virulently contagious than small-pox; and there is none whose virus remains effective for a longer period.

Symptoms and progress.—The period of latency of the inoculated disease has been distinctly ascertained to be seven or eight days. On the second day a small papule shows itself at the seat of puncture which by the fourth day is converted into an umbilicated vesicle. On the seventh day the vesicle has formed a pustule; and about the same time the lymphatic glands above have become swollen and tender. And on this day, or the eighth, rigors and other symptoms indicative of the invasion of the disease occur. About the tenth or eleventh day the pustule is fully developed, and at the same time the general variolous rash appears. By the fourteenth day the pustule has dried

up into a scab. The period of incubation is always longer when the disease has been acquired in the usual way. It is generally considered then to range between ten and sixteen days. According to Mr. Marson, it is almost invariably twelve days. The facts connected with inoculation prove that the variolous contagium is present, in a concentrated form, in the mature pustules. There can be little doubt, therefore, that small-pox is especially infectious about the period of maturation. But it is probably infectious during the whole period of its duration, from the first signs of invasion up to the separation of the last scab.

The incubative stage of small-pox is, with rare exceptions, unattended with symptoms. But occasionally the patient suffers from languor, peevishness, and other vague feelings of illness. The invasion is more or less sudden, and is indicated by—rise of temperature, chills or rigors, followed by or alternating with heat of skin, and generally (in adults) copious perspiration; severe sickness, with anorexia, thirst, and constipation or (in children) diarrhœa; headache, aching of the limbs, and intense pain in the lumbar region of the spine; drowsiness, and not unfrequently delirium, stupor or coma, and (in children) convulsions. There is sometimes maniacal excitement. The most characteristic of the above symptoms are the vomiting, constipation, and acute lumbar pain; it is important, too, to note the frequency of perspirations, and of convulsions which for the most part are unattended with danger to life. The symptoms of this stage are severe in proportion to the severity of the attack which they usher in. *Cæteris paribus*, therefore, the higher the temperature, the more persistent the vomiting, the acuter the pain in the back, the more pronounced the implication of the brain, the more quickly will the disease assume grave proportions, and the greater will be its intensity and the prospect of a fatal issue. Absence or scantiness of perspiration, and in adults the presence of diarrhœa are also indications of a severe attack.

The above symptoms usually attain their maximum on the third day—the day on which the characteristic rash first manifests itself. In a small proportion of cases, and these are for the most part fatal cases of great malignancy, the eruption appears on the second day; and occasionally it is delayed to the fourth or even later. In modified small-pox, it is not unusual to find the true eruption preceded for a day or two by a roseolous efflorescence, which has some resemblance to the scarlatinal rash. And in cases which threaten to be unusually severe there may be on the second or third day of the disease, 1st, a sub-papular patchy redness on the face and elsewhere, which is almost undistinguishable from the rash of measles, but is in fact the commencement of the small-pox eruption in a papular form; or 2nd, an abundant petechial rash chiefly about the sides of the chest and abdomen and on the loins. The rash usually commences, however, on the third

day, in the form of minute reddish papules, which are first visible on the face, head, neck and wrists, and in the course of the next two days invade successively the upper part of the chest, the arms, the rest of the trunk, and the lower extremities. The spots are hard, solid, hemispherical or acuminated, and feel like shot imbedded in the skin; they gradually enlarge, and in the course of two or three days get vesicular; then, still increasing in area, their contents become opaque and milky, and about the sixth day (eighth day of the disease) distinctly purulent. With their conversion into pustules, there is a marked extension of inflammation; each pock acquires a deep-red areola, and the subjacent tissues swell with inflammatory effusion. The pustules still increase in size, and the surrounding inflammation still augments, up to about the ninth day (eleventh day of the disease). The process of maturation, as it is called, is then completed. The above remarks apply more particularly to the eruption on the face; on the lower part of the trunk, and on the extremities, its several stages occur somewhat later. The eruption of small-pox is always more abundant and close-set on the face and neck than elsewhere; and is generally, even in severe cases, scanty on the lower part of the trunk. When sparse the papules, like those of measles, often appear in crescentic groups; but when they are more thickly clustered this arrangement is not observed. If the primary papules are much crowded, the pustules which result from them tend to coalesce, and thus to form extensive tracts of suppuration, in which the limits between the constituent pustules are scarcely or not at all distinguishable. When the pustules remain distinct from one another on the face, the attack of small-pox is termed '*discrete*'; when they run together in the same situation, it is called '*confluent.*' The pustules of discrete small-pox are always larger than those of the other variety, and the surrounding inflammatory areola is more obvious. The confluent form, however, is always much the more severe, and attended with far greater subcutaneous œdema and ultimate destruction of tissue; the face, and especially the eyelids, are apt to get enormously swollen; and the hands are often so much enlarged and tense that the patient cannot close them. The variolous rash is not limited to the skin, but is generally developed also, more or less abundantly, on the mucous surface of the nose, mouth, fauces, and pharynx, and even on that of the larynx and trachea, and sometimes upon the conjunctivæ. The fully-developed cutaneous pustules are circular in outline, unless altered in form by coalescence or other accidental circumstances, vary from about $\frac{1}{4}$ to $\frac{1}{3}$ inch in diameter, are somewhat flat, and mostly depressed in the centre, or 'umbilicated.' In some cases their contents, even from an early stage, are mixed with blood; and not unfrequently they are associated with petechiæ and vibices.

In all cases of small-pox, there is, on the first appearance of the rash, a sudden diminution of the severe symptoms which characterised

the invasion; the temperature falls, and becomes in some cases nearly normal, the pulse lessens in frequency, the vomiting ceases, the febrile pains and pains in the back subside, delirium and other nervous symptoms disappear, appetite perhaps returns, and the patient seems to be convalescent. At the same time, however, the cutaneous eruption is producing some inconvenience; and he begins to complain of soreness in the mouth and tongue, with ptyalism, and his throat gets painful, his voice hoarse, and a ringing or metallic cough probably comes on—phenomena which are due to the involvement in the rash of the mucous surface of the upper parts of the respiratory and alimentary tracts. The degree in which the symptoms of invasion subside, and the duration of the period of their abeyance, depend on the severity of the attack. In very mild cases, the pocks, at the period at which they usually suppurate, begin to contract and dry up, and there may then be no interruption to the favourable progress of convalescence. In cases of medium severity, the period of apparent convalescence continues up to the sixth or seventh day of the rash (eighth or ninth of the disease) at which time the maturation of the pustules commences. It is then interrupted by a sudden recurrence of febrile symptoms, which last for some three or four days, or until about the completion of maturation. This is the period of 'secondary fever,' and is marked by chills or rigors, increase of temperature (which may even surpass that of the period of invasion), acceleration of pulse, dry furred tongue, and delirium. When the disease is of the confluent kind, the remission of symptoms at the commencement of the eruptive stage is very slight; the temperature may, perhaps, sink a degree, and there may be some slight general amelioration for four and twenty hours, or less; after which, the febrile symptoms and delirium increase with the progress of the eruption, attaining their maximum severity, without any particular change in quality, during the period of maturation. It is in such cases that the swelling of the face, hands, and feet is greatest, that salivation is most profuse, that other symptoms referrible to the mouth and throat are most violent, and that delirium is most continuous. There are generally also, in these cases, tremulousness, subsultus, want of control over the evacuations, extreme prostration, and not unfrequently diarrhœa.

After the completion of pustulation, and at the end of the secondary fever, which events are generally nearly simultaneous, a period of very uncertain duration and of very variable phenomena, during which the pustules dry up and disappear, supervenes. During the first three or four days, that is from the eighth or ninth up to the eleventh or twelfth day of the eruption, the pustules ooze or dry up, dark-coloured, thick, adherent scabs form, and the skin begins to exhale a characteristic fetid odour—the cutaneous inflammation at the same time rapidly subsiding. The separation of the scabs usually takes place during the third week of the disease; but the healing of all the sores may not be

completed for a week or two more, being preceded by the formation and detachment of successive crops of scabs. If the case be going on favourably, the febrile symptoms rapidly subside, the functions of the various organs are restored, the appetite returns, and convalescence is established. But it is during this period that many of the serious complications and sequelæ of small-pox manifest themselves, and delay the patient's recovery, or carry him off. These are most frequent after confluent small-pox, but may supervene on the milder forms. The following list comprises the chief of them. During the third or fourth week, boils are apt to appear on different parts of the surface; and then, though more generally later, subcutaneous and even deep-seated abscesses often form rapidly, attain a large size, and are long in healing. Erysipelas, more especially of the face and head, is not uncommon; and gangrene, or pyæmia, occasionally supervenes. Pustules sometimes form on the conjunctivæ; and from these or other causes ophthalmia is apt to ensue, which may be suppurative and end in ulceration or sloughing, and perforation of the cornea. Otitis is sometimes observed. Of internal complications, the most serious are suppurative pleurisy, pneumonia, and bronchitis. Inflammation or œdema of the larynx may also be fatal about this time; but this event is chiefly to be feared during the period of secondary fever.

The eruption of small-pox generally leads to more or less destruction of the cutis vera, and the formation of indelible cicatrices. In some cases (especially of the discrete variety) only a few scattered pits may result. But in the confluent disease, the destruction, especially on the face, is often most extensive, and the patient recovers, pitted, seamed, and scarred in all directions.

The description of small-pox just given is so full that we shall now, instead of discussing at length the groups of symptoms referrible to the various systems and organs, merely supplement it by adding certain details, which have either been omitted from it, or only slightly touched upon, or are of special importance.

The temperature, during the stage of invasion, usually rises rapidly to 104°, or even as high as 106·5°; during the early period of eruption, it falls several degrees, but for the most part remains distinctly febrile; at the period of maturation, the temperature again rises, in mild cases to 102° or 103°, in more severe cases to 104°, and when a fatal result threatens to 107°, or even beyond this.

The pulse is quickened, especially during the periods of primary and secondary fever, but otherwise presents no special peculiarity. The respirations also are accelerated in relation with the amount of febrile disturbance, and, under conditions of great prostration and danger, become shallow and suspirious. Vomiting is a characteristic symptom of the period of invasion, and anorexia with thirst, of the whole duration of the malady. In adults the bowels are generally constipated; and the occurrence of diarrhœa during the development of the rash is an

unfavourable symptom. In children, however, diarrhœa is a common, and on the whole a favourable sign, both in the period of invasion and subsequently. Salivation is almost invariable in confluent cases; comparatively rare and ill-marked in mild cases.

The urine presents the ordinary febrile characters; and in some cases (about one-third of the total number) contains albumen, with casts and occasionally blood-corpuscles. Albuminuria appears early in the disease and may continue to the end; but it rarely, if ever, leads to permanent renal mischief or to anasarca. According to Mr. Marson, suppression never occurs. Inflammation of the ovary or testicle is occasionally observed during the eruptive stage. Perspirations are usual in discrete variola from the beginning of the disease up to its termination; but they are generally absent in confluent cases, and are not common in children. According to the older authors, and also according to Trousseau, the swelling of the hands and feet which takes places in confluent small-pox during the period of maturation is a favourable sign.

The invasion-period, in children, is often marked by drowsiness; and coma and convulsions are not unfrequent; in adults, there is more or less giddiness and dulness; and convulsions occasionally supervene even in them; there is also frequently, and especially in severe cases, maniacal, busy, or muttering delirium. In confluent cases, the delirium may continue during the early period of efflorescence; and it generally reappears or becomes more severe at the time of the secondary fever. At this time, too, the patient is liable to outbreaks of violent mania. Tremulousness of muscles, subsultus, and picking at the bed-clothes, occur in the worst cases. The pain in the back, which is so characteristic of the onset of the disease, appears to be spinal, and is often associated with temporary paraplegia and loss of control over the bladder and rectum.

Many varieties of small-pox have been enumerated. Exceedingly mild cases are sometimes observed in which the period of invasion is well-marked, but in which no appearance of rash follows, or a few scattered pocks only are discovered on the skin or mucous membrane. Other exceptionally mild cases are met with, in which the disease begins with all the symptoms that usher in a well-marked attack of the disease; in which the pocks appear numerous yet discrete; but in which, at the period when suppuration should take place, the vesicles dry up. In both of these cases there is no secondary fever, and the patient rapidly convalesces. The most important forms of natural small-pox, however, are those which are known respectively by the names of 'discrete,' 'confluent,' and 'malignant' small-pox. In the *discrete* form the invasion-phenomena are generally well-pronounced; but the subsidence of febrile symptoms on the first appearance of the rash, and their abeyance until the commencement of suppuration, are constant; the secondary fever, too, is generally slight; and the patient

for the most part recovers without any complication. Nevertheless, in discrete small-pox there is some danger of death on the eighth or ninth day of the disease, from the sudden accession of cerebral symptoms, especially of coma. In the *confluent* variety, the symptoms are at all stages far more severe than in the discrete form; especially, there is little and very temporary remission of febrile symptoms; and, moreover, phenomena which are rare or absent in the latter, and have already been considered, assume considerable prominence here. It is in this variety, too, that complications and sequelæ are specially liable to come on. Death from confluent small-pox usually occurs from the tenth to the fifteenth day of the disease, and is due for the most part to a combination of coma and asthenia. But it may also supervene during the next month or two from the effects of sequelæ. *Malignant small-pox* is characterised especially by the early appearance of petechiæ and vibices, hemorrhagic effusion into the pocks and conjunctivæ, discharges of blood from the various orifices, and rapid collapse. The symptoms of invasion are usually intense, the patient looks from the first as if struck down by a mortal disease, and often dies on the fourth or fifth day, or before the eruption has had time to become distinct. Occasionally, indeed, the patient dies collapsed on the third day, before the appearance of the eruption, but possibly presenting chemosis, together with a few petechial spots about the lower part of the abdomen. There may be delirium; but the patient often remains conscious to the last.

Small-pox occurring after vaccination is generally modified in character and is termed '*modified small-pox*,' or sometimes and inappropriately '*varioloid*.' It commences with all the usual symptoms of small-pox, and may assume the characters of the discrete, confluent, or even malignant forms; but, about the time when the tissues around the pustules should inflame and swell and secondary fever be established, or even before that period, the eruption begins to dry up, and the febrile symptoms subside or present only very slight and transient exacerbation. Trousseau says that delirium is more common in modified than in natural small-pox, but is less serious; and that salivation rarely occurs in the modified confluent affection. It need scarcely be added, that the degree of modification varies; that the attacks, though generally benign, are sometimes serious; and further, that those occurring, even after successful vaccination, sometimes do not deviate appreciably from the natural disease.

Modified small-pox is for the most part a mild disease, and rarely fatal. Natural small-pox, on the other hand, is fatal in a very high degree. The statistics of the Small-Pox Hospital for twenty years show, that of those patients who had previously been vaccinated the mortality was at the rate of 6·56 per cent.; and that of those who had good vaccine cicatrices only 2·52 per cent. died. It is very different, however, as regards unmodified small-pox, which destroyed 37 per cent., or more than one-third of the total number attacked. Discrete small-

pox was attended with a mortality of 4 per cent., semi-confluent with a mortality of 8 per cent., and confluent with a mortality of no less than 50 per cent. Statistics from the same hospital show that the mortality among patients under five years of age was 50 per cent., and among those upwards of thirty still higher. The lowest rate of mortality was between five and twenty. According to Trousseau, children under one year never recover from small-pox—a statement, however, which is not absolutely true,—those between one and two rarely. Mr. Marson states that persons above sixty also almost invariably succumb. Pregnant women usually abort and die. They do, however, occasionally recover, whether abortion takes place or not.

Morbid anatomy.—The post-mortem examination of small-pox cases reveals but little beyond what has been already described. In most cases the blood is dark and imperfectly coagulated; although, in the ventricles of the heart fibrinous clots may be discovered. In the malignant form of the disease, extravasations of blood may be found beneath all the serous and mucous surfaces. The heart is generally flabby, the liver pale and soft, and the spleen more or less pulpy. The tongue presents a thick fur, which may be detached at the edges and elsewhere in patches. And the palate, fauces, nasal fossæ, larynx, trachea, and bronchial tubes, and even the œsophagus may be found more or less deeply congested, and covered with a granular film due to increase and softening of the epithelial layer; and may present, in addition, numerous excoriations which from their size and distribution are suggestive of their origin in the small-pox rash. Under such circumstances, the bronchial tubes are loaded with muco-purulent fluid, and the lungs are congested and œdematous, and possible pneumonic. As regards the skin-eruption, we may here add a few details which were out of place in a clinical account of the disease. The papules are due, partly to punctiform hyperæmia and germination of the cutis, partly to swelling, mucous degeneration and vacuolation of the cells of the rete mucosum. The central vacuolated cells of the thickened rete presently rupture, and unite to form an irregular anfractuous cavity. Into this central cavity, and into the surrounding vacuoles, serum exudes from the subjacent vessels, together with abundant leucocytes, and often a greater or smaller number of red blood-discs. By the continuance of these processes the pock enlarges in area and becomes purulent—its superficial wall being formed by the horny layer of the epidermis, its deeper wall by the surface of the corium, and its cavity, even to the last, presenting a multilocular or anfractuous character. The umbilicated form of the pock appears to be connected with its mode of development, and to be due to the fact, that while it extends peripherally its centre remains crossed by bands and filaments. The suppurative process need not implicate the true skin below; but not unfrequently it involves and destroys it to a greater or less depth, and is prolonged inwards along the hairs or glands. Under the former

circumstances the pustule leaves no permanent trace; under the latter a depressed cicatrix results presenting numerous pits upon its surface.

Treatment.—In the mildest forms of small-pox medicinal treatment is scarcely called for; in the severest it is useless; and indeed, under any circumstances, it has but little influence over the course of the disease. The patient should be placed in an airy chamber, which should be well ventilated, and kept at a uniform and medium temperature. He may take as medicine some cooling drink—lemonade, soda-water, or other saline or acidulated solution. If the bowels be confined, they may be acted upon by some mild laxative; if there be diarrhœa (especially in adults), they must be restrained by opium, or other astringents. The soreness of the throat may be relieved by warm bland drinks, or black-currant jelly; and, if there be much discharge from the nose and about the fauces, these parts may be washed with some mild detergent or astringent solution. Opium is often of value both in relieving the delirium and assuaging the pain of the invasion period; but it is especially useful during the period of secondary fever. If there be great tendency to collapse, ammonia may be serviceable. Nourishment should be regularly administered, and should consist of the materials generally suitable for febrile conditions, namely, milk, rice-water, gruel, beef-tea, and such-like. Alcoholic stimulants must be given according to circumstances; but are especially important in the malignant form of the disease, and in the later periods of confluent small-pox, or whenever there is tendency to collapse. As to local treatment, the patient should be kept clean, and frequently sponged with tepid water; and, as the eruption reaches its height, and in its decline, the eyes and various mucous orifices need especial care. They should be sponged, and dried; and anointed with olive oil; and if there be any tendency to conjunctival inflammation and ulceration, weak solutions of nitrate of silver or sulphate of zinc should be occasionally dropped into the eyes. Various plans have been suggested and employed to prevent pitting; but it is questionable if any is really efficacious. It has been recommended, to puncture the pustules, to wash away their contents, and then to insert into each a fine point of nitrate of silver. If this be done, it should be when the pocks first distinctly contain fluid; but the plan is scarcely applicable to the cases in which the prevention of pitting is most needed, namely confluent cases. The local application of strong carbolic acid has also been recommended. It is probably best, generally, to anoint the surface with carbolised oil. During the period of decline of the eruption, and that of convalescence, the strength of the patient needs to be supported in every way, by good diet, by stimulants, and by quinine or other tonics. The various complications of small-pox must be treated according to ordinary principles, bearing in mind, however, that their presence as a rule enfeebles the patient, and is therefore an indication for sustaining strength.

But the most important treatment of small-pox is the preventive, by means of inoculation with the small-pox virus, or that of cow-pox. The former plan has fallen into disuse, and is now penal in this country, yet no doubt under certain conditions it might be revived with advantage. The inoculated small-pox is a much milder disease than that contracted in the usual way; and, according to Dr. Gregory's analysis of the records of the Small-Pox and Inoculation Hospital of London, from the year 1746 to 1822, the deaths from it were at the rate of only three in a thousand. The mildness of the inoculated disease appears to be promoted by using the virus from a mild case, and by repeated selection of inoculated cases for the purposes of inoculation. It may be further promoted by inoculating those only who are at the age at which small-pox is least dangerous to life. The virus should be taken from a pock which has not yet begun to suppurate; and the operation of inoculation should be performed exactly like that of vaccination. Our remarks on vaccination will be given in the next article.

IX. COW-POX. (*Vaccinia:*) VACCINATION.

Definition.—A contagious disease of cattle, characterised by the local developmen of pustules (almost exactly resembling in their progress and results the pocks of variola) and communicable by inoculation.

Causation and relations with small-pox.—Cow-pox has been found to prevail epidemically at times in every country in Europe. Yet, although thus common, it is doubtful if it is communicable from animal to animal either by the breath or by the secretions. It is certain, however, that it is eminently contagious by inoculation from its specific pocks. Like most other affections originating from contagion, cow-pox by one attack protects against future attacks; but it similarly confers immunity against attacks of small-pox. It is this fact which gives so great an interest to all questions relating to its intimate pathology, and especially to the question of its exact relations with small-pox. Its identity with the latter disease was early surmised; and many arguments, in addition to the fact that it is protective against it, have been adduced in favour of this view. Thus, there is scarcely any appreciable difference between the pocks of the two affections, either in their anatomical characters or in their progress; it has been over and over again observed that epidemics of small-pox and cow-pox occur in relation to one another; and it is certain that since the introduction of vaccination the so-called 'natural' cow-pox has in great measure disappeared. But far more important than such facts as these are the experimental proofs which have been obtained by

Messrs. Ceely and Badcock, and some foreign observers. They have inoculated cows with small-pox lymph; have succeeded by this means in producing pustules at the seat of inoculation exactly like those of cow-pox; and with their contents have successfully imparted cow-pox to healthy cattle, and to the human being an affection exactly like that induced by ordinary vaccination. Further, by lymph thus obtained many years ago from bovine small-pox successful human vaccination has been perpetuated down to the present time. As confirmatory of this view of the relation between small-pox and cow-pox, it may be pointed out, that natural cow-pox occurs only in the teats and udders of *cows*—that is, in exactly the situations in which small-pox would be most likely to be given to them by inoculation from man; and also that cow-pox when experimentally inoculated from cow to cow, instead of being perpetuated, as it is in man, tends before long to die out. It seems clear, therefore, that cow-pox is small-pox, modified and deprived of its virulence by transmission through the cow.[1]

Symptoms and progress in cattle.—Natural cow-pox affects chiefly the udders and teats of cows, and is indicated in them by the development of a number of pustules which individually run through all the stages characterising the small-pox pustule. They begin as papules, in a few days become vesicular, and by the seventh, eighth, or ninth day attain their full development, measuring then from $\frac{1}{2}$ inch to $\frac{3}{4}$ inch in diameter. From that date the contents become purulent, and a congested areola, with much subcutaneous induration and thickening, forms. A thick dark adherent scab is developed by about the thirteenth or fourteenth day, which gets detached in the course of the following week, leaving a depressed cicatrix. The febrile symptoms which attend the progress of the disease are very slight, and for the most part of no importance; generally, moreover, the local affection is quite free from untoward complications. When cow-pox is given by inoculation, the papules as a rule first make their appearance at the end of three days; occasionally, however, on the second or the fourth day.

Symptoms and progress in man.—Cow-pox as it affects the human subject differs but little from the same disease in cows. No specific change is observable at the point of inoculation until the end of the second day, or the third day, when a small congested papule makes its appearance. This gradually increases in size, and on the fifth or sixth day has become a circular greyish vesicle, with a somewhat depressed centre. By the eighth day it has attained its full develop-

[1] Basing our opinions on some experiments of Chauveau, we adopted the opposite view in the former edition of this work. We have since then reconsidered the evidence on both sides; and are now satisfied that no merely negative evidence can invalidate the positive results obtained by Messrs. Ceely and Badcock; especially when we bear in mind that, as is admitted by all, small-pox is not readily inoculable on the cow.

ment—forming then a well-marked prominent greyish vesicle with a flat or cupped surface, and containing in its interior a colourless transparent viscid fluid. On the eighth or ninth day the contents of the vesicle begin to get purulent, a red areola forms, and some thickening and induration of the inflamed area take place. These phenomena increase during the next two days; the induration and thickening become greater and more extensive, the areola attains a diameter of from one to three inches, the pock itself undergoes some little extension, and its contents get wholly converted into pus. After the tenth or eleventh day the pustule begins to dry up, and the areola and other signs of inflammation to subside. By the fourteenth or fifteenth day a hard dark-coloured scab has formed, which contracts and blackens, and from the twentieth to the twenty-fifth day falls off, leaving a depressed pitted permanent scar.

The vaccinated patient does not usually present general symptoms or complications until about the eighth day, and during the two or three days immediately following. There is generally then some febrile disturbance, with restlessness, irritability, and slight derangement of the digestive organs; the glands next above the seat of operation usually get enlarged and painful; and sometimes a roseolous rash spreads over the vaccinated limb, and thence, may be, to other parts of the body. This rash is sometimes vesicular or papular.

When vaccination is performed directly from the cow, the progress of the eruption is usually somewhat retarded; and the local and general symptoms are all said to be more severe than when humanised lymph is employed.

In cases of revaccination one of three results may follow:—if the patient be fully protected, it produces no effect beyond a little local irritation due to the lancet-puncture and the introduction of irritant matter; if all protection have ceased, the operation is followed by the development of the typical pock; if there be simply impairment of protection, the results of the operation are modified. In the last case, the local effect comes on early, the papule (which may remain a papule or become an acuminated vesicle) attains its full development on the fifth or sixth day, and immediately after forms a scab which falls off in the course of a day or two; but there is generally a good deal of attendant local and constitutional irritation—much more, in fact, than occurs in primary vaccination.

Other circumstances besides those which have been considered occasionally modify the results of vaccination—among them, the age of the pock from which the lymph has been taken, and the health of the patient operated upon.

It must not be forgotten that cow-pox, whether in the cow or in man, is not comprised within its local manifestations; but that (however mild its attack may be) it is a disease involving the whole orga-

nism, as is proved by the marvellous influence which one attack has in protecting the body from subsequent attacks both of cow-pox and of small-pox, by whatever route and in whatever manner they may be introduced. Guided by what we know of inoculated small-pox—namely, that at the seat of inoculation, a papule appears, which gradually becomes a well-developed pock; that this is simply a local affection, which is followed about the eighth day by feverishness and other symptoms of invasion, and in two or three days more by the general eruption—it seems obvious to assume, that the pustules of cow-pox which appear on the udders of cows, and those which result from vaccination on the arms of men, are simply, as they appear to be, local affections, on which the true generalised disease (in this case abortive and altogether trivial in its symptoms) supervenes at about the period of maturation — in other words, that the period which elapses between inoculation and the full development of the pock corresponds strictly to the latent period of other exanthems.

Protective influence of vaccination against small-pox.—A belief in the protective influence of cow-pox against variola seems to have been commonly entertained in Gloucestershire during the latter half of the eighteenth century. And a similar belief appears to have prevailed about the same time in some parts of Germany. It is said, indeed, that a schoolmaster, named Plett, in Holstein, vaccinated two children in the year 1771; and it seems to be established that an English farmer, named Benjamin Jesty, performed the same operation on his wife and two sons in the year 1774. The value of vaccination was, however, first established on a solid basis by the scientific investigations of Edward Jenner, whose attention was directed to the subject while he was yet an apprentice, and whose first publication in reference to it appeared in the year 1798. We need not pursue in detail the further history of vaccination. It is sufficient to say, that its practice has been adopted since then throughout the whole civilised world; that the claim which Jenner originally made for it—namely, that it is as protective against subsequent attacks of small-pox as an attack of small-pox itself is, and neither more nor less so—has been verified by universal experience; that experience and experiment alike have shown that its protective influence is in no degree diminished by its continued transmission from man to man; and, lastly, that small-pox has died out or diminished in severity, in exact proportion as efficient vaccination has been generalised. It is certain, indeed, that thorough vaccinal inoculation confers in most cases absolute exemption for life; but that in some cases the protective influence diminishes in the course of years, so that if the patient contracts small-pox he has it in a modified and mild form; and that where small-pox has been rife, or epidemics have prevailed, the unwonted occurrence of the disease has been distinctly traced to neglect of vaccination, or to imperfect vaccination, or both. Mr. Marson's tabulated results of the experience at the Small-Pox

Hospital, during twenty years, show at a glance the accuracy of the above statements:—

Patients admitted with small-pox.	Number admitted.	Mortality per cent.
1. Having one vaccine cicatrix . . .	2001	7·73
2. „ two „ „ . .	1446	4·70
3. „ three „ „ . .	518	1·95
4. „ four or more „ . .	544	0·55
5. Stated to have been vaccinated, but having no cicatrix . .	370	23·57

It will be recollected that the mortality of primary small-pox is shown, by the same authority, to be 37 per cent.

Dangers of vaccination.—The only valid objection to vaccination is that it may, and occasionally does, induce or introduce maladies which the patient would otherwise have escaped. We do not here refer to the immediate accidental results of vaccination, such as erysipelas and pyæmia, which may equally follow on a mere prick or the simplest scratch; but to certain constitutional disorders, such as scrofula and syphilis, which have been attributed to it. There is no doubt that syphilis has been thus imparted; but the recorded cases are marvellously few, and these have been the result of gross carelessness or ignorance; for there is no reason to believe that a vaccinated child, who presents no visible indications of syphilis, could impart that disease, and but little even to believe that the pure lymph of a distinctly syphilitic child is charged with the syphilitic virus. As regards scrofula, the only ground for the belief in its inoculability by vaccination is the circumstance that lichen, eczema, and impetigo—affections which are common in children, especially about the period of teething, and by some erroneously regarded as scrofulous—occasionally supervene on vaccination, as they do on other forms of local irritation.

Performance of vaccination.—The operation of vaccination should be performed at as early a period of life as possible, especially if small-pox has been in any degree prevalent. It is now required by law that a child shall be vaccinated within three months of birth. It is desirable that it should be in good health, and free from skin-disease. In order to obviate the tendency which the vaccinal influence has to die out, it is now almost universally held that the operation ought to be repeated about the period of puberty. And further, it is always important, in the case of persons who are, or are liable to be, exposed to small-pox (especially if they have only imperfect vaccinal marks and have not been successfully revaccinated), that the operation should be at once repeated. But it should be borne in mind that vaccination has no modifying effect on small-pox which has been previously contracted, unless it be so timed that the maturation of the vaccine vesicle shall precede the period of the variolous invasion. Thus, since the primary vaccine vesicle attains its full development on the ninth

or tenth day, and the latent period of small-pox is usually twelve days, primary vaccination, to have any beneficial effect, should be performed certainly not later than the second or third day after exposure to the variolous contagion. The vesicle, however, which follows revaccination attains its maximum on the seventh or eighth day; so that, if the patient has been previously vaccinated, the operation may possibly be beneficially performed as late as the fourth or fifth day after exposure.

The lymph for vaccination should never be taken from persons who are diseased, or in whom there is any suspicion of syphilis or other infectious disorder; nor from pocks which are ill-developed or purulent; nor from those which are the product of revaccination. Good vaccine lymph is yielded by normal pocks from the fourth or fifth to the eighth day after inoculation. That of a later date should never be employed. As a rule the lymph is taken on the eighth day. The vesicles should be freely punctured with the point of a lancet, care being taken to avoid hemorrhage; and the fluid which exudes should then at once be employed for vaccination, or should be preserved on ivory points which may be dipped into it, or between glasses, or preferably in capillary glass tubes. No squeezing of the vesicle should be had recourse to; but if, after all the lymph which first flows has been used, the surface be gently wiped, a fresh exudation of good lymph usually takes place. Lymph may also be diluted with glycerine, in the proportion of from one to two parts of glycerine to one of lymph, and thus preserved—a method of special value when lymph is scarce.

Vaccination is generally, and certainly most conveniently performed on the upper and outer part of the upper arm. There, four or five distinct punctures should be made at $\frac{1}{2}$ or $\frac{3}{4}$ inch distance from one another. Various modes of performing the operation are recommended. The simplest is to make with a sharp, clean, well-charged lancet, in the stretched skin, a valvular puncture directed from above downwards, and sufficiently deep to wound the vessels of the cutis. A second method, of which there are numerous modifications, is to make groups of parallel or crossed scratches, or fine punctures, so as to allow of a little oozing of blood, and then having wiped the blood away to anoint the surface with the vaccine lymph. If the groups be small they should be five in number; if large three will suffice. If the lymph which is employed be fresh, or have been preserved in capillary tubes, it may at once be applied on the point of the lancet; but if it have been preserved in the dry condition, it is essential that it be first moistened thoroughly with a small quantity of water. If no result whatever follow the operation, whether it be in a case of primary vaccination or in one of revaccination, either the lymph employed is inefficient, or the operation has been imperfectly performed, or (which is less probable) the patient is insusceptible. Under any circumstances, the operation should be repeated until a definite local result of some kind or other is obtained.

X. CHICKEN-POX. (*Varicella.*)

Definition.—A specific contagious disorder, characterised by the appearance of vesicles in successive crops, which in the course of two or three days form scabs.

Causation.—Varicella has been largely confounded with small-pox, of which it has been regarded as a modified variety. This view is still entertained by Hebra and some other writers. Of the perfect distinction, however, between them there can be no doubt; for the one disease is not protective against the other, although each is protective against its own future attacks; the one disease never imparts the other; and they occur in independent epidemics. Chicken-pox is contagious in a very high degree, and spreads both by means of the air and through the medium of fomites. It is doubtful whether it has hitherto been imparted by inoculation. It occurs epidemically; but its epidemics seem to be neither so frequent nor so widespread as those of measles, hooping-cough, and scarlet fever. It attacks children mainly, yet adults are by no means exempt.

Symptoms and progress.—The period of incubation is somewhat uncertain. According to different authors, it varies between four or five and sixteen or seventeen days. In some cases this stage is of exactly a week's duration. But more commonly perhaps it lasts a fortnight. The invasion is marked by febrile symptoms, which are occasionally severe but present no distinctive character, and which, generally in a few hours, at all events before the completion of twenty-four, are followed by the appearance of the rash. This consists in the first instance of a number of rosy papules, not unlike the spots of typhoid fever, appearing singly, or in groups of two or three, on various parts of the body—head, face, trunk, limbs—but most commonly, perhaps, first upon the chest. These in the course of the next day or two, or even after a few hours, become distinct vesicles, containing a transparent fluid, and usually surrounded by a more or less distinct inflammatory halo. The vesicles, which are at first small and rounded or acuminated, increase in size for a day or two, becoming sometimes as large as a split pea, occasionally irregular in form, and often umbilicated; their contents at the same time get milky. They then rupture or dry up, and small dark-coloured adherent scabs result. The formation of the scabs is completed at the end of four or five days or a week from the first sign of illness; and they may remain adherent for two or three days or even a week longer, when they separate, leaving red stains, which are slow to disappear, and not unfrequently permanent depressed cicatrices. The eruption is not limited, however, to the generally scanty crop which first appears. But during the first three or four days of the disease fresh crops of papules in largely increased numbers, and irregularly distributed, spring up day by day;

and these go through the same stages as those which were first developed. During the progress of the disease vesicles with inflamed areolæ usually appear, in small numbers, on the palate, sides of the tongue, and mucous surface of the lips and cheeks.

The general symptoms of varicella are for the most part slight and unimportant. There is commonly some feverishness, languor, and loss of appetite; and the fever is liable to nocturnal exacerbations during the maturation of the vesicles. The temperature often rises to 101°, and may reach 104°. The tongue probably remains clean throughout. Occasionally, the symptoms are much more severe, though never probably so severe as to excite serious alarm. Death rarely if ever results.

The malady usually attains its height in a week or ten days, and runs its course in ten days or a fortnight. The complications and sequelæ are unimportant; nevertheless, children often remain weak and out of health for some time after an attack.

Treatment.—The patient should be separated from those who are liable to take the disease, and confined to his room, if not to bed. He should be prevented, if possible, from scratching his pimples, those at least upon the face, in order to diminish the liability to pit. No further special treatment is necessary.

XI. TYPHUS.

Definition.—A highly contagious fever, lasting from two to three weeks, and attended with a characteristic measly eruption coming out from the fourth to the seventh day.

Causation and history.—Typhus fever seems to be a disease especially of temperate climates. No European country is free from its occasional epidemic prevalence; but from Ireland it is probably never entirely absent; and indeed Great Britain and Ireland may be regarded as its headquarters. Epidemics have occurred in the United States and in Canada. There is even now some doubt as to whether it has ever been observed in India; but, excepting this doubtful case, it is quite unknown in tropical countries. It has been introduced into Australia and New Zealand, but has not spread there. Typhus appears, for the most part, in casual outbreaks which assume an epidemic character, spread widely, and after lasting for months or years subside and die out. Almost all recorded epidemics seem to have been satisfactorily traced to long-continued overcrowding, in association with defective ventilation and personal filth. With these conditions starvation no doubt is often to a large extent combined. But starvation alone, such as results from famine or widespread want (from whatever

cause) of the necessaries of life, leads to the development rather of relapsing fever than of typhus; while, on the other hand, typhus has not unfrequently become epidemic where there has been no starvation, but where the other conditions which have been enumerated have prevailed in a marked degree. Epidemics of typhus have originated mainly, in the overcrowded parts of great cities, during seasons of distress and want and consequent exceptional overcrowding; in armies, under equivalent conditions; and in prisons. There can be no doubt, indeed, that overcrowding and bad ventilation are most effective agents in concentrating the typhus poison, and in promoting the spread of the disease; and it may be added that anything which depresses either body or mind—want of food, fatigue, intoxication, fear, anxiety, perhaps even the debility of convalescence—must be regarded as a predisposing cause. In the countries in which typhus chiefly occurs, season and weather appear to exert no direct influence over either its origin or its spread. All ages are liable to its attacks, although it appears from statistics that it is most common between fifteen and twenty-five; and males and females suffer from it in nearly equal proportion. One attack confers almost complete immunity against subsequent attacks; yet, occasionally, two and even three seizures have been observed in the same individual. Excepting those who have thus acquired protection, everyone is liable to take typhus. It is true that some unprotected persons, even when exposed daily to the influence of the disease, fail to contract it; but many cases are on record where such persons, after years of immunity, have been attacked with it at last and have then succumbed to their attack.

That typhus is a highly contagious disease is established by overwhelming evidence. Its poison is carried by the atmosphere, and is absorbed and retained in a potent condition for a considerable time by fomites. But it presents certain marked peculiarities of behaviour external to the system; thus it clings, as it were, around the body of the patient, and seems to be rapidly destroyed by diffusion through the atmosphere: so that while its operation is intense under appropriate conditions of overcrowding and bad ventilation, it is almost *nil* under opposite circumstances; and hence the disease rarely spreads (excepting to the immediate attendants) in the wards of a well-arranged hospital, or among the households of the middle and upper classes. The contagium of typhus is probably exhaled with the breath and from the general surface. It is doubtful, however, whether the other excretions are infective, and whether the disease can be imparted by the dead body. Both the breath and the sweat of typhus patients yield a characteristic offensive odour, and there is reason to believe that the contagiousness of a case has some direct proportion to its smell. Dr. Murchison considers that the disease is most contagious from the end of the first week up to convalescence.

Although it is now admitted by all the best observers that typhus

when once it has made its appearance is eminently contagious; it is still a moot question, whether typhus epidemics owe their origin to new developments of the typhus poison, or are due to the presence in a latent form of the contagium, which is rendered operative by the concurrence of suitable conditions. The former hypothesis is strongly advocated by Dr. Murchison. His arguments, however, though forcible are not conclusive; and we must confess that the latter view seems to us infinitely more consonant than his with the analogies afforded by the exanthemata, and with the present state of pathological knowledge.

Symptoms and progress.—The latent period of typhus appears to be of very uncertain duration. Cases are recorded in which the symptoms of invasion manifested themselves almost immediately after exposure to the concentrated poison. On the other hand, the primary symptoms have in some cases failed to appear until after the lapse of twenty-one days, or even more. The usual period varies probably between five or six and twelve or fourteen days. The invasion is occasionally heralded by an ill-defined sense of poorliness lasting for a day or two; at the end of which time, or much more commonly without any such warning, the initial symptoms manifest themselves. These generally consist in a sense of chilliness or slight rigors, pain in the forehead and back, and soreness in the thighs and other fleshy portions of the limbs; with which are associated before long, or from the commencement, increased heat of skin, occasional slight sweats, diffused dusky redness of face and congestion of conjunctivæ, acceleration of pulse, furring of tongue, anorexia and thirst, scanty and high-coloured urine, muscular weakness, lassitude, giddiness, and loss of sleep, or disturbed sleep with tendency to dream. Occasionally there is some nausea or even sickness, and generally the bowels are constipated. For the first two or three days, notwithstanding gradual aggravation of the symptoms, the patient may not feel sufficiently ill to take to his bed. From the third to the seventh day—generally on the fourth or fifth—the characteristic measly eruption makes its appearance on the sides of the chest and abdomen, and on the backs of the hands, wrists, and elbows, and in the course of a couple of days becomes general over the trunk, arms, and legs, and sometimes, but much more rarely, shows itself on the neck and face. It remains out, well developed but undergoing slight changes of colour, for two or three days more, then gradually fades, and finally disappears by about the fourteenth day, unless it assume a petechial form, when its disappearance is retarded. About the time when the eruption commences, or a little earlier, the patient has probably taken to his bed, and has begun to be apathetic and forgetful, to present a dull and listless expression, and to ramble at night. Presently he loses his headache, becoming, however, increasingly dull, forgetful and stupid; and the delirium, which had hitherto been nocturnal and probably limited to the moments between waking and

sleeping, becomes constant. Occasionally the delirium is violent and maniacal, and the patient requires restraint; sometimes it is the busy delirium of delirium tremens; but much more commonly it is of the low muttering kind, known by the name of 'typhomania,' into which, indeed, the other varieties tend soon to merge. In this condition the patient can at first be readily recalled to himself, and will answer correctly, and do what he is told to do. His aspect becomes more oppressed; the redness of his face and eyes, and his rash, assume a more dusky tint; sordes begin to collect on his teeth, and his tongue becomes dry and brown; his respirations and pulse increase in frequency, and the latter gets small, weak, and sometimes dicrotous or irregular; his temperature falls somewhat; his skin becomes clammy, his limbs tremulous; and general debility increases rapidly. By about the tenth day the typhoid symptoms of the disease are fully developed; the patient has become still feebler; he lies in bed on his back with his mouth half open and his eyes half closed, taking no notice of what is going on around him; he is in a semi-comatose condition, muttering at times unintelligibly and incoherently, breathing sometimes more rapidly sometimes less rapidly than natural, and probably moaning or groaning with each respiratory act; his lips and teeth are coated with sordes, his tongue is small, hard, dry, and black; he tends to sink towards the bottom of the bed; his muscles are tremulous, and he has subsultus tendinum, especially in the arms, and floccitatio or a tendency to pick at the bedclothes; his motions are passed unconsciously, but his urine is generally retained, though dribbling away perhaps from the over-distended bladder; his pulse has become extremely feeble, dicrotous, irregular; his temperature probably still shows an inclination to sink; the rash fades or becomes replaced by petechiæ; and perspirations break out. There is a tendency also to the formation of bed-sores.

These symptoms probably continue for several days, the patient meanwhile becoming more and more prostrate and comatose. And then, generally on or about the thirteenth or fourteenth day, either the coma becomes profound, the temperature rapidly rises, and the patient sinks; or he falls into a gentle sleep from which, after some hours, he awakes sensible and convalescent, with a greatly diminished temperature and pulse, but in a condition of extreme debility. If the case continues to go on favourably, the tongue quickly cleans, the appetite returns, and restoration to perfect health ensues at the end of three or four weeks.

We will now discuss some of the more important phenomena of typhus *seriatim*. The temperature rises at once, and generally attains its maximum, which rarely exceeds 106° in adults and 107° in children, between the middle and end of the first week. Exceptionally it does not rise above 103°. It remains at its maximum for two or three days, and then (usually between the seventh and tenth day) falls

slightly—continuing to fall until the period of crisis, when, according as death or recovery takes place, there is either a rapid rise which may exceed by several degrees that previously attained, or a sudden fall. The diurnal variations are slight and irregular, though on the whole tending to present an evening rise and a morning fall. If a high temperature be maintained or an unusual rise take place during the second or third week, some inflammatory complication is probably present.

The eruption of typhus embraces two factors, namely, a mere mottling of the surface, and distinct dusky-red spots. They are usually present together. The mottling, which soon becomes general, precedes the development of the rash, and first appears in those situations in which the rash subsequently commences. It is due to the appearance of abundant ill-defined dusky patches which are not elevated, vanish on pressure, and individually are scarcely perceptible. The rash presents the colour and very much the aspect of that of measles. The spots, however, are smaller and less elevated, and do not assume a crescentic arrangement. They are slightly raised, roundish, fading at the margins, and at first disappear on pressure. For the first day or two, their colour is comparatively bright, and due simply to stagnation of blood in the capillary vessels; during the subsequent two or three days they assume a dusky hue, the result probably in some degree of the transudation of the colouring matter of the blood; and then either they fade away, or hemorrhage takes place into them and they become converted into petechiæ. The typhus eruption is almost invariably present. In the year 1864 it was observed in the London Fever Hospital in 97·77 per cent. of the cases admitted. In children it is often very slight and of short duration, and may therefore be readily overlooked. In adults it is usually well developed; and generally the severity of the disease is in proportion to the abundance of the rash. The copious formation of petechiæ which often occurs towards the latter part of the second week is an unfavourable sign.

The respirations are generally slightly increased in number during the earlier period of the disease. In the typhoid stage they may rise to thirty or forty in the minute. From the beginning of the disease there is very often a slight cough; and this may continue throughout the illness; or it may increase and be attended with mucous expectoration which is sometimes tinged with blood. It is connected with the congestion of the bronchial tubes and lungs which so commonly attends typhus.

The action of the heart is weak; and towards the latter period of the disease the first sound often becomes inaudible. The pulse is always feeble and generally small; and its feebleness and smallness increase as the disease advances, until at length it becomes undulating, thready, irregular, and almost imperceptible. Its rate presents great variety. In adults it usually ranges between 100 and 120. During

the first few days it rarely exceeds 100. Subsequently it rises in frequency; and it may reach 130 or 140 or more in the minute. But when it exceeds 120 the danger is generally very great. Occasionally it falls in the second week to 40 or 50. In children the pulse is usually much quicker than in adults.

Sickness is not a common feature of typhus, although it occasionally marks its onset. The bowels are generally constipated and the motions normal. But occasionally diarrhœa occurs early in the disease; and it is by no means uncommon about the period of the crisis, when also it may be dysenteric. The tongue at the beginning may be only abnormally red, or even natural; but it is soon covered with a thick whitish fur, which gradually gets yellowish, and towards the end of the first week brown. Later the tongue shrinks and becomes black; and equivalent changes take place in connection with the lips, palate, and fauces.

The urine is scanty, high-coloured, of high specific gravity, and acid, during the early period of typhus, and contains an excess of urea and sometimes of uric acid and of urates; which latter may be deposited. Later on the urine becomes paler and more abundant, and the urea falls considerably below the normal standard. Chlorides are deficient and occasionally disappear during the pyrexial condition. Albumen in small quantities, sometimes accompanied by blood-corpuscles and granular casts, is frequently present in the urine. It is not certain at what date albuminuria generally appears, or when it generally ceases; nor is it a symptom of importance. It is most common, however, in severe cases, and probably usually commences on the third or fourth day.

Pregnant women rarely miscarry; nor does pregnancy or miscarriage add materially to the danger of the patient. The prematurely-born fœtus, if old enough, generally survives.

The symptoms referrible to the nervous system always form a characteristic part of typhus fever. Most of these have already been considered. The patient at first has headache, with some dulness and confusion of mind (which impress themselves on his manner and on the expression of his features), and sleeplessness. In a few days he begins to wander at night between waking and sleeping, gradually becoming more stupid and forgetful in the intervals. At the end of the first week or earlier, the delirium becomes constant, though still worse at night time; and the patient is perhaps drowsy in the day. The delirium, as has been pointed out, may vary in character, but generally soon lapses into typhomania. Gradually the patient becomes more and more unconscious; and if the case be about to end fatally, he probably falls into profound coma, occasionally preceded by convulsions. The coma sometimes assumes the character of what is termed 'coma-vigil,' in which the patient lies quite unconscious with his eyes open and fixed. In the early part of the disease there is generally some intole-

rance of light and singing in the ears. At the latter part deafness often comes on; and if the patient be comatose the pupils usually contract to mere points. The muscular pains of the first period, the muscular tremors which soon supervene, and the subsultus, floccitatio, and loss of control over the rectum and bladder of the later periods, are all more or less directly dependent on nervous implication.

Typhus fever varies in its severity. It is sometimes so mild, of such short duration, and so free from any distinctive character, that excepting under the guidance of attendant circumstances correct diagnosis is impossible. In many cases, again, even where the fever is present in a well-marked form, the typhoid stage is never developed; but somewhere between the seventh and tenth day, when usually the patient begins to manifest the gravest symptoms, amendment takes place,—the tongue never becoming dry and black, the delirium never occurring at other times than between sleeping and waking. Next, we have the typical case from which our description has been drawn, in which all stages are well developed and the commencement of convalescence is delayed to between the thirteenth and twenty-first day. Further, we meet with cases in which recovery is delayed by the supervention of complications or sequelæ. And, lastly, cases occur in which the patient dies prostrate and delirious, or comatose, within the first week of the attack or even within the first day or two.

Death is due for the most part to a combination of asthenia and coma. It is most common about the end of the second week. Occasionally, and more in some epidemics than others, the patient dies from the sixth to the eighth day. And many cases are recorded where death has occurred even as early as the first or second day. Death at the end of the first week is often due in some measure to pulmonary congestion; and after the fourteenth day either to this, or to some other complication or sequela. The fatality of typhus is considerable. Of patients treated in hospital the mortuary rate is about 15 per cent. But these comprise an exceptionally large proportion of the gravest cases; and there is reason to believe that the death-rate among all persons attacked with typhus is no more than 10 per cent. Among the causes which determine its fatality by far the most important is age. Under twenty the mortality is very low. Dr. Murchison's statistics, taken from the records of the London Fever Hospital, show a mortality in cases under five of 6·69 per cent.; between five and ten, of 3·59 per cent.; between ten and fifteen, of 2·28 per cent; and between fifteen and twenty, of 4·46 per cent. Between twenty and twenty-five the mortality rises to 10·33; from which date upwards it increases pretty uniformly, lustrum by lustrum, until between fifty and fifty-five it amounts to 49·62 per cent., and between seventy-five and eighty to 84·37.

The sequelæ of typhus are not very numerous or characteristic. Among the more important may be enumerated, bronchitis and pneu-

monia, which may occur during the progress of the fever or during convalescence; gangrene, in the form of bed-sores, or affecting the toes, fingers, nose, penis or pudenda, or in children mainly in the form of noma; erysipelas; abscesses in the parotid or submaxillary region, or in the axillæ or groins; suppurative inflammation (said to be pyæmic) of joints; anasarca of legs; and mental imbecility or mania. These sequelæ are all serious; and two of them—noma and suppuration of the joints—are almost invariably fatal.

Morbid anatomy.—The post-mortem examination of typhus patients reveals little that is special. There is a tendency in the body to rapid decomposition; the internal organs are for the most part softened and congested; and the blood is dark, stains the vessels which contain it, and coagulates imperfectly. The lungs are usually deeply congested and very lacerable in their dependent parts, and sometimes solid from inflammatory changes. The spleen is generally softened, and not unfrequently somewhat enlarged. The large intestines occasionally show traces of dysenteric inflammation.

Treatment.—It is important that typhus patients should be treated in large, airy, well-ventilated chambers, and therefore that they should be removed from the overcrowded tenements which as a rule they occupy. The attendants upon them should be seasoned and young. In the later periods of the disease, the bladder should, if necessary, be periodically emptied by means of the catheter, and the patient be kept scrupulously clean so as to prevent the formation of bed-sores.

The general medicinal treatment of typhus is of little importance. There is no specific remedy, and no means which enable us to cut it short. It is desirable, however, to relieve the thirst from which the patient suffers, and to promote the evacuation by the kidneys of the effete matters which speedily overload the blood. For this reason, so-called 'febrifuge' medicines, which are at the same time mildly diuretic, are doubtless useful. Among them we may enumerate, soda-water, and chlorate, nitrate, citrate, or other salts of potash well diluted, acetate of ammonia, and the like. It is desirable also to keep the bowels fairly open either by occasional laxatives or by enemata. On the other hand, if there be diarrhœa it should be checked by opium or other ordinary forms of astringents. When pulmonary congestion complicates the progress of the fever, a little ipecacuanha or antimonial wine with a few drops of laudanum may be added to the mixture, or, better still, ammonia. If there be much insomnia or acute or busy delirium, opiates in larger doses may be administered by the mouth or subcutaneously, or recourse may be had to chloral or bromide of potassium. Rest, too, may be promoted by cutting the hair short or shaving it and applying cold lotions or ice to the head. It need scarcely be said that opiates should not be given when there is any tendency to coma, or to suppression of urine. In the typhoid stage, ammonia is probably the most valuable medicine.

The exhibition of stimulants always becomes an important question. There is no doubt that in a large proportion of cases patients do not require them; but there is also no doubt, that many need them, and that few if any are injured by them in moderation. In persons of enfeebled constitution, in habitual drinkers, and in such as are of advanced age, it is for the most part desirable to commence their administration early; and in all cases where the heart shows signs of unusual feebleness, where there is extreme prostration, or where typhoid symptoms come on early or are severe, stimulants should be at once had recourse to. The amount to be given under such circumstances must depend on the condition of the patient, and on the effect which they produce. It matters little what form of stimulant is selected.

From the beginning the patient loathes food; but the maintenance of his strength is imperative. Hence, those foods which he can be made to take should be given to him systematically, in small quantities, and at frequent intervals. Nothing is better than good milk, of which, by judicious management, from two to three or four pints may often be given daily. But all patients will not take milk. Alternative articles of diet are rice-water, barley-water, gruel, and eggs beaten up with milk, wine, or tea. Beef-tea, broth, arrowroot, and jelly are useful adjuncts. Ice may often be added beneficially to the patient's drinks.

During convalescence quinine or other forms of tonics are important; and the diet should be gradually modified to that of health, and should be abundant, frequently administered, and wholesome.

XII. PLAGUE. (*Pestilentia.*)

Definition.—A contagious fever, closely resembling typhus in its symptoms, but distinguished from it by the absence of any true rash, and by the development of buboes and carbuncles.

Causation and history.—The early history of the disease to which the term 'plague' is now applied is uncertain. It is known, however, to have prevailed from an early period of the Christian era in the countries which it now mainly affects—namely, Turkey, Asia Minor, Egypt, and Morocco—and to have spread thence at various times over the continent of Europe. In the seventeenth century numerous epidemic outbreaks occurred in Holland and in this country, the last being the Great Plague of 1665. Since then it has occasionally been imported into the countries bounding the Mediterranean basin, and into Russia. In Asia Minor and Egypt it may almost be regarded as endemic; but occasionally, at irregular intervals, breaks out into terrible epidemics. Whatever the specific cause of plague may be, it is certain that its epidemic occurrence is materially influenced, if not determined, by

conditions almost identical with those which determine outbreaks of typhus—namely, privation, filth, and overcrowding. Like typhus it affects mainly the poor, is apt to break out in armies engaged in warfare, and among the inhabitants of beleaguered cities.

Plague is eminently contagious, and is communicable by the breath, by fomites, and by inoculation. The cause of its spread, therefore, is doubtless a specific contagium. Although an attack of the disease is to some degree protective, subsequent attacks have been abundantly met with.

Symptoms and progress.—The duration of the incubative period is uncertain. The symptoms generally commence with chills or rigors, rise of temperature, pains in the forehead, back, and limbs, giddiness, anxiety, and sickness; on which speedily supervene, great loss of muscular power, extreme feebleness of the heart's action—indicated by rapidity, irregularity, and smallness of pulse, and prostration—and marked dulness or stupidity of expression, with corresponding hebetude of mind, passing quickly into delirium and coma, and sometimes convulsions. The tongue, thickly coated from the beginning, soon becomes dry and black. The bowels are generally somewhat loose, the urine scanty and occasionally suppressed. And hemorrhages from the various mucous surfaces are not unfrequent.

Within two or three days after the first appearance of symptoms petechiæ not unfrequently appear over the surface of the body; and besides these, the more characteristic glandular swellings or buboes, which are chiefly to be detected in the neck, axillæ, and groins. Subsequently carbuncles become developed at various parts of the surface, generally, however, in the extremities. The appearance of petechiæ is by no means invariable, and is regarded as being of bad augury. The buboes enlarge, sometimes to a considerable size, reach their height (if the patient survive so long) at about the end of the eighth or ninth day, and then either subside, or (more rarely) suppurate. Carbuncles are comparatively unfrequent and for the most part show themselves towards the decline of the disease; they vary in size and intensity of inflammation, and in numbers from one to about a dozen.

Death from plague sometimes takes place within twenty-four hours after seizure. Severe cases not unfrequently prove fatal on the second or third day of the disease. Many patients die on the fifth or sixth day. Occasionally death is delayed until the second or third week; but is then probably due mainly to the effects of complications.

It is generally acknowledged that it is impossible to distinguish plague positively from typhus, either by its early symptoms, or by the first few cases that come under treatment—the mode of invasion and the general symptoms and progress of the two diseases presenting many points in common. Petechiæ are frequent in both diseases, and buboes are of occasional occurrence in typhus. But plague does not present the true typhus rash; and the buboes, which are quite excep-

tional in typhus, are almost constant in plague; and, further, the mortality of plague is much greater than that of typhus, and its fatal issue occurs much earlier.

Morbid anatomy.—Patients dead of plague show, as in typhus, a rapid tendency to decomposition, fluidity or imperfect coagulation of blood, congestion, softening and enlargement of organs, and petechial extravasations beneath the serous and mucous surfaces. But besides these phenomena, there is a general enlargement of the lymphatic glands, which vary individually from the size of a goose's egg downwards. This enlargement is not limited to the superficial glands, but involves those of the interior of the thorax and abdomen, and is often attended with congestion and softening, and in some cases with suppuration.

Treatment.—The rules and details of treatment which have already been given in regard to typhus are applicable to plague. No specific remedies are known. Buboes and carbuncles only call for the usual treatment of such affections.

XIII. RELAPSING FEVER. (*Famine-Fever.*)

Definition.—A contagious disorder, characterised by a sudden attack of high fever, lasting for about a week; and then apparent convalescence, followed after about fourteen days from the primary accession by a second attack of fever. A further relapse now and then occurs about the twenty-first day.

Causation and history.—The geographical limits of relapsing fever have not been fully ascertained. Our knowledge of it has been chiefly derived from epidemics originating in Ireland, whence it has spread to England and Scotland. It appears also to have broken out independently in Scotland. Epidemics of it have, within the last few years, been observed in Russia and Silesia; and there is good reason to believe that it is not unknown in America, India, and parts of Africa. There seems to be a very close relation between starvation and relapsing fever, which has hence been denominated famine-fever. All the more recent and most fully investigated epidemics appear to have arisen during the prevalence of extreme destitution, and among the classes who have mainly suffered from destitution. Further, although the disease is highly contagious and liable to affect all who come within its influence, it is mainly carried by tramps and vagrants; and when it spreads among populations not suffering from famine, still chiefly affects those sections of them that are least well-fed. Overcrowding and filth are almost necessary accompaniments of famine; but these are not thought to have any special influence in the production of relapsing

fever. At all events, when these conditions exist (as they often do) independently of famine, they are never known to promote the outbreak of the special famine-fever. Season and other climatic conditions appear to exert no influence over its development or spread; and its attacks are probably in no degree determined by age or sex; although it is true that statistics show a larger proportion of sufferers among males than females. The contagion of relapsing fever is carried by the atmosphere, and also by fomites. But there is good reason to believe that its influence extends but a short distance around the patient, that it is readily lost by dilution, and that in order to ensure its action a large dose of poison or a long exposure to it is essential. There can be no question that when the disease spreads its source is a specific contagium,[1] which is evolved by the body already diseased and is absorbed by that which is about to suffer. It is a debated point, however, whether those who are primarily affected breed in their systems the contagion which they afterwards evolve, or whether they have derived it from some external source where it has lain dormant; in other words, whether during the progress of starvation the specific poison is engendered within the body, or whether the effects of starvation are such as to render the frame liable to be affected by a poison, which under other circumstances is innocuous. The question is one which scarcely yet admits of a positive solution. Those who look especially to the close connection between this fever and famine, and to the long intervals which elapse between successive outbreaks, naturally lean to the one view; those who give weight to the analogies between it and the exanthemata lean as naturally to the other. A marked peculiarity of relapsing fever, as compared with other diseases of its class, is the fact, that one attack does not confer safety from subsequent attacks; at all events, many persons have been known during one and the same epidemic to suffer from it two or three times at short intervals. It may be remarked, however, that the fact of a patient recovering spontaneously from an infective disease is a proof that he enjoys at least a temporary freedom from liability to be affected by it. And hence it may be assumed that immunity is actually conferred by an attack of relapsing fever, but that the period of immunity is mostly of very short duration.

Symptoms and progress.—The latent period of relapsing fever varies. Its extreme limits are probably two and sixteen days. Cases, however, are recorded in which the attack seemed to follow almost immediately on infection. Dr. Murchison concludes that the period of incubation is, on the whole, shorter than that of typhus.

The onset of the disease is for the most part sudden. The patient is seized with a feeling of chilliness or with rigors, attended with severe pains in the forehead, trunk, and limbs. This condition is soon followed by intense heat and dryness of surface, increased frontal

[1] For further information on this point, see pages 134, 135.

headache, and lumbar and other pains, giddiness, frequency of pulse, thirst, and loss of appetite. The latter symptoms continue with some slight variation—the dryness of skin, however, frequently alternating with perspirations—until the third, or more commonly the fifth or seventh day of the disease; when, often preceded by a slight rigor, a copious perspiration almost suddenly breaks out, which lasts for a few hours, and is then followed by a remarkable reduction in the rate of the pulse and of temperature, and, with the exception of some remaining lassitude, almost complete restoration to health.

The following is a more detailed account of the several symptoms which attend the febrile attack. The temperature almost from the commencement is very high, often ranging from 104° to 108·5° F.; the pulse is rapid, generally over 110, and often reaching 130 or 140 in the minute; the tongue is thickly coated with a white fur—the tip and edges being red—and occasionally towards the termination the centre of the organ gets dry and brown; the teeth are free from sordes; the patient suffers from extreme thirst, generally from anorexia and often from vomiting; in rare cases there is slight hæmatemesis; the bowels are mostly constipated; there is often considerable tenderness in the region of the liver and spleen, both of which organs become increased in size, and in many cases jaundice appears about the second or third day; the urine varies in quantity, but presents an excess of urea, and occasionally contains albumen and even blood—towards the later period of the attack suppression may take place; the pains in the head, trunk, and limbs continue, all being severe, and the latter mainly affecting the joints and presenting, therefore, a rheumatic character; the patient for the most part retains perfect consciousness, but generally suffers greatly from want of sleep and from frightful dreams when he does sleep; delirium, which may be maniacal, sometimes occurs about the period of the crisis; stupor, coma, and even convulsions supervene, though rarely, about the same period, and are then probably due to uræmic poisoning. The patient seldom presents the congested conjunctivæ and dull puzzled aspect of typhus fever. The critical perspiration is occasionally attended with, or replaced by, an attack of diarrhœa, or of hemorrhage from the nose, bowels, or elsewhere. No rash is ever seen, except perhaps a few petechiæ towards the end.

During the intermission the temperature often sinks below the normal, sometimes to 96°, 94°, 92° or even 90·6°, and it continues low for the first two or three days; the pulse also drops to 40, 50, or 60 in the minute, though liable to sudden increase on exertion; the tongue becomes clean, and the appetite often voracious. Occasionally, at the commencement of this period, the patient falls into sudden collapse, or passes into a typhoid state; but far more frequently, with the exceptions above adverted to, he appears to be restored to perfect health.

Sometimes the first paroxysm of fever is the only one. But more

commonly, at the end of fourteen days (more or less) from the first accession of symptoms, the patient suddenly experiences a recurrence of his febrile attack. The symptoms which now ensue are as nearly as possible identical with those from which he formerly suffered. The temperature, however, is often higher, and the duration of the attack for the most part shorter. It generally lasts about three days; at the end of which time, convalescence is ushered in with the phenomena which previously ushered in the remission.

Occasionally a third paroxysm takes place on or about the twenty-first day; and a fourth and even a fifth recurrence have been observed, though very rarely.

The danger to life from relapsing fever is comparatively very slight. Dr. Murchison's statistics show a mortality of only 4·75 per cent. The causes of death are mainly asthenia and collapse (the latter of which may occur quite suddenly about the period of crisis), coma and other cerebral complications, and its sequelæ.

Convalescence is generally protracted—the patient very slowly regaining strength—but seldom complicated with serious sequelæ. Amongst the most common of these are pulmonary affections (more especially pneumonia), diarrhœa, and dysentery. The most characteristic of them all is ophthalmia. It is a remarkable fact that pregnant females affected with relapsing fever almost invariably abort, and this no matter what period of gestation they may have reached. The fœtus, moreover, dies; the mother, as a rule, recovers.

Morbid anatomy.—Excepting for the presence of such lesions as are due to accidental complications and sequelæ, nothing very characteristic is noticeable after death. The liver is usually enlarged and congested, but otherwise (even if jaundice be present) apparently healthy; and the spleen is invariably enlarged to several times its normal bulk, and generally softened or diffluent.

Treatment.—In the treatment of this disease it is of course necessary, in order to prevent its spread, to isolate the sick, and to take the ordinary precautions in respect of ventilation and the like. In every case the disease will probably run its course, whatever treatment be adopted. It is important, nevertheless, to alleviate symptoms and to avert complications. To diminish heat, cold sponging or the graduated bath may be serviceable; to check vomiting, ice; to relieve headache and other pains and to promote sleep, perfect quiet, opium or morphia in medium doses, chloral, and counter-irritant or sedative applications; to obviate constipation and portal congestion, mild laxatives such as castor-oil, or enemata; and to encourage diuresis, non-stimulating diuretics, such as bland drinks, and medicines containing chlorate, nitrate, or acetate of potash, or acetate of ammonia. If coma, attended with suppression of urine, occurs, it may be necessary to give purgatives, and to apply cupping glasses over the lumbar region. Emetics are recommended by many to be given early in the disease;

and bleeding has also been strongly advocated. During the febrile attack, the nourishment should be such as is usually proper for patients suffering from febrile disorders. Alcoholic stimulants are rarely necessary, excepting when there is any tendency to collapse.

XIV. DENGUE. (*Dandy Fever.*)

Definition.—A specific affection, characterised by high fever, inflammation of the joints, a peculiar rash, and a tendency to be continued for a few weeks by intermittent attacks of short duration.

Causation and history.—Nothing seems to have been known of this disease until the year 1824, when it broke out suddenly in Rangoon among a body of troops. Thence it spread; and since that time it has occurred in occasional epidemics in different parts of India, and also in the tropical parts of North America and in the West India Islands, into which it was introduced from the East Indies. It does not appear to have extended to temperate regions. Dengue is contagious in a very high degree, and doubtless, like other such diseases, depends upon a specific virus communicated from the sick to the healthy. Its contagiousness, indeed, is almost as virulent as that of influenza; and it spares neither male nor female, young nor old.

Symptoms and progress.—Little or nothing is known with respect to the period of incubation of dengue, or to the amount of protection one attack affords. The invasion is sometimes preceded by slight premonitory symptoms, but much more frequently is quite sudden. Among the early phenomena of the disease are—high fever, with sense of chilliness or actual rigors, alternating with flushes of heat; dryness of skin; severe frontal headache with vertigo; aching in the eyeballs; pain along the spine and in the limbs, but more particularly in the joints; great rapidity and hardness of pulse; acceleration of respiration; furred tongue, and heat and pain at the epigastrium, with loss of appetite and very frequently sickness; great muscular prostration, restlessness, and inability to sleep. With the advance of the disease, the prostration and the febrile symptoms undergo aggravation; the face and the conjunctivæ become congested; the pulse rises to 120, 130, or even 140; the tongue gets coated, except at the tip, with a thick, white, moist fur; and the pains (especially those in the joints) are augmented—the arthritic pains, indeed, tending to shift about as in ordinary rheumatism, and the affected joints (especially the smaller ones) to swell. In the course of a day or two, however, perspirations break out, and the severity of the symptoms seems to abate somewhat; but on the third or fourth day of the disease, or a

little later, some increase of pain takes place, and is attended with an evanescent eruption, which, commencing on the hands and feet, quickly spreads over the whole cutaneous surface. This eruption has been likened to that of scarlet fever, measles, urticaria or erythema. From the descriptions it would seem to be a kind of erythema papulatum, such as is not unfrequently met with in cases of acute rheumatism. It is said to disappear usually on the second day, to be attended with more or less itching, and to be followed by desquamation. It is not invariably present. With the subsidence of the rash, or about the fifth, sixth, or seventh day of the disease, the febrile and other symptoms abate, the patient becomes convalescent and is then soon restored to comparative health. In a short time, however, a relapse almost as severe in its symptoms as the primary attack but lasting only for two or three days, occurs; and to this, after intervals of apparent convalescence, a second and perhaps a third relapse succeed. Usually much debility, and not unfrequently pain, stiffness or swelling of the joints, persist after the final cessation of fever; and health is generally not completely restored under a period of three months. It is important to observe that, notwithstanding the high fever, the extreme pain, and the general severity of the symptoms under which the patient labours, he rarely suffers from delirium, or fails to make a good ultimate recovery. Occasionally death occurs early in the disease —during the period of defervescence—from syncope.

Other phenomena which patients suffering from dengue occasionally present are—bleeding at the nose; swelling of the parotids with salivation; swelling of the lymphatic glands, or of the testicles; jaundice, and ophthalmia. It may be added that the appetite in some cases continues unimpaired, and that pregnant women rarely abort.

It is obvious that the phenomena of dengue have a considerable resemblance, in some aspects, to those of rheumatism, ague, scarlet fever, and measles; with each of which it has been confounded. But it much more closely resembles relapsing fever. It resembles this, in its virulence of contagion, in its sudden access, in its high temperature with headache and arthritic pains, in the rareness of the occurrence of delirium, in its tendency to be continued by several successive relapses, in its little mortality, and even in some of the details of symptoms and sequelæ—such as the condition of tongue and appetite, the occasional occurrence of jaundice, ophthalmia, and inflammation of the salivary and other glands, and even in the occasional supervention of death from syncope during the period of defervescence. The eruption of dengue (if it be specific) may seem to indicate a difference between them, as also may the intensity of the arthritic inflammation which attends it. Can it be relapsing fever modified by climate?

Of the *morbid anatomy* of dengue nothing of any importance is known.

Treatment.—The treatment must be that applicable to other fevers over whose course we have no control. Emetics and purgatives have been strongly advocated. But, on the whole, it is probably best to administer saline or other cooling medicines. The headache and arthritic pains may be relieved by local applications, or by the use of opiates; and complications may call for special treatment. During convalescence, quinine or other tonics are indicated.

XV. YELLOW FEVER.

Definition.—A spreading continued fever, of short duration, characterised especially by epigastric tenderness, vomiting, hæmatemesis, and jaundice.

Causation and history.—This disease prevails in certain tropical regions, mainly in the West India Islands, which seem to be its home, and in the neighbouring portions of the continents of North and South America. But it occasionally invades countries correspondingly situated in the Old World, and has even been introduced into the seaport towns of England, France, and other parts of Europe. It seems never to spread, however, in these latter places, excepting at times of excessive heat. A high temperature appears to be an essential condition of its prevalence. It is said, indeed, that it never spreads when the thermometer stands at less than 72° Fahr., and that even when it is epidemic in a place, it rarely if ever attacks those who live more than 2,500 feet above the level of the sea. Outbreaks of yellow fever are probably promoted by local conditions of general insalubrity; and the intensity of the disease is doubtless augmented by them. Its contagiousness is denied by many, especially American, writers. It is admitted, however, that it attaches itself to fomites, and that it may be carried by infected ships into healthy sea-port towns, and there produce local outbreaks. The evidence, therefore, in favour of its contagiousness is very much of the same nature as that in favour of the contagiousness of epidemic cholera and enteric fever. We regard it as contagious, and as the product of a specific virus given off from the bodies of the sick. There is no good reason to believe that it ever arises spontaneously. It spares neither age nor sex; but one attack confers on the sufferer immunity from other attacks.

Symptoms and progress.—The period of latency of yellow fever is said to vary between two and fifteen days. Most commonly it ranges from six to ten. At the end of this time the patient is generally attacked suddenly with acute febrile symptoms, marked by shivering, increased temperature (101° to 105°), dryness of skin, congestion of

face, redness, suffusion and aching of eyes, acceleration of pulse, thirst, anorexia, pains in limbs, and intense frontal headache; to which are soon added acute lumbar and spinal pains, slight epigastric tenderness, and vomiting of the mucous and other contents of the stomach. The tongue is generally coated with a thick creamy fur, except at the tip and edges, which are preternaturally red. After these symptoms have lasted, with some variation, for a day or two, the febrile condition and the intense frontal and rhachidian pains are apt to subside somewhat. But, for the most part, the epigastric tenderness becomes more pronounced and the vomiting more constant; and slight yellowness of the conjunctivæ may perhaps be recognised. On the third or fourth day, or later, the vomited matters, hitherto colourless or yellow, begin to contain blood—sometimes bright, more commonly in the form of suspended particles of black pigment—and they soon assume from this cause a coffee-ground character, constituting the so-called 'black vomit.' At the same time the motions are often dark or black from the presence of blood. If the patient do not at once sink, symptoms of a typhoid character are apt to supervene; the vomiting may or may not continue; the skin probably becomes more decidedly jaundiced and at the same time dusky, the teeth covered with sordes, the tongue dry and black, the pulse quick and feeble; an eruption of red spots or of petechiæ often makes its appearance on the trunk; and drowsiness, convulsions, delirium, maniacal excitement, or coma, supervene. From the second or third day the urine contains albumen, and occasionally a little blood. Later on it gets scanty, and is sometimes suppressed.

Convalescence may (according to the severity of the attack) commence from any period of the disease, is marked by the gradual subsidence of the graver symptoms, and is generally completed at the end of two or three weeks. The jaundice, however, is slow to disappear. The fifth day is often regarded as critical.

The mortality from yellow fever is very high, and death occurs at various periods in its course. In some cases the attack is so sudden and so severe that the patient dies in a state of collapse at the end of a few hours. More commonly he sinks at the end of two, three or four days, during the period of black vomit—his death then being often due to sudden collapse probably determined to some extent by gastro-intestinal hemorrhage. Death is not unfrequently thus produced at this time in patients who have seemed to be going on quite favourably, and even in those who have hitherto suffered so little from the disease that they have not been confined to bed, and have been able to follow their employments. At a later date death is due, sometimes to cerebral complications, probably referrible to uræmic poisoning, sometimes to gradually increasing exhaustion.

The symptoms which collectively are most characteristic of yellow fever are, sudden onset with high fever, frontal and lumbar pain,

epigastric tenderness, hemorrhagic vomiting, and jaundice. But any of them, and more especially the last two, may be absent. Indeed, the symptoms of the disease are generally liable to great variation. This depends in great measure on the severity of the attack, and on the relative degrees in which the several parts of the organism are affected. Mild cases of the disease often present no characteristic features whatever, and may be readily confounded with similarly mild attacks of other continued fevers.

In its sudden onset with frontal headache, lumbar pain and vomiting, yellow fever closely resembles variola, from which, however, it soon becomes differentiated. Relapsing fever, again, in its sudden development with fever, headache, pain in the back, and vomiting, followed in a day or two by jaundice, presents a marked resemblance to yellow fever; but it differs from it widely in its little fatality, in the absence of black vomit, in its sudden cessation at the end of a few days, and in the subsequent relapse. Malarial remittent fevers may also be confounded with yellow fever, but are distinguishable by many features: they are endemic and not contagious; one attack favours subsequent attacks; the febrile paroxysms intermit; there is enlargement of the spleen; and gastro-intestinal hemorrhages, if they occur, are copious and sudden. Yellow atrophy of the liver may be distinguished by its gradual commencement, without marked fever, pain or other characteristic symptoms of yellow fever; at a later period, when the skin becomes yellow, the epigastrium tender, and delirium supervenes, the diagnosis may be difficult. Lastly, it may be remarked that jaundice is not uncommonly developed in the course of various fevers and inflammations, and cannot therefore be regarded as a distinctive mark of yellow fever.

Morbid anatomy.—The principal morbid conditions observed after death from yellow fever are, as might be predicted from the symptoms, to be discovered in the liver and mucous membrane of the alimentary canal. The liver is generally pale, soft, yellowish, or clay-coloured (as it is in many other acute febrile states attended with jaundice), and somewhat enlarged. The mucous membrane of the stomach is for the most part soft and injected, and the cavity of the organ usually contains disintegrated and blackened blood. Similar congestion and similar contents may also be met with in the intestines. Peyer's patches are unaffected. The spleen is soft, but not enlarged. Hemorrhages are not uncommonly met with in the lungs and various other parts. Nothing else noteworthy has been detected.

Treatment.—Many drugs have been recommended and used in the treatment of this disease. Large doses of calomel and large doses of quinine have both been tried. But it seems probable that they have done no good, if not harm. The patient should be confined strictly to bed, and not allowed to make any exertion. He should be kept cool, in an apartment well ventilated and devoid of hangings. The secretions

of the skin and kidneys should be encouraged by diluent drinks, and the bowels kept freely open—preferably by enemata. Vomiting should be counteracted by ice, and medicinally by limewater, hydrocyanic acid, spirits of chloroform, bismuth, or other stomach-soothing drugs. Wakefulness and delirium may be treated with opiates; headache, precordial uneasiness, and lumbar pains relieved by the local application of counter-irritants, cold, or anodynes. Constant vomiting generally precludes the successful administration of food. Under any circumstances, however, this should be bland and unirritating, and given frequently and in small quantities. Nothing can be better than milk, barley-water, rice-water, or gruel. No doubt the great tendency to fall into collapse is suggestive of speedy recourse to alcoholic stimulants. Of these brandy and the effervescent wines have been most recommended. But they should be given diluted and with caution; for, however beneficial they may prove if absorbed, their local influence on an irritable and bleeding stomach can scarcely be other than injurious.

XVI. CEREBRO-SPINAL FEVER. (*Epidemic Cerebro-Spinal Meningitis.*)

Definition.—A specific contagious fever, characterised by inflammation of the membranes of the brain and cord, and the symptoms which these lesions induce, and frequently attended with petechiæ, collapse, and early death.

Causation and history.—This disease has only been distinctly recognised from the time of its epidemic prevalence in various parts of France, between the years 1837 and 1848. Since its first appearance in that country, it has broken out at various times in Italy, Algeria, Gibraltar, Portugal, Holland, Denmark, Sweden, Norway, North Germany, and Ireland. In Ireland the disease prevailed between the years 1846 and 1850, and again with considerable severity between 1865 and 1867. In Dantzic a notable epidemic occurred in the years 1864 and 1865. In the United States cerebro-spinal fever became prevalent about the same time as in France; and since then there have been frequent outbreaks in different parts of that country. It is by no means clear that there has ever been any prevalence of the disease in Great Britain. Age and sex, social condition, and ordinary sanitary circumstances appear to exert little influence over the origin and spread of cerebro-spinal fever. Nevertheless, males seem on the whole to have suffered in larger proportion than females, and soldiers in garrison, in many epidemics, more severely than other sections of the population. It appears, also, to be indisputable that the disease occurs mainly

during the winter months; and Mr. Netten Radcliffe remarks that 'it is noteworthy that the northern and southern limits of distribution in both hemispheres but slightly overlap the isothermal lines 5° and 20°.' Cerebro-spinal fever is certainly epidemic. Is it also infectious? Of this we think there can be little doubt. It is important, however, to note that the mode of its epidemic prevalence is not unlike that of cholera or typhoid fever, in the facts that it is marked by numerous scattered and for the most part small outbreaks, rather than by a general widespread diffusion; and that the disease, like these others, although giving clear indication of its spread from the sick to the healthy, presents little or nothing of the virulence of direct contagion which characterises most of the exanthemata.

Symptoms and progress.—Cerebro-spinal fever is attended in some cases by premonitory symptoms, lasting from a few hours to several days, and comprising mainly feverishness, malaise, headache, and pains in the back, abdomen, and limbs; but in many cases it comes on quite without warning. In either case the first symptoms of the actual outbreak are—severe rigors; intense headache with vertigo; persistent vomiting with more or less severe pain in the stomach; and pains along the spine and in the muscles of the extremities, often attended with spasmodic contraction. The patient soon becomes restless or irritable, voluble or taciturn, more or less obviously delirious or the subject of delusions, and not unfrequently drowsy. His head is thrown back, and retained in that position; not so much from spasm in the muscles of the neck, as from a voluntary effort to relieve pain in that situation; and his limbs become flexed. He probably cries out at times, or screams with the intensity of the pain in his head and back. But gradually his mind gets more distinctly affected; he becomes less alive to pain and other subjective phenomena; he passes into a condition of busy or muttering delirium or into one of acute maniacal excitement, occasionally has convulsions, and then lapses more or less gradually into profound coma. In many cases a more or less abundant purpuric eruption makes its appearance from the second to the fourth day. Death may occur during the first day or two (occasionally after a few hours only) from collapse; or, from this time to the seventh or eighth day, from coma due to the cerebro-spinal lesion; or at a later period, even up to the sixth or seventh week, from one or other of the complications which are apt to ensue.

The above is a sketch of the symptomatic phenomena of the disease in its ordinary form; and, as will be observed, they are mainly those of non-specific inflammation of the membranes of the cord and brain. They vary much, however, in their severity in different cases, and are frequently conjoined with other symptoms which are also for the most part dependent on the cerebro-spinal lesion. We will consider them *seriatim*, as they are referrible to different conditions and different organs. *Fever* is not usually a marked feature of the disease. The

temperature appears in many cases never to rise above 101°; but it may reach 105°; and in cases which are rapidly fatal, with symptoms of collapse, it may even sink below the normal. The *skin* varies in its condition, but is seldom pungently hot and dry, or profusely perspiring. Besides the petechial eruption which has been described, it occasionally presents patches of erythema or roseola; or groups of herpetic vesicles appear upon the lips. *Respiration* in severe cases is more or less embarrassed. It is then generally slow and suspirious, but with the increase of depression it becomes hurried and shallow. The *pulse* is much enfeebled, but its frequency is liable to great variation; sometimes it is preternaturally slow, sometimes exceedingly frequent; and rapid alternations are apt to occur without any obvious cause. The *gastro-intestinal* phenomena are of some importance. Violent sickness is a noteworthy symptom of the disease during its earlier periods. It comes on without any necessary sense of nausea, and independently of the ingestion of food. As the disease advances it usually ceases. The severe abdominal pain which commonly occurs about the same time is also an important symptom; it appears to be strictly neuralgic, and like the vomiting itself referrible to the condition of the central nervous organs. The tongue may be clean, or furred, and with the progress of the disease is apt to become dry. The bowels are for the most part constipated. The *urine* in some cases contains albumen and blood. The more important symptoms referrible to the *nervous system*—namely neuralgic pains, delirium, and coma—have already been enumerated, and we need not recur to them. We may, however, point out that numerous additional phenomena are apt to present themselves. The patient not only suffers from intense pain in the head, not necessarily limited to any one locality, but also from pain in the course of the spine and especially in its cervical region, and from neuralgic pains in the belly and in the course of the limbs. Cutaneous hyperæsthesia is sometimes present. We have pointed out that general convulsions are occasionally observed; but more common perhaps than these are local spasms either of the tonic or of the clonic kind in various groups of muscles, or tremors and subsultus. Paralysis, either hemiplegic or limited to a limb or some other portion of the organism, occasionally supervenes; or there may be anæsthesia. Deafness, loss of sight, squinting, inequality of pupils, and the like, are also occasionally met with; and sometimes, intolerance of light or sound. With the supervention of coma, and often before that period, there is loss of control over the bladder and rectum. The *attitude* which the patient assumes is characteristic, at all events of cerebro-spinal inflammation; and his *aspect* generally affords clear indications of the condition of his cerebral and spinal functions.

If the case be of long duration, various phenomena, due apparently to irritation of the nerves or of the centres whence they emerge, are apt to ensue—and amongst them, destructive inflammation of the cornea or other parts of the eye, or of the internal ear; inflammation, often

attended with suppuration, of the large joints; parotid swellings; and bed-sores. Inflammatory affections of the thoracic organs are also not unfrequent.

The percentage of deaths in cerebro-spinal fever has varied in different epidemics between 20 and 80.

Morbid anatomy.—The morbid changes observable after death are definite and simple. They consist in congestion of the vessels of the pia mater of the brain and cord, and inflammatory exudation into the subarachnoid tissue and occasionally into the ventricles. This exudation may be transparent and watery, but is more frequently opaque, greenish, and distinctly purulent. The affection is sometimes general, but more commonly localised to some extent; and not unfrequently it is confined mainly to the base of the brain—especially its posterior part—and to the surface of the medulla oblongata and upper part of the spinal cord. There is often, also, more or less congestion of the substance of the brain. It is said that in some cases in which death has occurred speedily from collapse no characteristic lesions have been detected.

Treatment.—The treatment of cerebro-spinal fever has probably not been more successful in its results than that of any other of the specific fevers. It must, however, be borne in mind that the mortality of this disease is due, less to the direct influence of the specific poison of the disease than to the cerebro-spinal inflammation which is one of the immediate consequences of its operation. If, therefore, meningeal inflammation be amenable to treatment, it is reasonable to believe that that of cerebro-spinal fever should be to some extent within our control. Powerful depletory measures, however, and above all the abstraction of blood, are on several grounds obviously contra-indicated. Counter-irritation, or cold to the head and along the spine, and moderate purgation, may possibly be of some benefit, as also may cooling saline draughts. Opium in large and frequently repeated doses, and quinine in large doses, have found much favour with American physicians. The food which is administered should be in the fluid form; and its regulated exhibition should be enforced. When symptoms of collapse manifest themselves, stimulants may be had recourse to, and the surface should be kept warm.

XVII. DIPHTHERIA. (*Membranous Croup.*)

Definition.—A contagious disease, of which the more characteristic phenomena consist in the formation of whitish membranous pellicles on certain mucous surfaces (more especially those of the fauces, nares, larynx, and trachea), and on excoriated or wounded areæ of the skin; the rapid development of anæmia and extreme debility; and the supervention, during apparent convalescence, of temporary paralysis.

Causation and history.—This disease, although it has been described by many authors of ancient and modern times, has been known by its present name only since the publication of Bretonneau's treatise in the year 1826. He designated it 'diphtherite' (since modified into diphtheria) from the Greek word διφθέρα, a skin. Diphtheria, like most other infectious diseases, is met with in the sporadic form, and from time to time breaks out into virulent and widespread epidemics. Many of these have been recorded. The last of any serious importance prevailed in France during the years 1855, 1856, and 1857, and was imported thence into our own country, where, from 1859 to 1862, it committed great ravages. It was then regarded by a large number of the most experienced physicians as a disease almost, if not quite, new to the country. They were well acquainted with membranous inflammation of the trachea, or croup—a disease, too, which had been known to occur in an epidemic form; but they failed to see, as many indeed still fail to see, that between the characteristic forms of croup, from which the classical description of the disease was taken, and diphtheria, there is no essential difference. The Scottish and English physicians of the latter part of the last century, and the early part of this, had their attention particularly directed to the rapidly fatal laryngeal form of the disease, and described it as a local malady. Bretonneau, on the other hand, recognised that the laryngeal affection was only the occasional complication of a general disease, which was infectious, and presented other remarkable features besides the mere formation of a membranous lining to the air-passages. Thus, the same disease, described from different points of view and from different degrees of acquaintance with its pathology, and receiving different names, came to be regarded as two distinct diseases. And hence as much confusion has arisen, and as much difficulty in recognising the exact truth, as in the converse case of disentangling enteric fever and typhus from the discordant descriptions of the presumed single disease, continued fever.

Diphtheria is a disease of all countries and all seasons, and affects both children and adults. It is, nevertheless, far more common among young children, especially between the ages of three and six, than in persons of more mature age, and is both actually and relatively much more fatal to them. There is reason to believe, that the sanitary state of houses or localities, and the condition of health of those who are exposed to its poison, have much influence over its development. It is not very clear, however, what forms of uncleanliness or what constitutional conditions are most influential in this respect; for we know, that those who appear to be in the best of health often take it, while the weakly often escape; and that it attacks the wealthy and the clean as well as the poor, the filthy, and the overcrowded. Diphtheria is undoubtedly contagious; the epidemic of 1859–62 was distinctly imported into this country from France; the introduction of a case into a house, hospital or other institution containing many inmates, is

almost certain to be followed by an outbreak of the disease amongst them—and indeed it not uncommonly happens that every child of a large household is thus swept away; the nurse contracts it from her charge, the doctor from his patient, the mother from her suckling. The contagion is doubtless carried by the atmosphere. But it may also lie dormant in fomites, and thus present prolonged vitality; for it is certain that many cases have been met with in which children, brought into rooms which had been well purified subsequently to the occurrence of diphtheria in them several weeks or months previously, have taken the disease. There is no doubt that it can be imparted by inoculation. Many cases are recorded (such, for example, as that of Professor Valleix, in whom a fatal attack supervened on the reception into his mouth of a small quantity of saliva coughed out by a diphtheritic child) where accidental inoculation seems to have been efficacious; yet, on the other hand, both Trousseau and Peter have inoculated themselves without effect. Experiments upon the lower animals have latterly been largely performed, but with results which are not entirely conclusive. The most important are those of Letzerich, Oertel, and Trendelenburg, in which they claim to have given diphtheria to rabbits by the introduction of diphtheritic matter into the trachea.

It may be presumed that the patient is most apt to impart the disease while the membranous exudations are present; but it is by no means certain at what period he ceases to be infectious. Convalescent children—children, that is, who appear to be perfectly well and have been apparently well for two or three weeks—seem occasionally to give the disease to others.

Symptoms and progress.—The period of incubation is not accurately known. Some patients appear to have had the first symptoms of diphtheria a few hours only after exposure to its virus. In others the disease has not manifested itself for eight days. The incubative period probably varies between these extremes. Whether it is ever longer must be regarded as doubtful. The symptoms of invasion vary in some degree in their intensity with the virulence of the attack they usher in. For the most part they consist in elevation of temperature and other evidences of febrile disturbance, together with slight uneasiness or soreness of the throat. But these are often so slight, that the patient makes little or no complaint, and pursues his ordinary avocations, until perhaps (especially if he be a child) attention is attracted to him by the presence of pallor, languor, and dulness or tendency to mope. Sometimes the febrile symptoms are much more marked, and there may be distinct chills or rigors; but there is rarely even then any great complaint as to the condition of the throat. If, on the first evidence of illness, the interior of the throat be examined, there will probably be observed some degree of redness and tumefaction of the tonsils, pillars of the fauces, soft palate or

pharynx, or of all of these parts. And very soon afterwards, whitish, greyish or buff-coloured, opaque, well-defined, patches will be visible on some parts of the congested surface—often on one or both tonsils. These vary in thickness, are more or less coherent, admitting of removal in shreds or as a whole, and are moderately adherent to the subjacent surface, which is left excoriated but not excavated by their removal. They tend rapidly to spread, and hence if multiple to coalesce, and at the same time to become thicker and more adherent; and may thus, in the course of a few days, form a nearly continuous covering to the whole surface above indicated, including that of the uvula. And, indeed, the throat may be found already in this condition, at the time when attention is first seriously attracted by the general aspect of illness which the patient presents. By this time, the tonsils are often considerably enlarged, and the uvula swollen and œdematous; there is almost invariably manifest swelling and tenderness of the lymphatic glands about the angles of the jaw; there is generally, also, more or less mucous exudation and accumulation about the fauces; but rarely, either the total loss of appetite, or the great agony in mastication and swallowing, which attends ordinary tonsillitis.

The course which the disease may take from this point is very various. In some cases, the febrile symptoms soon subside, the morbid process ceases to spread, and the patient rapidly convalesces. In some cases, the membranous formation extends along the œsophagus, reaching it may be to the stomach. In some, it spreads to the larynx and trachea, and occasionally thence to the bronchial tubes. In some, it invades the posterior nares, extending possibly throughout the whole of the nasal cavity and even along the lachrymal ducts to the conjunctivæ. In some, the inflammation spreads in depth, and the glands and other soft tissues in the submaxillary and adjacent regions get swollen and infiltrated with inflammatory matter. And in some, diphtheritic pellicles make their appearance on other mucous surfaces, or on excoriated or ulcerated parts of the skin. We will discuss these various cases categorically.

1. The first of the above varieties of diphtheria is often a very mild disorder. The patient—with little or no fever at any time, with scarcely any complaint of soreness of throat, with no material thirst or loss of appetite, and with perhaps a small white patch on one or other or both tonsils, which may even have disappeared before the throat comes to be examined, or which may be detached at the end of three or four days, or a little later—becomes convalescent in the course of a week or ten days, and then, except probably for some unusually persistent anæmia and debility, and perhaps for some enduring enlargement of the cervical glands, is soon restored to health. When, however, the membranous exudation covers an extensive surface—especially if, at the same time, the tonsils and uvula are much swollen—the symptoms are far more serious, and the duration of the malady is pro-

longed; but even then, if no complications arise, the patient is generally convalescent at the end of ten days or a fortnight. There is commonly under these circumstances great and increasing debility, and anæmia; and not unfrequently the patient, who has been perfectly sensible all along, dies from asthenia or in a fainting fit following some slight exertion. Occasionally, and more commonly in adults than children, the breath acquires a fetid and distinctly gangrenous odour—the false membrane at the same time assuming a dirty grey or blackish hue, and a more or less pultaceous consistence. These phenomena are seldom due to actual gangrene, but are generally the result of mere decomposition of the diphtherial exudation.

This is perhaps the best place to point out, that diphtheritic patches not unfrequently make their appearance on the inner surface of the cheeks and on the gums, especially in the neighbourhood of the pillars of the fauces, and sometimes at the margins of the lips; and, further, that Bretonneau has described an affection of the gums (frequently associated with distinct faucial diphtheria and evidently of the same nature) in which an abundance of rust-coloured tartar accumulates about the necks of the teeth, in association with marginal pellicular formations on the gums, and a tendency to the development of similar patches on those parts of the inner surfaces of the lips and cheeks with which the diseased gums are in contact. There is excessive fetor of breath and disposition to gingival hemorrhage.

2. Extension of the diphtheritic inflammation along the œsophagus is not very common, nor is it attended with any marked special symptoms. Both difficulty and pain in swallowing, and complete and unconquerable anorexia, are not unfrequent accompaniments of severe cases of simple diphtheria, and hence would not be characteristic of this complication, although they would probably attend it.

3. Diphtheria of the air-passages constitutes one of the most frequent, and at the same time one of the most fatal, of the varieties of the disease. In some cases, no doubt, the larynx or trachea is the primary seat of inflammation and membranous exudation, the fauces remaining healthy. Under these circumstances, croupy symptoms manifest themselves simultaneously with the first onset of febrile disturbance, and we have in fact a case of typical croup. In a much larger number of cases, however, the laryngeal mischief supervenes on ordinary pharyngeal diphtheria, the membranous inflammation extending from the one part to the other by continuity. But since in this case the preceding affection of the pharynx is often exceedingly slight, not to say trivial, and has very likely given little or no positive indication of its presence, the laryngeal sequence is very apt to be assumed to be the primary disorder; and, again, the case falls in with the classical descriptions of croup. In many cases, however, the pharyngeal affection is severe; and has been recognised, before the symptoms of croup appear. Here the sequence of events is obvious.

Thus, diphtheritic affections of the larynx and other air-passages either may be secondary to pharyngeal diphtheria, or may commence in the larynx, trachea, or possibly even bronchial tubes, and then either remain limited to these parts or spread upwards to the pharynx. Under any circumstances the symptoms resulting from the laryngeal or tracheal affection are of the same kind, and of extreme gravity. The child (for although membranous croup occurs in adults, it is mainly children who suffer) is first attacked with a frequent, short, dry, perhaps metallic, cough, and slight hoarseness of voice—symptoms in this affection of the worst omen, even if in other respects he appears, as he often does, to be fairly well. But soon, some difficulty of breathing supervenes, commencing usually in the night. The symptoms now rapidly increase in severity; breathing (inspiration more than expiration) becomes noisy, sibilant, stridulous, or metallic, especially after an attack of coughing; the voice grows hoarser and weaker, or fails; the cough gets less frequent but more severe—paroxysmal, suffocative, harsh, unmusical, and wheezy, or far less commonly hard and metallic; and during the paroxysms the child tosses itself about, sits up, clutches whatever is near it, throws its head back, opens its mouth, dilates its nostrils, and struggles for breath; the general surface and especially the face become livid, the eyes staring, and the expression one of intense anxiety. Even now, in the intervals between the paroxysms of cough, the child often assumes a fallacious appearance of ease and comfort; the breathing may be little quickened, and, unless under excitement, attended with little noise; and the best hopes of recovery may arise. But the paroxysms return and increase in frequency and severity; until at length, overcome by his exertions and progressive suffocation, the patient passes into a condition of combined coma, asphyxia, and prostration, in which he dies. Death takes place sometimes in a few hours, rarely later than the fourth or fifth day after the commencement of symptoms. In adults the course of the disease is usually not so acute. It should be added that during the progress of the attack, the respirations increase in frequency; the pulse becomes small, weak and rapid; the surface, especially that of the extremities, gets cold; and perspirations break out; and further consciousness remains for the most part unimpaired almost to the close. The symptoms above detailed are clearly referrible to the gradual growth and extension of false membrane in the larynx and trachea, and are occasionally relieved by their expulsion in the act of coughing. The paroxysmal cough is probably chiefly dependent on the occasional blocking up by mucus of the narrowed rima glottidis or trachea, and on spasm. The spread of the false membrane throughout the bronchial tubes, and the supervention of lobular pneumonia, are indicated mainly by rapid advance of lividity and asthenia, increasing imperfection of the respiratory acts, with falling in of the lower ribs and intercostal spaces during inspiration, inefficiency and feebleness of

cough, and suppression of the auscultatory phenomena of the lungs. Emphysema of the connective tissue of the neck, head, and thoracic parietes, is occasionally developed.

4. Extension of the diphtheritic process to the nose, or the deeper tissues of the neck, constitutes an essential feature of the so-called 'malignant' form of diphtheria, and indicates severe concurrent constitutional poisoning, and an almost certainly fatal issue. Malignant diphtheria often comes on with no more severe symptoms than those which attend the commoner forms of the disease; and even when local signs indicate the course the malady is taking, and the observant physician foresees and dreads the impending change, there is frequently nothing in the patient's condition to alarm himself or his friends. The spread of the disease to the nose is indicated by catarrhal symptoms, by redness and soreness of the nostrils and by the discharge of mucus, frequently attended early with some degree of epistaxis, and, ere long, with a copious flux of bloody ichor. At the same time the lachrymal ducts become involved, the escape of the lachrymal secretion by the puncta is arrested, the eyes water, and occasionally false membranes form on the conjunctivæ. On inspection of the anterior or posterior nares the existence of the false membrane in the nose will probably be clearly recognised. The extension of the inflammation in depth is shown, partly no doubt by progressive enlargement of the tonsils and thickening of the soft palate and uvula, but more especially by rapid increase in size of the lymphatic glands about the angles of the jaw, and by infiltration with inflammatory products of the connective and other tissues which intervene between them. By these processes very considerable general tumefaction is produced; extravasations of blood and suppurating cavities appear here and there in the substance of the mass; ulceration or gangrene occasionally takes place at the mucous surface; and the cutaneous aspect, either uniformly or in patches, becomes brawny and congested or livid. In malignant cases, anæmia and prostration come on with great rapidity; the pulse early becomes quick, irregular, extremely small and feeble, and the surface cold; hemorrhage frequently takes place from the mucous orifices, and petechiæ and vibices appear beneath the skin; the patient is restless and occasionally delirious; and death results from asthenia.

5. Although, in the vast majority of instances, diphtheria commences either in the pharynx or in the mucous cavities which communicate directly with it, cases are occasionally met with (especially during epidemic outbreaks and amongst the members of infected households) in which the diphtheritic inflammation and pellicular formation first make their appearance in some other region—occasionally in the vulva or vagina, on the glans penis and foreskin, at the anus, in the external auditory meatus, or on excoriated or raw cutaneous surfaces. The local changes here are identical with those occurring in the more usual seats of the disease; the redness of the affected part is

more or less vivid and intense, especially in a narrow zone circumscribing the adherent pellicle; the pellicle is white, buff, grey, or black, not unfrequently looking like an eschar, and adherent to the surface; and (when the skin is the part involved) its extension is attended with the formation of vesicles at the margins, which run together, and lead to the development of spreading excoriations which presently get clothed with the enlarging pellicle. Just as in many cases of primary pharyngeal diphtheria false membranes appear after a while on various parts of the surface of the body; so, in the cases now under consideration, it is not uncommon to find the pharyngeal mucous membrane ultimately involved.

There are two or three important points in relation to diphtheria which have been either quite passed over, or only touched upon, in the foregoing account, but must not be forgotten. The temperature of diphtheria is never a characteristic feature, and is rarely high. In some, and even severe cases, it scarcely at any time exceeds the normal; generally, however, there is distinct elevation during the first day or two, and occasionally—but more particularly in those cases in which the larynx and trachea are implicated—the temperature rises in the course of the disease to 106° or 107° and upwards. The urine in a large proportion of cases (one-half or two-thirds, according to different observers) becomes albuminous at an early date, the amount of albumen being sometimes very great. Occasionally, and more especially in malignant cases, there is hæmaturia. Under the microscope will be found, in the former case hyaline and granular casts, in the latter blood more or less modified in character. These conditions of the urine are rarely of long duration, and scarcely ever usher in dropsy, uræmia, or permanent lesion of the kidneys. Urea is excreted in excessive quantities during the progress of the disease, and diminishes during convalescence. Inflammation now and then extends from the throat to the ear, and may produce suppuration and other serious lesions in that organ; and occasionally it spreads from the conjunctiva to the cornea, causing opacity, ulceration, and perforation. Delirium is of unusual occurrence, and generally forebodes a fatal issue.

The duration of diphtheria varies widely. When the disease ends in convalescence it rarely exceeds a fortnight; and it may be as little as a week. Death occurs at very different periods, which, however, are very much determined by the nature of the lesions inducing it. It may take place within the first twenty-four hours, or as late as the end of the second week, or at any intermediate period. The causes of death have been sufficiently considered. The mortuary rate of diphtheria is high; but it is impossible to make any exact statement on this point; for while in some epidemics, undoubtedly, many mild cases occur of which a large proportion are never suspected to be diphtheria, in other epidemics the fatality of the disease is frightful. The most fatal forms of diphtheria are those in which the air-passages are

affected, especially in children, and those which have been spoken of as malignant.

Diphtheria does not always cease with apparent convalescence. In many cases, morbid phenomena of a totally different kind to any which have preceded sooner or later supervene. These are affections, for the most part paralytic, of the sensory and motor nerves. They sometimes commence with the separation of the false membrane, but more commonly come on from a week to a month after convalescence seems to have been established. Usually the first, and not unfrequently the only, part affected is the soft palate. The patient, who had probably regained his voice and power of deglutition, begins to speak with a nasal tone; when he attempts to swallow, a portion of his food is apt to pass into the posterior nares; and on examining the throat, the soft palate is found to be more or less pendulous and motionless—motionless even when mechanically irritated; its sensibility also is impaired or annulled. It is worth while pointing out, as showing that the palatal paralysis is not the result of local inflammatory changes, that it occurs in cases in which pharyngeal or faucial inflammation has been very slight, and even in cases where there has been none. The paralysis, however, does not necessarily stop here; but soon, it may be, the patient begins to complain of numbness, tingling, and loss of power, in one or both lower extremities; then probably the upper extremities are attacked in the same manner; presently, perhaps, the sensibility of the trunk diminishes and its muscles lose their force, the intercostal muscles and the diaphragm fail, and even the rectum and bladder share in the general paresis. Further, the paralytic condition, commencing in the fauces, may spread so as to involve, on the one hand, the muscles of mastication, articulation, and expression, and on the other the larynx, lungs, and heart, and generally the organs to which the vagi are distributed. In addition, complete failure of sexual power and appetite often comes on, and more or less impairment of the organs of sense. There may be loss of smell or taste, or deafness. But it is chiefly the eyes that suffer:—squinting and double vision, and loss of adjusting power by reason of paralysis of the ciliary muscle are not uncommon; and temporary amaurosis sometimes takes place. It is important to note that, although all the forms of paralysis above specified may occur, they rarely all occur in the same individual, and never all at the same time, or in the same order. The paralysis, in fact, is progressive, and often tends to get well in one part while it is extending elsewhere; and, like hysterical paralysis, it frequently shifts from one region to another. In place of paralysis, we sometimes meet with hyperæsthesia and neuralgic pains. Notwithstanding the alarm which the presence of paralysis necessarily creates, the paralytic condition is rarely fatal, and generally ends in perfect recovery in the course of two, three, or at the outside four, months.

But it is not altogether devoid of danger. When death occurs

from it, it is mostly in those cases in which the paralysis is rapidly developed and extensive, and in which the nerves arising from the medulla oblongata and floor of the fourth ventricle are especially implicated. The patient may die from inability to swallow food, or from the accidental entrance of foreign matters into the larynx, or from gradual failure of the respiratory acts and consequent apnœa or asphyxia, or from enfeeblement of the heart's action, which is attended with remarkable slowness, or rapidity or irregularity of the pulse and tendency to syncope. Occasionally death is due to convulsions or coma. In reference to diphtheritic paralysis M. Duchenne points out, that sensation and motion are usually simultaneously affected, but that the impairment of sensation tends to preponderate over that of motion. The paralysed muscles retain their electric contractility, their bulk, and their healthy texture.

In speaking of diphtherial albuminuria we remarked that it is usually one of the early phenomena of the disease. It must be added that it sometimes comes on again, or for the first time, during the paralytic stage.

Morbid anatomy and pathology.—The morbid changes which attend diphtheria are almost limited to the circumscribed inflammations which have already been discussed. In most cases the affected parts are congested, swollen, and infiltrated with leucocytes and other inflammatory matters; and, when the inflammation extends deeply, extravasations of blood and foci of suppuration, terminating in distinct abscesses, occasionally appear. The inflamed surface secretes abundant thin mucus; and soon an opaque layer forms upon it. This increases by additions to its under surface and to its edges, and is attached to the subjacent mucous membrane, partly by general adhesion, partly by prolongations into the mucous and other follicles. In the first instance it consists only in the inflammatory proliferation of the epithelial cells, which become cloudy and are apt from the shrinking of their protoplasm to assume a stellate form, the resulting interstices being probably occupied by mucus. This appears to be its permanent condition in the pharynx. But in the air-passages a fibrinous exudation takes place before long at the surface of the membrana limitans, between it and the modified epithelial layer which it displaces, and coagulating there forms a more or less distinctly laminated network of fibres which entangle leucocytes but very rarely distinct epithelial elements. Under these circumstances the superficial cellular lamina undergoes gradual disintegration and disappears, and thus the diphtheritic membrane at length becomes purely fibrinous. Many lowly vegetable organisms have, as might be supposed, been detected in it. It is not clear that any of them can be justly regarded as specific. Heuter, Oertel, and some other observers, however, maintain that the contagium of the disease consists in certain forms of bacteria, which they describe as existing in great abundance not only in the diphtheritic exudation, but

in the lymphatic spaces of the subjacent corium. The membrane varies considerably in thickness and consistency, and when very thick, its superficial parts are apt to be pulpy or flocculent. Its detachment often exposes an excoriated surface, and sometimes distinct ulceration. Occasionally gangrene occurs. We have pointed out the localities in which diphtheritic membranes are chiefly formed. It remains to say that, when they extend into the nose or larynx, they adapt themselves accurately to irregularities of surface, and form complete solid casts of such diverticula as the sacculi laryngis; and that, when they involve the bronchial tubes, they extend sometimes to their finest ramifications, forming arborescent laminated casts. It is mainly when the air-passages are invaded that collapse of lung and lobular pneumonia take place, and, in children, interlobular emphysema, going on, it may be, to general emphysema. The only other organs ordinarily presenting obvious morbid changes are the kidneys. These may be enlarged, and somewhat pale, and on microscopic examination may present granular or fatty deposits in the renal cells, with hyaline casts occupying the canals of some of the tubules. In malignant cases, besides intense local mischief, hemorrhages take place beneath the serous and mucous membranes and into the substance of the lungs, heart, kidneys, and other organs; and sometimes the muscular tissue of the heart presents granular or fatty changes. The blood has been said to be distinctly modified in character; but this is certainly not always the fact; and even in the worst cases fibrinous clots may be discovered in the cavities of the heart.

That diphtheria, like the exanthemata, is a specific disease affecting the system generally can scarcely be doubted; its symptoms and progress, and especially its paralytic sequelæ, all attest the truth of this view. There may still, however, be a doubt as to whether the primary diphtherial patch, the formation of which attends the first onset of the disease, is a localised outcome of the general disorder and analogous therefore to the rash of variola; or whether it is to be regarded as the direct result of inoculation, and analogous therefore to the inoculated variolous pustule. In what way the diphtherial poison induces paralysis is a problem which does not at present admit of solution. The lesion, however, whatever its exact nature may be, is evanescent, and seems mainly to involve the medulla oblongata and neighbouring parts.

Treatment.—The treatment of diphtheria is a subject of much interest and importance, and not the less so that great variety of opinion has prevailed even in regard to points of vital moment. One of the most remarkable features in the disease is its tendency to produce anæmia and exhaustion, and death by asthenia. Such being the case, it is scarcely necessary to say that depletory measures cannot be adopted without grave risk. Indeed it is now almost universally admitted that the general treatment should be directed to the mainte-

nance of the bodily powers. To this end, nourishment by appropriate kinds of food, and the use of such tonic medicines as the patient can bear, must be firmly enforced. The liquid or pulpy foods generally administered in acute febrile disorders are suitable here; for medicine it is fashionable to prefer the solution of perchloride of iron, and doubtless the preparation is a valuable one; but there is no reason why other preparations of iron should not be given, or for the avoidance of quinine and other vegetable tonics. By some, chlorate of potash, or this with the addition of small quantities of hydrochloric acid, is strongly advocated. For local treatment of the affected mucous membrane various agents have been proposed. Bretonneau, and Trousseau following him, strongly recommended the free application of undilute hydrochloric acid; others prefer strong solution of nitrate of silver or of bicarbonate of soda, or pure tincture of the perchloride of iron, or creosote. Again, other practitioners regard the use of strong caustics as useless, if not injurious, and prefer to wash out the throat or have it gargled with solution of chlorate of potash, alum, or the like; and undoubtedly the administration of ice in small lumps is in many cases very grateful. Remedies to the nose must be applied either in the fluid form by means of a syringe or nasal douche, or as a powder by insufflation. The larynx must be treated, either by insufflation, by 'swabbing,' or by the use of the vaporising apparatus under the guidance of the laryngoscope. Emetics, which were formerly and are still often given for their supposed specific effects on inflammations of the respiratory mucous membrane, have been regarded as remedies of the utmost importance in croup, and therefore in all cases in which the diphtherial membrane tends to pass into the larynx. They are sometimes useful, indeed, but chiefly if not entirely by the mechanical influence of the vomiting which they induce, in promoting the expulsion from the larynx and trachea of the mucus, and even of the false membrane which obstructs them. They must, therefore, be regarded mainly as local remedies. Of emetics it is best to give those that act rapidly without inducing much depression; for these reasons, large doses of ipecacuanha or of sulphate of copper are preferable to equivalent doses of antimony.

As soon as distinct implication of the mucous membrane of the larynx or trachea occurs, the question of the performance of tracheotomy will necessarily and properly present itself. The extreme fatality of croup if left to itself, the little influence which drugs exert over its progress, and the fact that death is in the great majority of cases directly due to the affection of the larynx and trachea, render in many cases the opening of the trachea our only hope. It is doubtless generally difficult to decide at what moment the operation becomes imperative. Here the physician must do what he thinks best according to his own judgment, bearing in mind, however, that it is much better to perform the operation too early than too late, and that he ought not

to be deterred from doing it by the supervention of one of those deceptive intervals of calm and tranquil breathing, which are so common even while the disease is hastening to its fatal issue. Further, it is better to operate even when life seems ebbing away, or the patient is moribund, and in the face of every discouragement, than to let him die suffocated before one's eyes without making an effort to save him. Trousseau's vast experience of this treatment of croup gives an average of one successful operation out of four; he points out, however, that tracheotomy in children under two is almost never successful. Other writers (chiefly foreign) record results at least equally encouraging.

In the treatment of convalescence, and in that of the consecutive paralysis, all efforts should be directed to improve the general health of the patient and to give him strength. With these objects, change of air, tonics (especially quinine and iron), good diet, and a fair proportion of stimulants, are most important. Other agents may be serviceable in promoting the cure of the paralysis, especially strychnia, galvanism, and friction.

Lastly, looking to the established fact that breaches of the cutaneous surface have a great aptitude to become the seat of diphtherial inflammation, it should be regarded as a fundamental rule never to employ blisters or other remedies calculated to produce sores.

XVIII. ENTERIC FEVER. (*Typhoid Fever. Abdominal Typhus.*)

Definition. — A febrile disorder, characterised by an inflammatory affection of the agminated and solitary glands of the intestines, gastro-intestinal disturbance, and a peculiar rash.

Causation and history.—Enteric fever is a disease of world-wide prevalence, occurring for the most part in an endemic form, but occasionally assuming the proportions and the behaviour of a genuine epidemic. It seems to have no special connection either with overcrowding, poverty, or ill-health, and indeed to attack the denizens of town and country, rich and poor, healthy and ailing, with singular impartiality. Sex is without influence over it; but children and young persons are much more liable to it than adults, and these than such as are of advanced age. Dr. Murchison's investigations show that more than half the total number of cases admitted into the London Fever Hospital during ten years occurred in persons between the ages of fifteen and twenty-five; more than a fourth in persons under fifteen; one-tenth in persons between twenty-five and thirty; and that from the latter age onwards the numbers rapidly diminished. Considering, however, how few children attacked with enteric fever are likely to

become hospital patients, it seems not improbable that the tendency to contract the disease is pretty nearly equal at all ages up to about twenty-five, and that from that epoch it rapidly and uniformly diminishes. Undoubted cases have been recorded at various ages between seventy and ninety. Dr. Murchison also shows, from the records of the Fever Hospital, that enteric fever prevails chiefly in October, November, September, and August, and that it is at its minimum in April, May, February, and March; and he confirms the general belief that its prevalence is augmented by excessive heat of weather, and diminished by continuous low temperature. There is reason to believe that persons newly arrived in districts in which enteric fever is endemic are more likely to take it than those who have resided there for some time.

The confusion which prevailed up to within a recent period in regard to typhus and enteric fevers rendered any exact knowledge of their causation impossible. Since, however, they have been recognised as distinct and specific diseases, much light has been thrown upon the subject. It has been proved, indeed, apparently beyond all cavil, that enteric fever is above all fevers the fever of fæcal decomposition; that it occurs only among those who are exposed to the influences of defective drains or foul and overflowing cesspools, especially when these are so situated as to pour forth their fetid gases into the interior of houses, or to contaminate by their emanations, their soakage, or their leakage, water and other articles used for food. In opposition to this view, it has been asserted that persons who work in the sewers are never attacked with enteric fever; but, even if this were the fact (which it is not), it would weigh nothing against the positive evidence on the other side, which has been furnished of late years by repeated scientific investigations into the causes and circumstances of local outbreaks of the disease all over the country. The subject of its etiology is not exhausted, however, in the above remarks. It is admitted by probably all physicians that enteric fever is not, in the usual sense of the term, contagious; that it is not conveyed from one person to another person by the touch or by the breath; and that attendants on the sick rarely if ever take the disease from them; yet it is quite certain that the immigration of a patient, suffering from enteric fever, into an uninfected locality not unfrequently leads to an outbreak there. We have pointed out that it seems not to escape with the breath, or from the skin; and, it must be added, that if it escapes with the fæces in an active form it is difficult to understand how the nurses, and other persons brought into relation with the sick, so constantly escape infection. It has been observed, however, over and over again, that the fæces, which are probably at first wholly ineffective, become, in the course of putrefaction, virulent in a high degree, and impart their infectious properties largely to the contents of cesspools and sewers, and thence to well and other waters, with which the former happen to

communicate. In many cases indeed, the source of an enteric-fever outbreak has been distinctly traced to the water of a well, into which there has been percolation from a neighbouring cesspool recently contaminated with the evacuations of a patient suffering from that fever; and occasionally also, groups of cases seem to have been distinctly referrible to body-linen and bedclothes befouled with typhoid evacuations, which have been allowed to accumulate and remain unwashed. It seems clear, therefore, that persons suffering from enteric fever discharge in their fæcal evacuations (as do cholera patients) some specific but at the time innocuous organised substance; which, after its escape from the body, and under suitable circumstances, increases and at the same time becomes virulent, diffusing itself throughout the fluid media to which it gains access, and imparting to them its specific properties. The question then arises, does the specific poison of this disease, which is certainly developed from the stools of patients suffering from it, also arise spontaneously, or rather independently of such stools? The question is by no means easy to solve. Dr. Murchison especially argues forcibly in favour of its origin independently of the disease which it generates. Dr. Budd and others argue with equal vehemence in support of the opposite hypothesis. We incline strongly to the latter view, and, in accordance with it, are disposed at present to regard the essential cause of enteric fever not as a mere inorganic or even organic result of decomposition, but (like other contagia) as an organised living particle which has special endowments and unlimited powers of multiplication; not as the product of healthy bowels or of ordinary decomposing ordure, but as a specific virus yielded by the bowels of patients suffering from enteric fever, and probably by them alone. A further question here presents itself—namely, by what route does the virus gain admission into the system? It is certain that in many cases it is received into the alimentary canal; it is thus that the disease is imparted by contaminated water, and by milk to which contaminated water has been added. It is generally believed also that it may be inhaled with the breath, and that it is thus that the effluvia of cesspools and drains act in producing the disease. On the whole, there is reason to suspect that the virus in all cases enters the system at the surface of the alimentary mucous membrane, and that the intestinal lesions are to be regarded as points of inoculation.

One attack of enteric fever is believed to confer immunity against subsequent attacks. If, however, this be so, the immunity is much less perfect than in the case of the infectious fevers generally; for many second attacks have been recorded; and, moreover, true relapses are far more common than in other allied specific disorders.

Symptoms and progress.—The mode of attack and the initiatory symptoms of enteric fever present great variety. In exceptional cases its invasion is as sudden and well-marked as that of typhus, the symptoms moreover resembling those of that disease. But much more

commonly it comes on so insidiously, with undefinable feelings of malaise, or slight feverishness, or failure of appetite and strength, or some degree of gastro-intestinal disturbance, extending over some days, that the patient is quite unable to fix the date of the commencement of his illness. During the early period of enteric fever, the patient suffers in a greater or less degree from the following symptoms:—irregular chills and flushes of heat; increased frequency of pulse, and elevation of temperature; lassitude, and aching in the limbs; thirst and loss of appetite, with morbid redness or coating of the tongue; and headache or heaviness of the head, with tendency perhaps to drowsiness by day, to wakefulness, restlessness, and dreaming at night time. Vomiting and diarrhœa, with abdominal pain, and tenderness in the cæcal region, are generally associated with the above symptoms, and, though sometimes absent, are often the very earliest and generally the most striking of the phenomena which attend the earlier period of the disease. During the first week of the fever, although the symptoms gradually increase in severity, the patient is very often not confined to his bed. At the beginning of the second week, however, unless the case be exceptional either in its mildness or in its intensity, the symptoms become more fully developed and assume a more characteristic aspect. The fever reaches its acme; the skin is generally hot and dry, but liable to break out in perspirations; the pulse still increases in frequency, as also do the respirations, and not unfrequently there is some degree of cough; the tongue may continue clean or become coated with a moist fur, but generally, whether coated or clean, tends to get dry and to present cracks, mostly transversal, upon the dorsum; the vomiting has very probably subsided, but thirst and anorexia continue, and there may be some difficulty in swallowing and speaking in consequence of soreness of the throat; the patient sleeps badly; and occasionally, but by no means in all cases, delirium comes on, especially at night time and between waking and sleeping. It is about this time, too, that the rash which is peculiar to the disease first makes its appearance. It consists in lenticular rose-coloured spots, distinctly elevated and sensible to touch, disappearing on pressure, and varying when fully formed from half a line to a line and a half in diameter. Though generally rising above the general level in the form of segments of spheres, they occasionally become vesicular in the centre and thus more or less distinctly acuminated. They are rarely numerous, and always appear in successive crops—those of each crop attaining their full development, and disappearing, in the course of two, three, or four days. Thus, spots of various ages are generally present and intermingled at one and the same time. In perhaps one-fourth of the total number of cases no spots are ever discovered; and in the remainder their number may vary from a dozen or less up to many hundreds. They are chiefly developed on the chest, abdomen, and back; but occasionally are observed on the face and extremities. At this time too the intestinal symptoms usually

become pronounced; the abdomen is more or less tumid; tenderness and pain manifest themselves more distinctly in the right iliac region, where also on pressure gurgling may be detected; and the bowels become loose—open three, four, or a dozen times a day, and discharging liquid yellow stools which have been likened, not unaptly, to peasoup. From the condition above described the patient may gradually recover. But in a large proportion of cases he passes, in the course of the second week (probably towards its close), into a typhoid condition. The elevation of temperature continues; the rash still comes out; the diarrhœa persists; the tongue becomes dry and brown and traversed by deep fissures, the lips and teeth covered with sordes, the pulse quicker and more feeble; the general prostration increases; complaints of headache and pain cease; the mind grows dull and apathetic; drowsiness and delirium (sometimes violent, sometimes busy, sometimes muttering) supervene; and bed-sores tend to form. Blood, in greater or less quantities, is now not unfrequently passed with the stools. Finally, if the case be going on unfavourably, tremors, subsultus and involuntary passage of the evacuations come on, the somnolence or delirium passes into coma, and death ensues. If, on the other hand, the case be likely to do well, convalescence commences usually in the course of the third or fourth week. The change is in general quite gradual. The fever abates, the pulse falls, the cerebral symptoms pass away, the tongue cleans, the appetite reappears, the diarrhœa ceases, and the strength returns. The progress of convalescence is, however, always slow, and the patient often does not regain his former health until after the lapse of many months. Occasionally, when convalescence seems to be fairly established, a relapse takes place, attended with the rash and all the other symptoms and phenomena which characterised the original attack. A second relapse may follow.

The foregoing account applies, for the most part, fairly well to the ordinary run of well-marked, uncomplicated cases of enteric fever. No disease, however, is attended with greater variety of symptoms, or presents more frequent and greater departures from the typical character. It is desirable, therefore, to discuss briefly the various phenomena of the disease, and its varieties.

The pulse varies greatly in frequency. Occasionally, in very mild cases, it scarcely exceeds the normal throughout the whole course of the illness. In other cases, however, it mounts (in dependence very much on the severity of the case) to 90 or 100, and from this to 120, 140, or more, and becomes very feeble. It is generally quicker in the evening than in the morning, and in the typhoid stage than in the earlier period. Other things being equal, rapidity of pulse implies severity of attack. It is curious, however, that even during the presence of marked fever the pulse may at times sink below 50 or 60. In one of Dr. Murchison's cases it fell to 37.

The respirations are generally more or less accelerated, especially

with the advance of the fever, and not unfrequently some little cough is present. These symptoms are necessarily greatly aggravated when (as not unfrequently happens) bronchitis or pneumonia becomes developed. Then also the surface is apt to get dusky, and the local signs of the complication manifest themselves.

The character of the tongue varies. In some cases this organ remains almost normal throughout the illness, or is merely a little redder and drier than natural, or presents the slightest possible increase of epithelium only. More commonly it is covered, except at the margins, with a whitey-brown fur which tends to become dry, or it has a dry, glazed, morbidly red character, and in either case is apt to present transverse cracks which are often of considerable depth. The throat is not unfrequently congested and sore; and there may even be inflammation of the tonsils at an early period. Sickness is one of the most common of the initiatory symptoms, and is sometimes exceedingly severe. It may even last throughout the whole illness. Thirst and loss of appetite are almost invariably present. Diarrhœa is seldom absent, and is often very severe. Not unfrequently it prevails from the beginning; but in many cases it does not come on till the second week, or even later; and sometimes there is constipation throughout, or the patient has an occasional loose stool only. The motions usually have the appearance and consistence of peasoup, are alkaline, and often offensive; in the course of the second, third, or fourth week they may contain blood. The progress of the fever is generally attended with some abdominal pain, tenderness and gurgling in the right iliac fossa, and more or less flatulent distension of the belly.

In the early part of the disease the urine is scanty, dark-coloured, and of high specific gravity; later on it becomes pale and copious, and its specific gravity falls. There is almost always a large increase in the amount of urea and uric acid, especially at the commencement; and the chlorides are diminished. Albumen is not present in more than one-third of the total number of cases, and occurs for the most part in very small quantity and seldom before the third week.

The skin, though for the most part dry, is apt to become moist, especially in the morning; and during the latter part of the second, or in the third week profuse perspirations may occur. The cheeks, especially after meals or during the febrile exacerbations, are often flushed. The rash, which has already been described, continues by successive outbreaks for one, two, or three weeks. During convalescence perspirations are often very copious, and sudamina generally appear on the chest.

The fever, as indicated both by the thermometer and by symptoms, is always of a remittent character, presenting morning falls and evening exacerbations. The temperature begins to rise about noon and attains its maximum between 7 P.M. and midnight. After midnight

it gradually falls, the lowest point being usually attained between 6 and 8 A.M. In uncomplicated cases these daily alternations are almost constant, the difference between the morning and evening temperature varying from one to two or three degrees, or even more. The rise begins from the first day of illness, and gradually increases by daily waves until, on the fourth or fifth day or about the end of the first week, it attains its greatest elevation, which varies in different cases between 104° and 106°. From this period up to about the twelfth day there is but little change. Then, if the case be mild, the morning falls become lower and of longer duration, to be followed shortly by a corresponding decline in the evening rises; and gradually, as convalescence becomes established, the morning and evening temperatures approximate until they attain their normal level, or even sink below it. If, on the other hand, the case be severe and the commencement of convalescence be delayed, the temperature still continues high, and the morning remissions often become less marked than they had been. Again, if in the course of the disease serious complications arise, the usual course of the thermal variations is modified. Profuse diarrhœa, epistaxis, or intestinal hemorrhage causes the temperature to fall; as also does the condition of collapse, however produced. Pneumonia causes the temperature to rise, and modifies its diurnal variations. Sometimes it rises before death to 108° or even to 110·3° independently of complications (Wunderlich).

As regards the organs of sense: singing in the ears and deafness are not uncommon; the conjunctivæ are seldom congested; the pupils are usually dilated; epistaxis is of frequent occurrence. Most patients complain, at the beginning of the disease, of giddiness and headache, and of more or less pain and sense of lassitude in the limbs. There is often wakefulness at night; sometimes, on the other hand, there is somnolence, and this not unfrequently precedes delirium. Delirium is a variable symptom; in many cases it never occurs; in many it is slight, and shows itself only between waking and sleeping; in severe cases it usually comes on about the middle or end of the second week, and is then apt to vary in character and duration. It may present all the characters of the delirium of typhus; but, as Dr. Murchison remarks, it is more frequently of the violent and noisy kind than in that disease. In rare cases the invasion of the fever is attended with maniacal excitement. Coma occasionally supervenes before death. Convulsions are not usual; but are more common in children than adults; they generally come on late, and frequently prove fatal. Muscular weakness is always present, but is not so marked as in typhus; nevertheless, in the later stages of severe cases, tremors and subsultus are common. Occasionally there is muscular rigidity.

Enteric fever presents itself in many forms, and has been and still is frequently confounded with other diseases. It is especially important to know, that, for the most part, cases of so-called 'infantile remittent

fever,' 'worm fever,' 'gastric fever,' and 'bilious fever,' are cases of this affection. In the mildest form of the disease the patient perhaps complains only of slight feverishness and weakness, with loss of appetite, and more or less diarrhœa or irregularity of the bowels, and probably goes about his ordinary avocations, or at all events does not take to his bed, and, if no complication supervenes, recovers at the end of three or four weeks. In other cases the disease is much more severe in character, and its progress is more or less distinctly in accordance with the account we have already given; the attack is one of well-marked enteric fever, but varies according to the relative prominence of certain of the symptoms, such, for example, as vomiting, diarrhœa, thoracic symptoms, hemorrhage, and delirium. In other cases, again, the attack is from the beginning of exceptional severity, and, as in analogous cases of scarlet fever and other like affections, the patient dies, poisoned apparently and in a state of collapse, within the first week, sometimes on the first or second day.

Much of the danger which attends enteric fever depends on the complications which arise in its progress. The most important of these are intestinal hemorrhage, perforation of the bowels with peritonitis, and pneumonia or bronchitis.

It has already been pointed out that intestinal hemorrhage is not unfrequent. It may occur at almost any period of the disease, but is most common from the middle or end of the second week to the end of the fourth. It may be due, in cases where there is a general hemorrhagic tendency, to oozing from the mucous membrane; but far more commonly it takes place from the surfaces or edges of the intestinal ulcers. It has no necessary connection with the extent or size of the ulcers, or with the presence or absence of diarrhœa, or indeed with the mildness or severity of the patient's previous symptoms. The hemorrhage may be scanty, or so copious as to cause speedy death by syncope; and the blood which escapes may be fluid or clotted, black or of the normal colour of blood.

Peritonitis is one of the most frequent causes of death in enteric fever, and, like intestinal hemorrhage, has no necessary dependence on either the severity of the case or the urgency of diarrhœa. In the vast majority of cases it is due to perforation of the bowel in the floor of one of the intestinal ulcers, and is therefore sudden and unexpected in its onset. Not unfrequently perforation occurs in patients who have never taken to their beds; who are then seized, without warning, with intense abdominal pain, tenderness and distension, together with vomiting, collapse, thoracic respiration, and other symptoms of acute peritonitis. In such cases the nature of the complication is manifest. When, however, it takes place in patients who are already in a typhoid condition, the indications are very apt to be overlooked. Yet, even in these cases, there may be more or less evident abdominal pain and other local signs of peritoneal inflammation; but very often the dia-

gnosis must be made to rest mainly on the sudden supervention of collapse, with first a fall and subsequently a rise of temperature, increased rapidity and feebleness of pulse, hurried and thoracic respiration, duskiness of surface, copious perspirations and flatulent distension of the abdomen or tympanites. Indeed it may be said generally that the sudden occurrence in the course of enteric fever of symptoms of intense collapse, even when no direct evidence of abdominal inflammation is present, points to perforation. Perforation of the bowel may occur in patients of all ages, but is more common in males than in females. It cannot take place until ulceration has commenced, and, as might be supposed, is more common when ulceration is advanced than when it is beginning. Hence, although it occasionally happens during the second week (more especially towards its close), it is much more common during the third, fourth, and fifth weeks; and, indeed, all risk has not ceased until the expiration of two or three months. It may arise, therefore, during the period of convalescence; and even after apparently complete restoration of health. Death almost invariably follows this lesion; and generally occurs within a couple of days, sometimes in the course of a few hours. But occasionally life is prolonged for a week or two; in which case the peritonitis becomes circumscribed and an abscess forms. A few cases of recovery after the evacuation of such an abscess have been recorded. Dr. Murchison calculates that no less than one-fifth of the total number of deaths from enteric fever are due to perforation of the bowels.

Bronchitis is often present in a slight degree; but occasionally it gets severe, and may be so at any stage of the fever. The symptoms of bronchitis are then added to those of the primary disease and mask them. So pneumonia, mainly lobular, may creep on insidiously at any time, but most commonly appears during the third or fourth week. It is usually connected with the hypostatic congestion of the lungs which is generally present in a greater or less degree; and hence occupies mainly the back and basal portions of one or both lungs, and may fail to be detected unless the attention of the physician be specially attracted by the presence of symptoms indicating thoracic mischief. Pleurisy also ending in empyema is not unfrequent.

Many complications and sequelæ are described besides the above; but they are, for the most part, unimportant or rare. We will enumerate a few of the more important. *Ulceration of the larynx or trachea* is described by various good observers, but is certainly not common. *Thrombosis of the veins*, leading to œdema, frequently arises, especially in connection with the lower extremities. *Bed-sores* are very apt to form on the sacrum and other parts which are exposed to pressure or irritation; but, independently of such causes, gangrene occasionally attacks the mouth (noma), ears, penis, vulva, feet, corneæ, and especially parts to which blisters have been applied, or which are already inflamed from other causes. *Imbecility, mania*, and other mental disorders

occasionally follow on enteric fever, as they are apt to do on most affections attended with extreme exhaustion. So also does prolonged marasmus, or the development of tuberculosis. Pregnant women not unfrequently abort. But neither pregnancy nor parturition appears materially to interfere with the prospect of recovery.

There is probably no other disease in which death threatens from so many quarters, and in which it may occur at such diverse and unexpected times. It is due immediately either to asthenia, asphyxia, or coma, or to combinations of these. It may happen early in the disease, mainly from the intensity of the attack; in which case there is generally more or less pulmonary congestion. But it more commonly occurs later, either from pneumonia or other pulmonary complication, from perforation and peritonitis, from intestinal hemorrhage, or from coma coming on in the course of typhoid symptoms. Again, it may ensue, during the period of convalescence, from one or other of the sequelæ of the disease, or from sheer exhaustion. Enteric fever in hospital practice is fatal in about the same proportion as typhus—at the rate, namely, of about 15 or 16 per cent. But when we consider how large a number of mild cases occur, which are not only never admitted into hospital, but are not even recognised, it becomes obvious that the proportion of total deaths to total attacks must be much smaller than the above figures imply. The percentage mortality varies little with age; but, on the whole, the statistics of the London Fever Hospital show, that the death-rate is less below the age of 20 than in the later periods of life, and that it is highest in patients above 50.

It is not generally difficult to distinguish between a case of enteric fever and one of typhus. The main clinical distinctions are furnished: first, by the invasion, which is generally sudden in typhus, insidious in typhoid; second, by the rash, which is abundant, general, and of nearly simultaneous origin in typhus, scanty and coming out in successive crops in typhoid; third, by the abdominal symptoms, which in typhus are usually vague, but in typhoid comprise the discharge of liquid yellow stools, intestinal hemorrhage, pain and tenderness in the cæcal region, and tympanites; fourth, by the temperature, which does not in typhus present the gradual rise with regular diurnal variations which are so characteristic of typhoid; and, fifth, by the mode of convalescence, which is by crisis and rapid in typhus, but slow and followed by long-continued debility in typhoid. Many other distinctions of secondary value might be adduced. But it must not be forgotten that all may fail us, and that the discovery of the typical intestinal lesions after death may alone reveal the nature of the case which has been under treatment.

Morbid anatomy.—Enteric fever is always attended with characteristic anatomical lesions, affecting the solitary and agminated glands

of the bowels and the mesenteric glands in direct relation with them. These lesions consist in an apparently simple hyperplasia of the glandular elements, in virtue of which the organs undergo rapid enlargement, and then either slowly subside, reverting to their normal condition, or undergo softening, suppuration, ulceration or gangrene. Under the microscope the lymphatic corpuscles are found to be increased in number; and frequently hypertrophied or giant cells, containing groups of small corpuscles in their interior, may be discovered among them; later on the cells get granular and fatty, and break down into a granular detritus. The morbid process appears to begin with the first symptoms of the patient's illness; at all events, it has been found well advanced in those who have died during the first few days.

The intestinal lesions are in many cases limited almost entirely to the agminated glands, of which sometimes two or three only, sometimes all are involved. These gradually swell until they form oval plates from a line to $\frac{1}{3}$ inch thick, which present a more or less tumid margin, a reticulated or foveated but oftener more or less mammillated and smooth surface, and a consistence which is sometimes softer but more often denser, though more friable than natural. They generally attain their full development by the ninth or tenth day—sometimes a day or two earlier, sometimes a day or two later. And then they either undergo slow resolution or proceed to ulceration. The latter process may commence from the surface at numerous points, and thence gradually invade and destroy the whole of the diseased mass; or, as more frequently happens, the patch sloughs at once in the greater part or the whole of its extent. The resulting slough, which probably from bile-staining soon assumes a yellow or brown hue, becomes soft, spongy, and tumid, and separated by a line of demarcation from the still living tissues, and after a short time comes away either in mass or in successive fragments. The separation of the slough generally occurs between the fourteenth and twenty-first day, but may not be fully completed for another week. The resulting ulcer varies in character. Usually its form is oval, or round; its margin thick and vertical, as if made by a punch, and more or less congested; its floor pretty smooth and formed of the submucous tissue. Sometimes, however, the edge becomes more or less extensively undermined, and then perhaps intensely congested, and the floor irregularly excavated and flocculent, and formed partly of the exposed muscular coat, partly, it may be, of the peritoneal membrane only. Cicatrization does not usually begin before the end of the third week, and probably, as a rule, is completed in about a couple of weeks more. But the process may be delayed, either from mere sluggishness, or in consequence of a kind of phagedænic extension of the ulcer, or by other circumstances, and hence may not be accomplished under two or three months. The cicatrices rarely if ever lead to serious contraction.

The typhoid process as it affects the solitary glands is precisely similar, excepting that, the resulting tumours are much more numerous and much smaller—generally about the size of half a pea; and that, on the one hand resolution without ulceration is more common, and on the other the ulcers which form are of insignificant dimensions and tend to heal more rapidly.

The morbid process, whether it affect only the agminated glands or involve the solitary glands as well, is always most extensive and advanced in the ileum immediately above the ileo-cæcal orifice; whence in both of these respects it gradually diminishes upwards. The solitary glands are rarely affected to a greater distance than two or three feet above the valve; Peyer's patches rarely above the lower half of the ileum. The disease implicates the solitary glands of the large intestine in about one third of the fatal cases, and is always most advanced in the cæcum, rarely extending below the ascending colon. Perforation takes place only in those ulcers which have already destroyed the muscular wall. But when the floor is thus formed of peritoneum only, it sometimes happens that local peritonitis occurs and causes adhesion between the affected portion of bowel and some neighbouring organ, and thus averts the impending catastrophe. The actual perforation may be due to the forcible separation of such adhesions; but more commonly, probably, it is the result of the simple accidental laceration of the softened and unsupported serous covering. It occurs in the great majority of cases in the lower two feet of the ileum; but it has been met with at least six feet above the ileo-cæcal valve, and more rarely in the cæcal appendage or in the colon. The peritonitis which results is in the first instance always general; but not unfrequently when the rupture is small and but little fæcal matter has escaped, this latter and the suppuration which it necessarily excites are found after death to be strictly confined by adhesions to a very limited space. It is this tendency to limitation which gives an element of hope in the treatment of these cases, and to which the very few recorded recoveries after perforation are due. Sometimes the laceration is so extensive that large quantities of fæcal matter are discharged at once into the peritoneal cavity.

The mesenteric glands, especially those connected with the lower part of the ileum, enlarge from the beginning with Peyer's patches; they sometimes attain the size of a walnut, become soft and vascular, and at the end of either ten days or a fortnight undergo resolution, or soften, or suppurate. Under the latter circumstances they not unfrequently dry up eventually; sometimes, however, they induce peritonitis either by extension of inflammation, or by rupture into the serous cavity. Most other lesions in enteric fever, such as bronchitis, pneumonia, and pleurisy, have no specific characters, and need no description. The spleen, however, is enlarged and congested; and it may be

added that when the patient dies during the ulcerative stage of the fever, the contents of the bowels are generally peasoup-like, and the large intestines inflated with gas.

Dr. Klein's[1] enquiries show that the smaller typhoid growths do not originate exclusively in solitary glands, but that they often arise in the lymphoid tissue of the mucous membrane. He also shows that the typhoid process whether taking place in the intestines or mesenteric glands is attended with hyperæmia of vessels, increased development of lymphatic cells, and the development from these of giant cells, not unlike those of tubercle, and the rarefaction of the fibrous matrix. Further, he calls attention to the presence of a microscopical fungus in connection with the specific intestinal lesions. This is characterised by a distinct mycelial growth, by greenish spherical bodies two or three times as large as blood-corpuscles, and by micrococci or spores of extreme minuteness which occur singly or in couples, or in strings, or in irregular clusters. The fungus exists on the surface of the mucous membrane and within the tubular glands, but it pervades the epithelium and is especially abundant in the lymphatic spaces and channels, and in the small veins. Similar bodies are discoverable in the diseased mesenteric glands.

Treatment.—Knowing as we now do the source whence the contagium of enteric fever enters the system, it becomes our duty, nor is it difficult, to adopt suitable precautionary measures both against the contamination of water and atmospheric air, and against the exposure of persons to the influence of media thus contaminated. Whenever typhoid patients are under treatment their evacuations should be disinfected with carbolic acid, Condy's fluid, or chloride of lime before they are emptied into the sewer or the cesspool; and all articles of dress soiled by such evacuations should be similarly disinfected and washed. Water-closets and drains should be kept sound, clean, well flushed, and well ventilated, and all communications between drains and the interior of the house cut off by efficient traps. No water should be used for drinking or culinary purposes which has been exposed to sewage-contamination; hence the water of superficial wells, especially if these be near cesspools or sewers, should be looked upon with grave suspicion, as also should the water derived from streams or ponds receiving drainage, and that from cisterns or butts communicating by waste-pipes with closet-drains. If such waters must be drunk, they should first be boiled and filtered. It must not be forgotten that milk, from the presence of water which has been fraudulently or otherwise added to it, has on several occasions been the vehicle for the communication of the disease.

Many remedies have been employed for the cure of enteric fever; amongst others mineral acids, antiseptics—such as chlorine, hypo-

[1] Report of Medical Officer of the Privy Council, New Series, No. vi., pp. 80 *et seq.*

sulphites, carbolic acid and creosote—and emetics; other remedies, again, have been used with the special object of reducing the fever—such are quinine in large doses (10 or 15 grains), salicylate of soda, actual refrigeration, and bleeding. The last practice has properly fallen into desuetude. The use of cold is often beneficial, especially in cases in which the temperature reaches or exceeds 104°; it is best applied by means of baths, the temperature of which to begin with should be 10 degrees or more below that of the body, and then gradually reduced to about 68°, immersion being continued for about half-an-hour, or until the patient's temperature, as ascertained in the mouth or rectum, has become sensibly reduced, or shivering comes on; but cold or tepid sponging is also serviceable. Our chief aims, however, in the treatment of this disease must be to guard against and prevent the many sources of danger which attend it, and to relieve symptoms as they arise. The condition of the bowels must be carefully watched, and under no circumstances must drastic purgatives be employed. There is no harm, perhaps, in giving a mild laxative, such as castor oil in small doses or rhubarb, during the first week of the disease and before ulceration has taken place; but even then it is generally sufficient, and on the whole certainly more safe, to employ enemata. Subsequently, enemata only should be resorted to. When diarrhœa is present it should be restrained either by tannic acid, lead and opium, sulphuric acid, the compound kino powder, or some such remedy, or by opium or morphia suppositories, or opiate enemata. Trousseau, Dr. George Johnson, and others think that the diarrhœa should not be restrained, regarding it as a curative effort of nature; that view, however, is not generally accepted, and is, we think, erroneous and dangerous. When hemorrhage from the bowels takes place measures should be adopted to arrest it. Dr. Murchison has great faith in the use, under such circumstances, of turpentine, tannic acid, ergot of rye, or other forms of astringents. Hemorrhage occurring, however, during the first ten or twelve days is of little importance, and does not usually call for treatment. For the prevention of perforation, the avoidance of purgatives, the arrest of diarrhœa, and the maintenance of a quiescent condition of the bowels, are of extreme importance; it is further necessary to prevent the patient from using muscular exertion, and from taking articles of food likely to upset the bowels. If signs of perforation manifest themselves, our only hope lies in keeping the patient under the influence of opium or morphia—the dose and frequency of its administration being determined partly by the patient's age, but chiefly by its effects. Tympanites may be benefited by the use of stimulating enemata or hot fomentations to the belly. Sickness may be relieved by the use of lime-water and milk, bismuth or ice, or by counter-irritation. Pulmonary complications should be guarded against by the maintenance of an equable temperature, and by the avoidance of draughts. When present they

must be treated on general principles. The great tendency there is to the formation of bed-sores makes it very important, to keep the patient scrupulously clean and dry, to take measures to obviate or relieve pressure, and, if precursory redness makes its appearance, to anoint the part with some stimulating and protective application. The diet should consist of fluid and easily digestible food given frequently (every hour or two), and in small quantities. The best aliments are milk, gruel, barley-water, rice-water, and such like; but arrowroot, sago, chicken-broth, beef-tea, and eggs are valuable. Stimulants are necessary when there is tendency to collapse, when typhoid symptoms are present, or when there is great debility. In many cases, however, though their administration in moderate quantities can do no harm, they are by no means absolutely needed at any period of the disease.

Much care is necessary during convalescence. The great debility which endures so long demands the use of tonics, and an abundance of nutritious food. But the liability to perforation of the bowel (which may not cease until the end of two or three months) makes it specially important that the food should be easily digestible, and not of such a character as to derange the action of the bowels. Moreover, the liability to the supervention of pulmonary inflammation and of tuberculosis renders exposure and fatigue particularly liable to be injurious. Change of air is often extremely beneficial.

XIX. EPIDEMIC CHOLERA. (*Asiatic or Malignant Cholera.*)

Definition.—An epidemic disease, of which the attacks are very severe and rapidly fatal, characterised by copious discharge of watery fluid from the alimentary canal, suppression of the urine and other secretions, shrinking of the tissues, cramps, and extreme prostration.

Causation and history.—Epidemic cholera has been known in India for centuries, and probably from time immemorial. It is seldom entirely absent there, but at irregular intervals breaks out into wide-spread epidemics. The first Indian outbreak which specially interests us is that which, originating in the Delta of the Ganges in the year 1817, soon ravaged the greater part of Hindostan, and during the next ten or twelve years spread over nearly the whole of Asia, including the Burmese empire, China, Tartary, and Persia. In 1829 it commenced its progress through Tartary and Persia into Europe, and in that year it reached Orenburg. It then became temporarily arrested; but subsequently took a fresh start, and still travelling slowly westwards it appeared in the spring of 1831 in European Russia and Poland; and in October invaded Hamburg, Berlin, and Vienna. In

the same month cases were imported into Sunderland, and the disease remained endemic in this country for fourteen months. Having thus reached the north-western angle of Europe, the epidemic divided into two branches, one of which crossed the Atlantic and appeared in Quebec in 1832, thence diffusing itself over the North American continent; the other turned southwards, attacking successively France, Spain, Italy, and the Northern Coast of Africa. The disease did not finally leave Europe until the year 1837. Since the epidemic of 1817, numerous other epidemics have occurred in India, and several times the disease has slowly spread thence to Europe and to this country—not, however, always taking the same route as on the first occasion.

The first British epidemic was that, above referred to, of 1831–32, the second occurred in 1848–49, the third in 1853–54, and the last in 1865–66. On each of these occasions the disease was distinctly imported into this country by passengers or sailors coming direct from infected places, and its general prevalence was always preceded by local outbreaks in the seaport towns to which such infected visitors were admitted. The general history of these epidemics, so far at least as relates to England, has been that isolated outbreaks occurred in the autumn of the first year, that the disease died out with the approach of winter, and reappeared with extreme virulence in the later spring, summer, or early autumn of the second year, lasting for some two or three months, and then disappearing altogether. It might appear from this that its prevalence was largely determined by season; and, indeed, there is strong evidence to show that high temperature is on the whole favourable, and cold inimical to its spread. Yet, on the other hand, the disease has prevailed with the greatest severity in Moscow, Sweden, and other northern countries in the depth of winter.

To what cause or causes is epidemic cholera due? This is a question which has been the subject of innumerable discussions and investigations during the last fifty years. The horror which the disease occasions, the slowness yet certainty of its onward march, its sudden and capricious outbreaks, and its equally capricious subsidence and then total disappearance, all conspire to invest it with an atmosphere of mystery. Like influenza, it is the very type of an epidemic disease; and therefore, like epidemic diseases generally, has been largely held to be due to some atmospheric or telluric condition, some peculiar 'epidemic constitution' which, diffusing itself from country to country, gives to the prevailing maladies a choleraic character, and produces where local circumstances are favourable an outbreak of the fully-developed disease. There is much to be said, no doubt, in favour of this view; but the questions then naturally arise—'on what does this epidemic constitution depend?' and 'what are the local conditions which favour its operation?' These questions are not easy to answer. We may point out, however, as bearing on them,—that, although heat and climate have (as has been stated) some influence over the propa-

gation of the disease, there is no good reason to believe that moisture or drought, or excess or deficiency of electricity or ozone, affects it either one way or the other; that, according to Pettenkofer, localised outbreaks of cholera are determined in great measure by peculiarities of soil—the ground must be porous and a superficial layer of it unoccupied by 'ground water' and penetrable by air; that, as shown by numerous observations, the disease is much more apt to prevail in low-lying districts than in those which are much elevated above the sea; and that vegetable fungi, which have been detected by numerous observers in cholera-stools, have often been assumed to pervade the atmosphere and to be the specific cause of cholera. These latter have been specially investigated by Hallier, who recognises in the stools and vomit a form of urocystis consisting, partly of membranous spore-cases containing yellowish or brownish spores, and partly of cells of extreme minuteness which he believes to have been developed within these spores. These fungi he has cultivated in various ways; and he believes that he has obtained from them forms of penicilium, mucor, and the like, all of which he regards as polymorphous conditions of one and the same fungus. It must be added, however, that this particular form of fungus has certainly not been recognised by most others who have been engaged in similar investigations. Lastly, in relation to the subject now under discussion, it may be pointed out that cholera has often been attributed to the accidental or designed poisoning of springs, and to the use of diseased cereals, especially rice, and even of unripe fruit.

Again, in favour of the dependence of cholera on some miasm or epidemic constitution was the striking fact that, although cholera affected large numbers of persons within a short time, there was little evidence of its communicability by direct contagion. It was noticed, and has been constantly observed, that nurses and medical attendants seldom, if ever, take the disease from patients under their charge, and that the introduction of cholera patients into a general hospital is by no means necessarily followed by the spread of the disease to other patients.

Nevertheless, it has always happened that the spread of cholera epidemics has followed lines of traffic, showing that human intercourse, not winds, has been instrumental in their propagation. In every invasion of this country, the disease has first been distinctly imported into our seaport towns by the arrival thither of infected persons from infected localities; and has thence been carried by like means to other localities in direct relation with them by railways or other lines of traffic, and has thus gradually become distributed throughout the country, not generally, but by local outbreaks. The fact that cholera though obviously not directly contagious, or at all events not directly contagious in a high degree, yet had some mysterious relation with the movements of mankind, and never broke out

in any isolated country or town without having been distinctly imported into it by human agency, was manifestly opposed to most of the theories of its causation which have been previously referred to and had generally prevailed. Dr. Snow, now some years since, first shrewdly suspected that the cholera contagium was contained in the cholera evacuations, and that the disease was propagated by the entrance of minute quantities of such evacuations, for the most part through the medium of contaminated water, into the alimentary canal. And numerous subsequent investigations, some of the most remarkable being conducted by himself, have entirely confirmed the correctness of his prevision. The matter is so important that we may quote a few of the best established and most striking cases.

The cholera epidemic of 1849 was specially severe in the south of London, which was supplied with drinking water mainly from surface wells and by two water companies, the Southwark and Vauxhall and the Lambeth, which derived their water from the Thames—the one in the neighbourhood of Hungerford Bridge, the other in that of Battersea Fields—and supplied it in a very imperfectly filtered condition. At that time all the sewers of London discharged themselves into the Thames, the water of which was consequently very foul. The cholera epidemic of 1854 also was very severe in South London. But between 1849 and 1854 the Lambeth Company had removed its intake from Hungerford Bridge to Thames Ditton, and consequently furnished an infinitely purer water than it had done in 1849; the other company continued to draw its water from the neighbourhood of Battersea Fields. At this time the two companies were acting in rivalry, so that in many streets their mains ran side by side, and houses, under the same sanitary conditions in other respects, received a different water-supply. A careful investigation of the distribution of cholera in South London in this year, conducted mainly by Dr. Snow but with the assistance of the Registrar-General, gave the following results:—

	Population in 1851	Cholera Deaths in 14 weeks.	Cholera Deaths per 10,000
Houses supplied by Southwark Co. . .	266,516	4,093	153
„ „ „ Lambeth Co. . .	173,748	461	26

The facts were even more remarkable when examined in detail; inasmuch as in streets and localities which both companies supplied the disease singled out the houses furnished by the Southwark Company.

During the same epidemic a remarkable outbreak occurred within a limited area, in the neighbourhood of Golden Square, London, the facts of which were also examined into by Dr. Snow. There had been a few cases in the neighbourhood during the month of August, including altogether up to the 30th nine deaths. On the 30th at least eight cases which ultimately proved fatal occurred; on the 31st, fifty-six; on September 1, one hundred and forty-three; on the 2nd, one

hundred and sixteen; on the 3rd, fifty-four; and then daily until the 9th, forty-six, thirty-six, twenty, twenty-eight, twelve, eleven; after which the disease rapidly disappeared. No less than six hundred and sixteen persons were ascertained to have been fatally attacked with cholera within this area between August 19 and September 30, of whom at least four hundred and fifteen contracted the disease between August 31 and September 4 inclusive. It would take much more space than is at our disposal to enter fully into details; suffice it to say that Dr. Snow's investigations proved beyond the shadow of a doubt that this sudden and evanescent outbreak was distinctly due to the use of the sewage-contaminated water of the Broad Street pump occupying the centre of the affected area, the water of which was held in great repute, and was largely drunk by those who lived in its neighbourhood.

Again, the epidemic of 1866 was remarkable in the fact that it was almost limited to a circumscribed area in the East of London, including Bethnal Green, Whitechapel, St. George's, Stepney, Mile End and Poplar, together with the suburban districts of Stratford and West Ham. The enquiries of Mr. Radcliffe, conducted under the direction of the Medical Officer of the Privy Council, demonstrated with almost mathematical precision that the localisation of the epidemic was almost entirely due to the distribution to these districts of impure and unfiltered water by the East London Water Company.

It must be assumed therefore as a fact that the choleraic poison, at all events in a large number of cases, is conveyed through the medium of foul drinking-water, and necessarily, therefore, by means of all articles of food or drink to which such water is added. But it still remains to ask—'how does the poison reach the water, whence does it come, and what is it?' It would naturally be supposed that the choleraic poison is contained within the cholera stools; and indeed there is plenty of evidence to show that the drinking of water directly contaminated with small quantities of rice-water evacuations has induced cholera; and as regards the local outbreaks above adverted to, it is certain that the incriminated waters were contaminated with sewage, and that there was at least the probability that that sewage contained the evacuations of cholera patients. But, on the other hand, there is good reason to believe that the freshly passed stools are not specifically noxious. Much, however, of what seems mysterious in reference to these matters appears to be explained by the important experimental enquiries first conducted by Professor Thiersch, and since repeated by Dr. Sanderson in this country. The experiments which yielded the most striking results were those performed on mice. It was ascertained by these gentlemen that when, under certain conditions, mice were fed with cholera evacuations, they were attacked with symptoms which proved rapidly fatal, and that both symptoms and post-mortem appearances had a very close resemblance to those of human cholera. The

chief points of likeness consisted, in the rapidity and intensity of the disease; in a remarkable lowering of the temperature (sometimes as much as 20 degrees); in the accumulation in the intestines of thin fluid containing bacteria, other lowly organisms, and abundance of shed epithelia; and in the discharge of loose stools from the anus. The method adopted by Dr. Sanderson to infect the mice was to soak pieces of filter paper in fresh cholera evacuations, or in the contents of the bowels of patients dead of cholera, to dry them, to ascertain by weighing the quantity of solid matter thus added to them, to cut them into pieces an inch square, to soak them in bacon fat, and then to administer them to the mice. The mice under these circumstances ate them greedily. The consequences were—that of mice fed with paper prepared, from evacuations which had not been allowed to stand more than twenty-four hours, or on the first day, 11 per cent. were affected; of those fed with paper prepared on the second day, 36 per cent.; of those fed with paper prepared on the third day, every one; of those fed with paper prepared on the fourth day, 71 per cent.; and of those fed with paper prepared on the fifth day, 40 per cent. Paper prepared subsequently had no effect. These experiments show—that the cholera evacuations have little or no intensity of action when perfectly fresh; that their virulence increases up to the third day, diminishing during the fourth and fifth days; and that they lose all specific properties after that date. It should be added, that the evacuations from the diseased mice produced the same effects on healthy mice as did true cholera evacuations; and, further, that all experiments made by Dr. Sanderson in the month of November failed absolutely, probably, as he suggests, on account of the low temperature then prevailing.

The application of the above results in explanation of the phenomena connected with the causation of cholera is obvious. And it is fair to conclude from them, and from the other facts which have been adduced—that the specific poison of cholera is furnished by the discharges from the alimentary canal; that these are not operative when completely fresh, but acquire virulent infectious properties in the course of the following two, three, four, or five days, and subsequently lose them; that the poison of the disease is taken up by, or acts upon, the mucous membrane of the bowels, which it reaches through the mouth: and that, while undoubtedly it may be conveyed to the mouth under uncleanly circumstances from saturated bedclothes, and direct contamination of culinary utensils, food, or fingers, larger outbreaks of the disease are due to the infection of drinking water (well, pond, or river) with cholera poison derived from cesspools, sewers, or other such sources.

There can be little doubt, from the fact of its active powers of multiplication, that the cholera poison is an organised contagium; that one phase of its normal active existence is passed externally to the

body; but that that phase is commonly of short duration, and probably readily arrested or rendered innocuous by cold and other agencies.

Symptoms and progress.—The duration of the incubative stage of cholera is not known certainly. It probably varies generally between a few hours and three days. The symptoms of invasion present considerable variety. In some cases an indefinable feeling of malaise, associated with noises in the ears and lowness of spirits, precedes all other symptoms. In a very large proportion of cases (either in succession to the last or arising independently) there is more or less looseness of the bowels (premonitory diarrhœa) coming on a few hours, a day, or even two or three days, before the nature of the disease is distinctly revealed. Premonitory diarrhœa of even longer duration has not unfrequently been observed; but in most such cases there is reason to suspect that the relation of the diarrhœa to the subsequent attack of cholera was accidental only. Lastly, in some instances the invasion of cholera is quite sudden. Omitting the premonitory symptoms which have just been considered, the first indication of an ordinary attack of cholera usually consists in the sudden and uncontrollable evacuation (with or without pain) of an abundant loose stool, composed mainly of the proper contents of the alimentary canal in a fluid or semi-fluid state. To this succeeds a continuous or intermittent flux of fluid, at first bile-stained, but subsequently thin, colourless, or opaline, without fæcal look or smell, and containing in suspension whitish flocculi. The amount of fluid thus discharged is sometimes enormous; four or five pints, or enough to fill a chamber-pot, may be passed in the course of an hour or two. Sickness for the most part attends the diarrhœa, but generally comes on a little later. The matters first vomited are the ordinary contents of the stomach and of the duodenum; but after these have been got rid of, the vomited fluid exactly resembles that which is flowing simultaneously from the anus, and may be almost as abundant. Shortly after vomiting and diarrhœa have become established, severe cramps, attended with agonising pain, come on in the thighs and calves, in the arms, hands, feet, and parietes of the abdomen. And very speedily the patient falls into a state of extreme collapse—the so-called 'cold' or 'algide' stage; his tissues shrink; his fingers and toes get shrivelled and corrugated, and his eyes sink into their sockets; his surface becomes more or less notably livid, and sometimes as blue as that of a cyanotic patient—this change being especially noticeable in the hands, feet, cheeks, lips, around the eyes, and in the tongue, which looks like a piece of lead; his respirations grow rapid and shallow, and his voice hoarse or squeaking, feeble, and reduced almost to a whisper; his pulse gets rapid and thready, and soon scarcely, if at all, perceptible at the wrist or even in the brachial artery. At the same time his temperature falls; his surface becomes cold and clammy, and sometimes covered with cold sweats; and his

tongue and breath also get manifestly cool. The temperature in the mouth and axilla falls rapidly to 95°, 94°, or even 92°; and much lower temperatures than these have been recorded. But while the general temperature, and especially the surface temperature, thus fall, that in the rectum and adjoining parts may stand at 101°, 102°, or even 105°. The urinary and biliary secretions are totally suppressed. The patient is wakeful and restless, throwing his arms about, probably complaining much of intense thirst and burning at the chest, but withal singularly apathetic. When the condition of collapse is fully established, the vomiting and diarrhœa either cease completely or greatly diminish, and the patient lies ghastly and livid like a corpse, with eyes open and pupils dilated, torpid, yet still retaining his senses. During this period the muscular power is extremely enfeebled; yet occasionally the apparently moribund patient will rise up in his bed, and even get up and walk across the room. The duration of this stage varies from two or three to thirty hours or more, and then ends in either death, secondary fever, or convalescence. Death, in collapse, sometimes occurs in the course of two or three hours; more frequently it supervenes after the eighth hour—especially between the tenth and fourteenth; but is seldom delayed beyond the twenty-fourth.

The symptoms which have been above described are not all developed in every case of cholera. The muscular cramps are sometimes altogether wanting; while, in some cases, and these perhaps cases of no great severity, they are constant and agonising. Again, vomiting and diarrhœa are not invariably present; and indeed, their absence is almost characteristic of some of the most formidable attacks of the disease—those, namely, in which the patient is suddenly struck down with symptoms of extreme collapse, and dies in the course of an hour or two, or less.

In those patients who survive the period of collapse a gradual change of symptoms supervenes. The stage of reaction sets in. This stage is said to be often wanting in the cholera of hot climates. In our own country, however, it is always present; but its duration, and the severity of its symptoms, depend very largely on the intensity and duration of the cold stage which preceded it. It generally comes on between the twelfth or fourteenth and the thirtieth hour after invasion. Its first indications are slight and vague. A general improvement is visible in the patient; he becomes less restless, his breathing slower and more natural, his pulse just perceptible at the wrist; the lividity of surface slowly disappears; the shrunken tissues expand; the temperature rises; perspiration breaks out; and not improbably he falls into a comfortable sleep; urine begins to be secreted; and the motions are again stained with bile. The temperature, however, generally rises somewhat above the normal, and more or less obvious febrile disturbance takes place. In some cases the reactionary symptoms remain mild and end in convalescence in from twelve to twenty-four hours; but

more commonly they undergo aggravation, and may then be prolonged (unless cut short by death) to between four and twelve days, sometimes longer. The general symptoms have some resemblance to those of enteric fever; the face becomes flushed, the eyes injected, the skin hot and sometimes studded with roseolous patches, the pulse increased in power and volume and accelerated, the respirations a little more rapid than natural, the tongue furred, sometimes dry and brown, and the temperature one, two, or three degrees above the normal; the patient may also present more or less delirium, or lie in a torpid or comatose condition. The motions, according to Dr. Sutton's observations, often consist on the first establishment of reaction of a thin, yellowish fluid, which looks like and may be mistaken for urine, and often contain a kind of gelatinous substance; but soon they get green from contained bile, next peasoup-like, and then, consolidating, gradually acquire the normal character. Occasionally, early in the stage of reaction, the stools contain blood—the quantity varying from a mere trace, just sufficient to impart to them a pale pink tinge, up to a flux sufficient to undergo very complete coagulation. The stools of the reactive period are often very fetid. The re-establishment of the urinary secretion is a most important element in the progress of the disease. In mild cases it sometimes takes place in twelve hours or less; but it is more common on the second or third day, and may be delayed until the fourth, fifth, or sixth day. The urine first passed is in extremely small quantity, and often, during the first twenty-four hours, remains far below the healthy average. Subsequently the patient may pass four, five, or six pints daily. At first it is a little turbid, contains traces of albumen, casts of the urinary tubules, and epithelial cells from other parts of the urinary passages, but presents a very small amount of urea and uric acid, as also of chlorides, phosphates, and sulphates. The colour varies. Subsequently, while during the progress of fever the urine becomes more copious, the amount of urea in it increases, and may even exceed the healthy standard. Urocyanogen is sometimes found in the urine.

The causes of death in the stage of reaction, and the phenomena which precede it, present considerable variety. Sometimes cough and difficulty of breathing, with pulmonary engorgement or consolidation, carry the patient off. At other times he seems to sink under the continuance of intestinal flux, especially when hemorrhage accompanies it; or symptoms much like those of enteritis supervene. In some cases convulsions, coma, or other cerebral symptoms, which there is good reason to believe are not unfrequently due immediately to uræmic poisoning, precede and apparently cause death. Lastly, the patient sometimes sinks from mere asthenia, arising directly out of his primary symptoms, or supervening on his typhoid condition.

In the description of cholera above given we have adverted to some of the varieties which its attacks present. Especially we have pointed

out, or incidentally mentioned,—that in some cases the patient is struck down by the disease, and dies in extreme collapse at the end of perhaps two or three hours, without ever having passed an evacuation; that in a still larger number of cases the characteristic vomiting and diarrhœa are present, the stage of collapse gradually supervenes, and the patient dies in this stage at the end of from (say) ten to twenty-four hours; that in many cases again, even of considerable severity, the patient emerges from the condition of collapse into one of febrile reaction, during which he may perish in one of the modes above enumerated, or from which he may glide into convalescence; and, lastly, that in some cases, notwithstanding the presence of rice-water stools and other quite characteristic signs of the disease, the patient scarcely becomes collapsed at all, and very speedily regains health and strength. This enumeration leads up to the important questions—as to how far cholera may be so mild as to simulate in its attacks mere summer or autumnal diarrhœa, and how far also it is possible that the latter which (in this country, at all events) concurs with the epidemic prevalence of cholera is influenced by the choleraic poison. As to the former question, there can be no doubt, we think—that, just as enteric fever, typhus, scarlatina, and other like affections, are sometimes so mild and slightly developed as to be (except it may be from associated circumstances) incapable of identification, so cholera may be so mild and so shorn of everything characteristic as to be unrecognisable as cholera; and that hence cases of undoubted cholera may simulate, and be taken for, cases of ordinary unspecific diarrhœa. As to the latter question, it may be remarked that those who regard cholera as being the outcome of some 'epidemic constitution' of the atmosphere, or of some all-pervading miasm, might reasonably believe that all morbid conditions tend during the prevalence of cholera to take on a choleraic character. Those, however, who believe the choleraic poison to be a form of contagium, and accept those views of its operation which we have endeavoured to uphold, would necessarily discredit its general influence, excepting in the face of overwhelming evidence in favour of the existence of such influence. But no such evidence, we think, exists. It seems to us, indeed, a fundamental and mischievous error to regard the diarrhœa which precedes and accompanies epidemics of cholera as having any other than a fortuitous connection with them.

The mortality of cholera is very great; it varies in different countries and in different epidemics, but in round numbers may be estimated on the average at about 50 per cent. It is said to be less fatal towards the close of an epidemic than at its commencement; and further to be more fatal to the very young and very old than to those whose age lies between these extremes.

Any affection attended with sudden and extreme collapse, especially if there be at the same time gastro-intestinal disturbance, may be mistaken for cholera; among those most liable to be thus confounded are

arsenical poisoning, and poisoning by croton oil; severe summer cholera; perforation of the stomach or bowel; extensive enteritis; and the onset or cold stage of severe remittent fever.

Morbid anatomy and pathology.—The post-mortem appearances found after death from cholera differ according as death takes place in the stage of collapse or in that of reaction. In the former case, the body retains much of the shrivelled character and lividity which it presented during life, and the dependent parts are often more or less deeply congested. The muscles not unfrequently contract for some little time after death, causing movements of the limbs; and for the most part rigor mortis is well-marked and prolonged. The tissues of the body are preternaturally dry, the muscles firm and dark-coloured, and the systemic veins loaded with blood which is manifestly thicker and perhaps darker than normal. For the most part the serous cavities are empty of fluid and their surfaces sticky to the feel, and they not unfrequently present subserous petechial extravasations. The right cavities of the heart are always more or less distended with dark-coloured, imperfectly coagulated blood. The left ventricle is sometimes firmly contracted and empty, sometimes contains a little fluid blood or clot. The left auricle also presents a small quantity of blood. The lungs are usually much diminished in weight, pale, anæmic, and dryish on section. Sometimes, however, they are congested and œdematous below, and they may even be more or less congested and œdematous throughout. The pulmonary arteries are usually gorged with blood, the veins nearly or quite empty. The liver presents no decided departure from health; and the gall-bladder is full of bile. The spleen is generally reduced in size. The outer surface of the bowels is often injected or of a diffused rosy tint. Their mucous membrane is sometimes of a nearly uniform pink tinge, increasing in intensity towards the cæcum; or it may present irregular patches of congestion, with submucous extravasations; or it may be quite pale. It often exhibits a corrugated and sodden appearance; and the solitary and Peyer's glands are for the most part enlarged. The contents are an opaline or gruel-like fluid, which is sometimes white, sometimes pink from admixture with blood. The mucous lining of the stomach is often congested and mammillated, and the contents generally resemble those of the bowels. The kidneys are congested on the venous side, so that the medullary portions and the superficial veins are injected, while the cortical substance remains more or less pale. The urinary bladder is firmly contracted, and empty or containing a little pus-like fluid. The brain presents numerous *puncta cruenta*.

If death occurs during reaction, the tissues are found moist; blood occupies, perhaps in equal degree, both sides of the heart, and not unfrequently thick fibrinous coagula are prolonged thence into the aorta; the lungs are congested and œdematous; and the contents of the intestines present the appearance of pea-soup. Besides which changes

pneumonia is sometimes met with, sometimes distinct inflammation of the intestinal mucous membrane.

Other pathological facts of great interest have been ascertained with respect to this disease. Although, as has been stated, the blood is inspissated, it is not by any means so much so as is commonly believed; but (according to Dr. Thudichum) it is more adherent to the blood-vessels than natural. The proportion of albumen and salts to its other solid constituents is diminished; and the white corpuscles are often increased relatively to the red. The rice-water fluid, as found in the intestines, is alkaline, in a state of rapid decomposition, evolves gases (chiefly nitrogen and carbonic acid), and contains, besides bacteria, shed epithelium in abundance, mucine, albumen, and also butyric acid, acetic acid, ammonia, leucine, and inorganic salts. It does not, however, contain urea. There is no doubt that after death the mucous surface of the bowels loses its epithelial covering, which is thrown off in flakes and suspended in the intestinal contents. But it is uncertain whether this is merely a post-mortem change or a lesion occurring during life. It is probably the latter, however, for there appears to be a similar tendency to shed the epithelium in almost every other part in which epithelium exists, especially in the bladder and urinary passages, in the bronchial tubes, and in the ducts of the liver and of the salivary glands. Dr. Thudichum's observations show that during the period of collapse the blood and the tissues contain very little urea; but that its quantity increases during the period of reaction, and soon, if urine be not secreted, becomes excessive.

It remains briefly to discuss the relations between the post-mortem appearances and the vital phenomena of the disease. It is obvious that we here have an affection which is characterised primarily and mainly by a sudden and profound impression on the mucous surface of the alimentary canal; in dependence on which, active destructive changes take place (as evidenced by the raised temperature of the parts) and large quantities of imperfectly filtered blood, with tendency to rapid decomposition, are poured forth with sudden impetuosity. This rapid and profuse discharge tends to cause inspissation of the circulating blood, and consequently indirectly, but very thoroughly, to drain the tissues of their interstitial fluid, and to cause them to shrivel up. Anasarca, indeed, if present becomes thus temporarily cured. The absorption of extra-vascular fluid into the blood-vessels tends, of course, to maintain the fluidity of the blood; but, notwithstanding this, the blood almost invariably becomes thicker than natural, and less easy of transmission through the minuter vessels. On these conditions follow contraction of all the smaller arteries, excepting, probably, those connected with the bowels; general failure of the circulation; arrest of normal destructive changes, and therefore of formation of urea; arrest of urinary, biliary, and salivary secretions; and diminution of the normal action of the lungs, with cyanosis, lowering of temperature, and generally collapse.

All the above phenomena flow directly or indirectly from the effects of the cholera poison. But how and where does the poison act? Some believe that it acts simply on the intestinal mucous membrane as a violent local irritant, just as croton oil or elaterium acts, and that all the symptoms which ensue are the result of this irritation of the mucous membrane and of the discharge which takes place from it: and there is no doubt that symptoms almost identical with those of cholera may be produced by the local action of irritants and irritant purgatives. But if it be true, as it seems to be, that the fœtuses of mothers dying of cholera themselves give clear indications of being affected with the disease, it is clear that the poison must be diffused throughout the system in addition to being contained in the alimentary canal. And, indeed, it is most consonant with all we know of similar diseases to regard cholera as a systemic affection. But whether we are therefore to assume, with Dr. George Johnson, that the choleraic virus is contained in the blood; that by its presence there it causes cramp of the voluntary muscles on the one hand, and of the capillary arteries of the lungs on the other, so as to prevent the passage of blood through them; that the general collapse, loss of temperature, and suppression of secretions are due to this mechanical obstruction; and, lastly, that the discharge from the bowels is an effort of nature (which should be encouraged) to eliminate the poison from the blood, is quite another matter. We confess that, in our view, the intestinal flux is not eliminative, but connected, as is the eruption of small-pox, with the local growth and multiplication of the poison; and that there is ample explanation in the processes which are going on in the bowels of nearly all the subsequent phenomena of the disease, including collapse. It is obvious, however, that the presence of inspissated blood in the vessels, the drying up of the moisture of the tissues, the contraction of the smaller branches of the pulmonary artery (assuming it to take place) must all co-operate to maintain the patient in the condition of collapse.

Treatment.—The value of precautionary and hygienic measures in preventing or limiting the outbreak of cholera has never been better shown than in the history of our own epidemics. Pure water, well filtered, and carefully guarded from fœcal contamination; thorough domestic cleanliness; and when cholera is present, the immediate disinfection by carbolic acid or Condy's fluid of all evacuations, and contaminated articles, are conditions of the utmost importance in preventing the spread of the disease.

The medicinal treatment of cholera resolves itself into that of the prodromal stage, that of the period of collapse, and that of the stage of reaction. It is commonly believed that the treatment of the premonitory diarrhœa is a matter of vital importance to the patient; and the assumption that the diarrhœa, which so often prevails when cholera is epidemic, is actually cholera, or simple diarrhœa modified

by choleraic influence, has led to a general belief in the importance of treating at such times all diarrhœal cases with the object of preventing their development into the graver malady. But unfortunately, while the majority of physicians laud astringents for this purpose, others prefer castor oil, and all refer to statistics in proof of the efficacy of their respective modes of treatment. We have asserted our own belief that if a case be one of simple diarrhœa, it will not run on to cholera under any form of treatment; and we may add that, if the case be one of commencing cholera, there is no more ground for believing that it can be cut short than for believing that typhoid fever or hooping cough can be cut short. We do not believe that either castor oil or astringents have any such influence.

In the period of collapse all sorts of remedies have been adopted; some have given calomel in large doses, some opium, some brandy, some castor oil; but it seems clear that drugs administered by the mouth must in such cases prove quite inoperative. And this is certainly the opinion of nearly all except the enthusiastic supporters of some special drug. During this stage the patient should be kept in the horizontal position; he should be allowed cold or ice-cold water to relieve his insatiable drought; and his surface should be kept warm by the application of hot bottles or flannels, or by friction. The placing of the patient in a bath, two or three degrees above blood heat, is often very comforting and apparently of much service. The vapour bath is equally beneficial. It is in this stage that the injection of saline fluids into the veins has been so frequently tried, and occasionally with success. The immediate effect of the injection is often marvellous, the moribund patient regains his healthy appearance, his respirations, pulse and voice resume their normal characters, and he sits up in bed conversing cheerfully. But the improvement is generally of short duration; he falls again into collapse, and probably dies. The solution employed should resemble as nearly as possible the serum of the blood, and should be injected slowly and cautiously, in quantities varying, according to its effects, between 10 oz. and one or two pints. Schmidt recommends the following:—chloride of sodium 60 parts, chloride of potassium 6, phosphate of soda 3, carbonate of soda 20; of which mixture 140 grains are to be dissolved in 40 oz. of distilled water, and filtered. The temperature of the fluid as it enters the veins should be a little over that of the blood. Cramp may be relieved by friction, or the inhalation of chloroform.

Great care must be taken of the patient during the reactionary stage. He should be kept cool. Diarrhœa and vomiting must be restrained—the former by astringents, such as Dover's powder, compound kino powder, or the aromatic powder of chalk and opium; the latter by lime-water, bismuth, and the like, or the use of ice or the application of counter-irritants. The food should be fluid, nutritious, and unstimulating: milk, broth, arrowroot, sago, barley-water and

eggs, are the most appropriate. It is questionable whether stimulants are beneficial. If given they should be in small doses much diluted. It is of essential importance that the urinary secretion be restored; but it is unwise to employ stimulant diuretics for the purpose. Saline effervescents may relieve sickness and at the same time promote urine. Cupping glasses and counter-irritation to the lumbar region are believed to be sometimes serviceable. If dysenteric or enteritic symptoms come on opium must be freely used.

XX. HYDROPHOBIA. (*Rabies.*)

Definition.—A disease special to dogs, wolves, foxes, and animals closely related to them, among which it spreads by direct contagion, and from which it is imparted (but by inoculation only) to other animals and to human beings. Its most characteristic features in man are the spasms and terror which are induced by the attempt to swallow fluids, or even by the thought of swallowing, and its invariably and rapidly fatal issue.

Causation and history.—There is no evidence to show that this disease ever arises spontaneously among dogs any more than small-pox does among men; and further, there is reason to believe that it spreads among them by inoculation only, or rather, perhaps, by the introduction of the saliva of diseased animals into the tissues of those which are healthy, by whatever process that introduction is effected. The cause of the disease is evidently a specific virus which resides mainly in the viscid secretions which are furnished by the mucous membrane of the mouth and fauces and by the salivary glands. The prevalence of rabies, like that of other infectious diseases, varies very greatly at different periods; sometimes it is scarcely observed for many years together, at other times it prevails widely in an epidemic form. The circumstances on which these differences depend are obscure; for climate, season, dearth of water and of food, and other such conditions do not seem to have any influence over it. It is important, however, to know that the virus never inoculates when it is applied to the surface of the sound skin; and that only a small proportion of those who are bitten by rabid dogs become hydrophobic. This proportion has been variously estimated at from 5 to 50 per cent. One main reason doubtless of the immunity which so many who are bitten enjoy, is the fact that they are wounded through their clothes, and that the fangs are thus cleansed from all moisture before they enter the skin.

Symptoms and progress.—After a man has been inoculated with the

saliva of an animal suffering from rabies, the wound in most cases heals as readily and quickly as a wound not so inoculated would heal; at all events, there is nothing in its progress to indicate the existence of anything unusual. A period of latency follows, which is generally remarkable for its long duration. In most cases the first symptoms show themselves between the fourth and eighth week, but they have appeared in the course of a few days, and have been delayed for months and even it is asserted for several years. They rarely, however, appear after four months. The outbreak of hydrophobia is in some cases preceded for a day or two by heat, tingling or pain at the part on which the injury was inflicted, the pain being sometimes intense and extending upwards in the course of the sensory nerves. There is occasionally also renewed inflammation and suppuration or ulceration. In many cases, on the other hand, no such phenomena present themselves.

The period of invasion, which is sometimes termed the '*melancholic stage*,' is attended with a variety of symptoms, most of which have no particular significance, and which gradually merge in those of the fully-developed disease. The patient complains of feverishness and shivering, with dryness of mouth and thirst, want of sleep, epigastric uneasiness and indefinable anxiety. He is pale, anxious, but distraught in his aspect, with restless eyes and dilated pupils, restless and fidgety in his movements, garrulous, but speaking in short sentences and in a jerky, abrupt manner. He suffers also from increased frequency of the heart's action and loss of appetite, perhaps nausea and vomiting; and not improbably has even now some feeling of constriction about the fauces with a disinclination to swallow fluids, quickened and sighing respiration, general hyperæsthesia, and a tendency to priapism and seminal discharges.

At the end of two or three days the next stage has become fully developed. This is sometimes termed the '*stage of excitement*,' and in it the disease assumes all its typical features. The strange agitation of the patient has become more marked; his eyes are bright, mobile, wild, and glance with suspicion or terror about him; his hair is rough, his skin pale, his brow contracted, his aspect indeed closely resembles that of a patient with acute mania; he is still inclined to be talkative, frequently making odd but pertinent remarks; he is probably quite sensible, and capable of understanding and reasoning; at the same time he is obviously under the domination of some indefinable but great horror; and occasionally perhaps he has hallucinations, and is liable to outbreaks of violent maniacal excitement in which he may endeavour to injure himself or others. The thirst has increased; his mouth and fauces are congested and dry; and a quantity of tenacious saliva accumulates, which he is constantly hawking up and spitting about him with a noise which has often been taken for a bark. But, above all, the disinclination to swallow fluids has now become an almost perfect in-

ability to swallow them, and a dread of making the attempt. He will still perhaps resolutely try to drink, will take the glass of water in his hand, prepare himself with strange calm and deliberation to make one supreme effort, put the vessel hurriedly to his lips, make a sudden gulp, and then, with or without swallowing a little of it, eject the bulk of it spasmodically and violently from his mouth, and throw the glass away. A convulsive attack has been induced, marked by general tremors or shuddering, and violent spasmodic action of the muscles of deglutition and respiration, which lasts for a few seconds, and leaves the patient for a minute or two in a state of painful agitation. The fear of the recurrence of these terrible convulsions is constantly before him, and their actual recurrence is soon induced, not merely by the attempt to swallow, but even by the sight or sound or thought of fluid. The general hyperæsthesia, which has already been adverted to, becomes more acute. The patient will often complain of the mere weight of the hand, or of his bed-clothes; and a draught of cold air upon the surface suffices to induce a convulsive attack. Bright objects, and loud or harsh or unaccustomed sounds are painful to him, excite a feeling of terror, and not unfrequently also provoke convulsions. The sexual excitement, of which the patient complains bitterly, may also continue. He passes urine frequently.

As the disease progresses all the symptoms become more severe; the patient gets feebler, his pulse quick, irregular, and small, his skin clammy, his voice hoarse, the tenacious mucus which is secreted by the mouth and fauces accumulates and becomes more difficult of expulsion; the paroxysms of general convulsive action and of spasm of the respiratory muscles increase in severity and frequency; and at length he dies either of sudden asphyxia in one of these convulsive attacks, or of slow asphyxia induced by their rapid recurrence, or of exhaustion, aided possibly by a general paralytic condition.

The most remarkable phenomena of the disease are, first, the hyperæsthesia of the skin and organs of sense; second, the tendency which impressions on these organs, and attempts to swallow, or thoughts of swallowing liquids, have in producing clonic and tonic spasms of the respiratory muscles; and, third, the wakefulness, horror and tendency to yield (while apparently still quite rational) to insane impulses. The last condition is occasionally absent; or the patient only rambles slightly immediately before death. The disease is invariably fatal, and generally terminates between the second and fourth day.

Rabies in dogs presents in great measure the same symptoms as hydrophobia in man. There are, however, one or two important points of distinction:—namely, dogs are not afraid of water, and will indeed, on the contrary, bury their muzzles in water while at the height of the disease; cutaneous hyperæsthesia seems to be absent in them; and towards the close a paralytic condition supervenes, involving especially their hinder extremities and the lower jaw.

Morbid anatomy has not as yet thrown any important light upon the phenomena of hydrophobia. The muscles retain their rigidity for some time after death, and there is more or less obvious congestion of the posterior surface of the corpse, and of the fauces, pharynx, œsophagus, larynx, trachea, and lungs. Recent investigations[1] by Drs. Coats, Gowers, Greenfield, and others have demonstrated the presence of hyperæmia of the central nervous organs, with the accumulation of leucocytes around the smaller vessels and capillaries of the cerebral convolutions, the ganglia at the base of the brain, the grey matter of the cord, and especially that of the medulla oblongata. Small extravasations of blood have also been found in the grey matter of the dorsal and cervical regions of the cord. Dr. Coats further describes extravasation of leucocytes into the salivary glands, mucous glands of the larynx, and kidneys. There can be little doubt that the hydrophobic virus exerts its influence mainly on the sensory and emotional regions of the central nervous organs. Dr. Marochetti, in 1820, described the formation of small vesicles beneath the tongue in persons bitten by mad dogs. These vesicles, which have also been described subsequently by one or two other physicians, are said only to occur during the second week after inoculation.

Treatment.—Whenever a patient has been bitten by a rabid animal or one suspected of having rabies, the wounded part should at once be excised and the remaining raw surface freely treated with caustic potash, nitric acid, the acid nitrate of mercury, the actual cautery, or some equally efficient destructive agent. No remedy has been discovered competent to arrest the progress of the once established disease. Drugs producing narcotism and anæsthesia might seem to offer some chance of benefit, but it is doubtful if any has been found of service, except perhaps in the relief of suffering. It should be observed, however, that a case of recovery is said to have occurred in 1874, in the practice of Dr. Offenburg, of Winkrath, under the use of injections of curara, of which about a third of a grain was administered every fifteen minutes or so. The patient was well on the eighth day. Any drug that may be employed should be administered by inhalation, by the rectum, or by subcutaneous injection. Tracheotomy has been suggested in the hope of averting death by asphyxia. Great care should be taken to prevent the patient from doing violence either to himself or to those about him, and especially to prevent inoculation of wounds by the saliva which he disperses.

[1] *Lancet*, vol. ii. 1877, p. 882.

XXI. GLANDERS. FARCY. (*Equinia.*)

Definition.—A specific disease, special to the horse and animals of the same genus, but communicable to man, and characterised by a peculiar tubercular affection of the nasal and respiratory mucous membranes and of the skin, lungs, lymphatic glands and other parts of the body.

Causation and history.—Whether or not the disease originates spontaneously in the horse is a matter of dispute. It is certain, however, that it spreads readily among horses and from them to men by contagion—mainly by the virus contained in the secretions of the nasal mucous membrane; and, further, that it is similarly transmissible from man to man.

Symptoms and progress.—The period of incubation probably varies between one and about fifteen days. It is said to be occasionally much prolonged. Two varieties of equinia are met with, which go by the respective names of '*glanders*' and '*farcy*'; the difference between them depending mainly on the seat of inoculation, and on the absence or presence of early affection of the nose and air-passages. These varieties run into one another even in the horse; in man they are generally combined. The symptoms of invasion are those of intense febrile disturbance—heat of skin, rigors, acceleration of pulse, headache, febrile urine, pains in muscles and joints, and often nausea and vomiting, and profuse perspirations. The specific phenomena of the disease soon follow. These consist in an affection of the nasal mucous membrane and of the mucous surfaces which are continuous with it, and an eruption on the skin. The mucous surface of the nostrils becomes congested, and secretes a thin, acrid, watery fluid, which soon gets thick, tenacious and profuse, and probably assumes at length the characters of sanious pus. The cutaneous eruption is thinly and irregularly scattered, and chiefly on the face, extremities, neck, and abdomen. It consists at first of red points; but these soon increase in size, ultimately perhaps attaining the bulk of peas, and feeling hard and shotty between the fingers, and not unlike syphilitic chancres. A vesicle or pustule soon makes its appearance on the summit of each spot, enlarges, bursts, exudes a more or less abundant purulent fluid, and leaves an irregular sloughy ulcer, with a livid margin. A little later, other phenomena manifest themselves: the conjunctivæ yield a purulent secretion; sores arise on various parts of the mucous surface of the oral cavity and pharynx; and bronchitic, pulmonic or pleuritic symptoms are added; erysipelatous redness and swelling of the eyelids, nose, cheeks and forehead become developed; and subcutaneous or deeper-seated tubercles and abscesses—the latter often of considerable size—appear in various parts, but mainly in the face and in the vicinity of joints. Whilst these symptoms are in progress, the

patient becomes weak and prostrate, his pulse quick and feeble, his muscles tremulous, his tongue dry and brown, and delirium comes on; in a word, typhoid symptoms rapidly develop themselves, on which coma supervenes, and death soon follows. The breath during this period is generally very fetid, the perspiration abundant, there is often diarrhœa, and gangrene sometimes attacks the nose, eyelids, and other parts. The course of the disease is generally acute; the temperature may rise to 104° or even to 106°; and death supervenes, sometimes during the first few days, but more commonly between the seventh and fifteenth or sixteenth. Occasionally in man (but much more commonly in the horse) the disease is chronic. The invasion is then more gradual, the various phenomena follow one another at longer intervals and the eruption is often absent; but the subcutaneous abscesses which form become larger, the resulting ulcers are often attended with sloughing, and the affection of the nostrils extends and leads even to the exposure and destruction of the bones. The patient passes into a hectic condition, and may linger for weeks, months, or years. The blood is said by Colin to be greatly surcharged with white corpuscles.

Farcy is generally dependent on the inoculation of a wound on some part of the trunk or limbs. The inoculated part gets inflamed and painful, and the absorbent vessels and glands in relation with it soon become similarly affected. Then supervene more or less of the febrile disturbance that characterises glanders, and the formation of subcutaneous lumps (farcy-buds) and abscesses; frequently, too, the absorbent glands become generally inflamed and suppurate. The cutaneous rash is not so frequently present in farcy as in glanders, and the nasal inflammation is often absent. It must be added, however, that all the special symptoms of glanders occasionally supervene. This variety of equinia may occur in either the acute or the chronic form. The latter is sometimes exceedingly ill-marked and difficult of diagnosis.

Equinia is generally a fatal disease. The chronic forms are most likely to be followed by recovery, and farcy more so than glanders. In its early stage, and in the absence of rash or nasal implication, equinia may be readily mistaken for acute rheumatism or pyæmia.

Morbid anatomy.—The anatomical phenomena of equinia consist mainly in the formation of tubercles, presenting to a great extent the structural features of true tubercles, and like these tending rapidly to undergo caseous degeneration and liquefaction or suppuration. When superficial, they speedily form unhealthy-looking ulcers. When deeper seated they become converted into abscesses, which then gradually enlarge and ultimately burst. The tubercles vary from the size perhaps of a pin's head to that of a pea or bean. It is to their development in connection with the mucous membrane of the nose that the peculiar symptoms referrible to this organ are due. They also form in the mouth and fauces, in the larynx, trachea and bronchial tubes; and they appear in the substance of the lungs, producing a condition not

unlike that of ordinary lobular pneumonia, and often inducing pleural inflammation. The cutaneous eruption is due to the growth of these tubercles in the skin; and the subcutaneous lumps and abscesses, and those which arise in the substance of muscles, are of the same nature. The kidneys, spleen, testicles and other organs are also occasionally affected. Implication of the lymphatic glands is not unfrequent, but must be regarded as generally, if not always, secondary to specific lesions occurring in parts with which they are connected. More or less of simple inflammation is generally associated with the specific lesions.

Treatment.—It is impossible to lay down any authoritative rules for the treatment of equinia. No specific is known, and no drug which has any favourable influence over its course. Iodine, arsenic and strychnia have each been recommended. All that can be done, probably, is to support the patient by nourishment, stimulants and tonics; to relieve pain and other symptoms; and to cleanse, and treat with stimulating or astringent lotions, or other applications, the nasal mucous membrane and other inflamed and ulcerated parts which are within reach. During convalescence change of air and good diet are of course important.

XXII. SYPHILIS.

Definition.—A specific disorder, communicable only by inoculation, resembling the exanthemata in the facts, that it presents a period of latency, and a period during which characteristic eruptions make their appearance, and that one attack confers protection; but differing from them in the remarkably long duration of these periods, and in the tendency to the recurrence, it may be for many years, of specific lesions.

Causation and history.—Syphilis has occasionally prevailed in the form of widespread and severe epidemics. One such epidemic passed through Europe during the latter part of the fifteenth century; and it was probably in great measure owing to this fact that, for a time, it came to be assumed that the disease first made its appearance in Europe after the discovery of America, and had been imported from that continent. There is no doubt, however, that this was an erroneous assumption; and that, just as syphilis prevails now, so it has prevailed from the earliest times both in Europe and in the other quarters of the Old World. Like many other diseases, syphilis was long confounded with affections which, though often associated with it or arising under analogous circumstances, are essentially distinct from it. Hunter regarded

gonorrhœa as one of its manifestations, and even until quite recently other forms of circumscribed inflammation of the surface of the genital organs have been confused with the true chancre—the sore which arises at the point of syphilitic inoculation. But, thanks to the labours of Ricord and other recent observers, including Mr. Henry Lee, the phenomena of syphilis apparently have now been fully disentangled from those of the maladies which simulate it, and our knowledge of syphilis is as accurate as is our knowledge of scarlet fever or of small-pox. The symptoms of syphilis are quite characteristic, and when fully developed, can rarely escape ready recognition; yet the disease, though maintaining its identity and typical features, has varied very greatly in its virulence at different times and in different countries, under circumstances the nature and relative importance of which it is not easy to estimate. Of the specific nature of syphilis, therefore, there can be no doubt. There is equally no doubt that it spreads by means of a specific contagium, and that there is no evidence to show that it ever originates spontaneously. The specific poison of syphilis is never imparted, like that of typhus, by atmospheric conveyance, or, like that of cholera, by means of diffusion through water; it acts only when directly introduced by inoculation. For the most part it is imparted in the act of sexual intercourse by the secretions which are furnished by primary or secondary sores—the thin cuticular covering of the glans penis and inner surface of the prepuce, and the mucous membrane of the urethra, and the corresponding parts in the female, becoming readily inoculated even when no breach of surface exists. It is also not unfrequently transmitted from the sucking child to its nurse, or from the nurse to her suckling, either from the mucous membrane of the mouth to the nipple, or conversely, or from mouth to mouth. But, indeed, inoculation may take place at any part, provided only the cuticular layer be not too thick, or there be an excoriation or wound; thus syphilis has not unfrequently been accidentally inoculated on the hands of medical men, and occasionally has been imparted by the operation of vaccination. And, lastly, it is a common thing for syphilitic parents to procreate children who also are syphilitic. Thus a syphilitic mother may have a syphilitic child, the father remaining uncontaminated; or a syphilitic father may beget a syphilitic child, and may infect the mother either directly, or indirectly through the fœtus. The contagium of syphilis resides in its most virulent form, doubtless, in the primary syphilitic sores, and in the indurated glands which succeed to them; but the contagious influence persists during the secondary phenomena of the disease, and also during the period of so-called 'tertiary' manifestations, as is distinctly proved by the fact already adverted to—namely, the transmission of the disease in its later stages from parents to their offspring and from these to healthy wet-nurses. Experiments have been made which seem to prove that the blood of syphilitic patients possesses contagious properties; but there can be no

doubt that, as well in the later as in the earlier stages of the disease, the virus is mainly concentrated in the specific lesions. It may be added that the secretions of syphilitic patients, more especially the milk, semen, and products of the mucous surfaces, have been supposed to possess infectious properties. But Mr. H. Lee is probably right when he insists that only those organs yield infectious discharges which are either distinctly implicated in the syphilitic process or are in a condition of inflammation. The protective influence of one attack of syphilis has only been fully recognised since the true disease has been disencumbered of the maladies which had grouped themselves with it. It is now established beyond doubt that syphilitic inoculation affords as secure a protection against subsequent attempts at inoculation as does one attack of small-pox or scarlet fever against subsequent attacks of either of these affections; that a person fully under the influence of the syphilitic poison, or who has had an attack from which he has recovered, very rarely acquires a chancre even when inoculated under the most advantageous circumstances, and even more rarely suffers in consequence from the secondary symptoms which so surely follow on the primary inoculation; and, further, that a person inoculated a second time, during the period which elapses between a primary inoculation and the maturation of the primary chancre, has as the result of his second inoculation a modified chancre—a chancre which runs its course with exceptional rapidity, and attains its full development concurrently with its elder brother.

Symptoms and progress.—1. *Primary symptoms.* When a successful inoculation has been effected on an unprotected person, the virus remains apparently quiescent for a period of uncertain duration, but which is estimated by Lancereaux at from eighteen to thirty-five days, with a mean of twenty-eight days. At the end of that time, a minute dusky red papule makes its appearance, which for the most part is unattended with either pain or itching, and slowly enlarges. Soon a thin greyish crust, the result of superficial necrosis, forms on its most prominent part. Whilst the papule gradually increases in area, successive crusts are formed and shed from its surface, which thus becomes more and more eroded; so that before long the papule, which has now become a tubercle, displays an elevated dusky red margin surrounding a concave excavation, with a grey dry surface. Almost from the beginning the papule has a remarkable indurated character and appears imbedded, as it were, in the substance of the skin. These characters it retains, the induration extending a little beyond the area of elevation, and presenting a very obvious edge, so that the mass can be readily grasped between the finger and thumb. At the end of about six weeks the tubercle has attained its complete development, and is perhaps of the size of half a pea, or somewhat larger. It then begins slowly to subside, and after a while cicatrises, generally however leaving behind more or less dusky discolouration, induration, and permanent depression. This is the

course of the true Hunterian chancre. But, just as the inoculated cow-pox vesicle presents many deviations from its natural course, so does the pimple which results from syphilitic inoculation. For a description of these reference must be made to surgical works. It should be stated, however, that inoculation sometimes takes place without the development of any appreciable local sore, and that a sore may have existed and yet no visible cicatrix remain.

A week or two, usually, after the first appearance of the chancre, the lymphatic glands in relation with the affected part begin to enlarge. If, therefore, the chancre be on the genital organs the glands of one or both groins suffer. The enlargement is slow and painless. For the most part several glands are affected, and each probably attains the size of an almond shell. They remain freely movable under the integuments, and are characterised, like the chancre itself, by extreme induration. They seldom undergo suppuration, but remain with little change for months or years.

The period, to which the above phenomena belong, corresponds exactly to the incubation of the exanthemata, and consequently to that period in the inoculated small-pox during which the primary pustule attains maturity, and which precedes the general variolous outbreak.

2. *Secondary symptoms.*—From six weeks to three months, generally perhaps between sixty and seventy days, after inoculation, the eruptive stage, or stage of secondary symptoms, supervenes. The invasion of this stage is often indicated by slight febrile symptoms, attended with more or less obvious recurring exacerbations, increased frequency of pulse, loss of appetite, weakness and emaciation, cachexia, restlessness, want of sleep, and pains more or less variable but augmenting towards night in the head, joints, and back. Shortly afterwards, or sometimes concurrently with the febrile disturbance, phenomena of a more characteristic kind manifest themselves. Among the earliest of these are certain affections of the skin and mucous membranes, and inflammations of the joints, bones, and eyes. The cutaneous affection, which is a form of roseola, generally first appears on the trunk, but before long involves the face and extremities, including the palms and soles. It is in the beginning a mere subcutaneous rash of roundish dusky-red spots, varying from one to two or three lines in diameter, and fading at the edges. But they soon become slightly elevated and lenticular in form. They are variously scattered, but are not unfrequently grouped in segments of circles or in circles. The rash comes out in successive crops, and may continue off and on for some two or three months. In association with it the hair not unfrequently gets dry and loses its gloss, and presently begins to fall out; and thus more or less complete baldness is apt to ensue. This roseola is sometimes the only rash which makes its appearance, but very commonly it constitutes the first stage of some other variety of skin-disease. Thus, sometimes the individual roseolous spots or the patches formed by the coalescence of

several, gradually enlarge, and fading away in the centre form circles or irregularly rounded marginated tracts of erythema circinatum; sometimes as they enlarge they get covered with thin scales, and acquire a close resemblance to lepra or psoriasis; sometimes they assume the form of distinct but flat tubercles; sometimes they become the seat of vesicles or blebs, and occasionally even of pustules. And hence the secondary eruption may acquire an erythematous, scaly, papular or tubercular, vesicular or pustular condition, or may present several or all of these characters at the same time variously combined. But besides this peculiar polymorphous character, which of itself points to syphilis, there are generally certain peculiarities about the eruption which, apart from all other considerations, indicate its specific character. In the first place, it often presents a peculiar dusky-red or coppery tint, which is due to some pigmentary deposit in the substance of the cutis, and tends to persist long after the actual eruption has disappeared. It may be worth while to remark that occasionally the course of the superficial veins in the extremities, and especially along the shins, becomes mapped out by similar dusky pigmentary stains. This condition is not peculiar, however, to syphilis. In the second place, syphilitic eruptions have a singular aptitude to affect those parts which the non-specific eruptions they resemble specially avoid; they are common on the flexor aspects of the joints, about the forehead, where they often cause the so-called 'corona veneris,' and especially in the palms and soles. It may be added that, in syphilitic lepra, the formation of scales is usually much more scanty than in the non-specific variety of the disease; that it is almost impossible to make any real distinction between the several syphilitic affections of the palms and soles, inasmuch as all are generally attended with desquamation; and that scabs, due apparently to the interstitial effusion of serum or pus, sometimes form on leprous or tubercular patches, which thus pass by easy gradations into the truly vesicular and pustular conditions.

The morbid processes of the mucous membranes first show themselves in the fauces and pharynx, generally upon the tonsils. On the last, which then present an inflammatory blush, shallow ulcers, for the most part reniform in shape, make their appearance; they are generally unattended with pain or even uneasiness, and disappear after a few weeks. Similar sores are also apt to form on the palate and internal surface of the cheeks, on the tongue and lips. In addition, condylomata or mucous tubercles often become developed in the mouth, fauces and pharynx, about the anus, upon the mucous or delicate cuticular surface of the genital organs, and in those parts of the skin which are in constant apposition and consequently always moist. Mucous tubercles are roundish, oval, or more or less irregular congested tabular elevations, not fibrous, warty, or villous, but uniform in texture and soft, with a tendency to be covered with a greyish or yellowish film, to exude abundant moisture, to secrete pus, or to undergo ulceration.

Similar formations may arise in the rectum, œsophagus, nose, larynx, trachea, and bronchial tubes, and not improbably in other parts of the mucous tracts.

The pains in the joints and bones are of a rheumatic character, and are apt to become especially severe at night. There may be no visible change in the parts affected; sometimes, however, there is more or less obvious periostitis, or arthritis and effusion into the joints. True nodes are comparatively seldom developed at this time.

The affections of the eyes are twofold. The most obvious is a form of iritis, attended with little pain, uneasiness, or intolerance of light, but with more or less of the ordinary form of sclerotic injection which accompanies iritis. Exudation of rust-coloured lymph occurs at the surface of the iris, mainly, however, at its inner margin, and, though much less frequently, at its outer margin. In the former situation the lymph may form a uniform tumid ring, and in either situation a series of reddish beads. At the same time the iris becomes sluggish or immovable, and probably adheres to the surface of the lens, and the aqueous humour gets turbid and yellowish from admixture with inflammatory products. The less obvious but more serious form is retinitis, coming on insidiously without external congestion, pain, or intolerance of light, but marked by increasing haziness of vision and indications of retinal congestion and extravasation.

3. *Tertiary symptoms.*—The period of secondary symptoms, after lasting for a few weeks or months, for the most part terminates in spontaneous convalescence; and the patient may possibly remain henceforward free from disease. But more commonly, after the lapse of an uncertain period, generally from six months to two years, but sometimes twenty years or more, other characteristic lesions manifest themselves, distinctly referrible to the syphilitic poison, and usually termed tertiary symptoms. It must however, be added that, although there is generally a distinct interval between the subsidence of the secondary and the onset of the tertiary symptoms, they do occasionally, and perhaps not unfrequently, become intermingled, or pass without break one into the other. The chief characters by which tertiary symptoms are distinguishable from those of earlier occurrence are, first, their dependence, for the most part, on a specific overgrowth of tissue —the formation of gummata; second, their great inveteracy and tendency to recur; third, their involvement of internal organs as well as of parts that are superficial; and fourth, their want of symmetry. We will consider the more important of these lesions *seriatim*, and in reference to the organs which they affect.

a. Skin.—The most common form of skin disease is characterised by the appearance of dusky red or coppery flat tubercles, which differ little if at all, in the first instance, from those which have been described among the secondary symptoms; they are, however, generally larger, more prominent and more indurated, and occur sometimes

widely scattered, sometimes collected into irregular groups, sometimes arranged in the form of crescents, circles, or sinuous lines. In the first case they gradually increase in number, and coalesce; in the second, the groups tend to grow in area, and not unfrequently also in thickness, so as to form irregular tuberculated elevations of considerable extent and thickness; in the last case the affection tends to spread centrifugally, slowly invading the healthy surface by a line of ever new tubercles, while the parts primarily affected return to a state of comparative health. In some instances the tubercles become scaly on the surface, the affection presenting a certain amount of resemblance to some forms of lepra; in other instances they undergo superficial molecular necrosis, they get more or less deeply eroded, and a scab forms without a vesicle or pustule having ever been developed; in other cases each papule undergoes suppuration, and a thick adherent ecthymatous scab results; and, lastly, ulceration is not unfrequent. But whichever of these processes takes place, the disappearance of the active lesion is always followed by the formation of indelible depressed cicatrices; and the progress of the serpiginous form can always be traced by the cicatricial surface which it leaves in its wake, and the pre-existence of large patches always recognised by the persistence of a corresponding cicatrix. The tubercular eruptions here described, although essentially identical with one another, are often denominated, according as one or other peculiarity predominates, tubercular, pustular, or serpiginous syphilide, or syphilitic lupus, or psoriasis. They affect almost any part of the body, but are perhaps especially common on the face, neck and shoulders, buttocks and extremities. When they occur, as they frequently do, on the palms and soles, there is little to distinguish them from secondary lepra. Another well-marked form of skin-affection is that known by the name of syphilitic rupia. It consists in the scanty formation, indiscriminately on various parts of the body, of isolated blebs, each of which arises on a congested indurated base, and may attain the diameter of a fourpennypiece. Their contents are clear and limpid, or turbid and sanious, and soon concrete into scabs, each of which, from constant additions to its edges and base, rapidly attains large dimensions—a thickness, for example, varying from a quarter to three-quarters of an inch, and a form which may resemble that of a limpet-shell or oyster-shell, or may be merely irregular and rocky. The base at the same time becomes deeply excavated; and on removing the scab a deep unhealthy slowly healing ulcer is revealed. These rupial sores leave remarkably deep cicatrices.

Very frequently, altogether independently of any primary cutaneous disorder, hard nodules, from the size of a pea to that of a filbert, appear singly or in groups in the substance of the subcutaneous connective tissue. They are unattended with pain, and very slow in their progress; but after a while they adhere to the skin, which then becomes somewhat prominent over them, assumes a dusky red tint, and gives

to the finger a sensation of elasticity and resistance, or 'bogginess.' Before long the central portion of the involved skin becomes perforated in one or more points, and a viscid, turbid, or sanious fluid escapes, together with shreds or a mass of subcutaneous slough. In this way a deep cavity results, the boundaries of which are formed of ragged greyish or yellowish tissue. If groups of such masses soften, we get a number of such cavities side by side, the skin appears irregularly honeycombed, and the bridles which intervene between the adjoining openings get undermined by the coalescence beneath them of the contiguous cavities. Thus extensive and deep destruction of skin and subcutaneous connective tissue takes place, which is very slow of repair, and followed by deep cicatrices.

b. Mucous membranes.—The affections of the mucous surfaces have much resemblance to those of the skin. They are chiefly superficial and tubercular, or sub-mucous and gummatous. The former are especially frequent on the tonsils, fauces, soft palate, pharynx, tongue and other parts of the mucous surface of the oral cavity, and in the larynx; are followed by deep unsymmetrical and obstinate ulceration; and lead to extensive destruction with permanent loss of tissue and contraction. Thus, the uvula and soft palate may be more or less perfectly destroyed; the isthmus of the fauces may be narrowed; stricture of the œsophagus may ensue; or destruction of the epiglottis, vocal cords, or other parts of the cartilaginous skeleton of the air-passages may take place; and, following upon these several lesions, loss of voice, difficulty of swallowing or breathing, and other serious or fatal consequences. Gummatous tumours also appear in the same parts, frequently in the tongue, where they may attain the size of a hen's egg, and sometimes in the connective tissue and muscles of the larynx. These not unfrequently assume many of the superficial characters of epithelioma, and undergo the same processes as do subcutaneous gummata.

Similar affections to the above take place in the male urethra and in the vagina and os uteri, as well as in the external parts of the organs of generation of both sexes; they also occur within the anus and in the lower part of the rectum; and may, in all of these situations, in addition to other forms of mischief, lead ultimately to more or less serious contraction or stricture.

c. Organs of locomotion.—Voluntary muscles are occasionally affected in the same way as the subcutaneous connective tissue. Gummata invade their texture, separating from one another their fibres, which then undergo degeneration. Such growths occur quite irregularly and may be mistaken for tumours of a far more serious character. We have already pointed out that they may form among the muscles of the larynx; they have also been observed implicating the masseter, the muscles of the scapula, and indeed those of most other parts. The bones are sometimes affected with diffused periostitis; but

more commonly nodes are developed on various parts of the long or flat bones, including the ribs, sternum, and bones of the face and skull. Nodes are gummatous growths in connection chiefly with the periosteum. They are usually extremely painful and tender, of various extent and prominence, more or less hard and unyielding at the periphery, but elastic or even fluctuating in the centre. They seldom end in suppuration, and generally on healing leave some irregularity behind. Nodes do not usually result in caries or necrosis. A more frequent cause of these conditions is the extension of syphilitic ulceration in depth until subjacent bone is involved. But from one or other of these causes caries or necrosis may attack any bone; the bones most frequently thus affected, however, are those of the nose, palate, and skull, to which may be added the cartilages of the larynx and trachea. The bones of the ear also may suffer. Syphilitic affections of the skull are generally limited to the outer surface and diploe, but occasionally involve the inner table as well, and are then apt to cause more or less serious cerebral symptoms. The joints occasionally suffer, the surrounding soft parts becoming thickened and infiltrated, and the cavities distended with fluid effusion.

d. Viscera.—The affections of the internal organs are scarcely so well known as those of the parts which have already been considered, but they are even more serious. The liver is perhaps their most frequent seat. The chief conditions which have been recognised here are, first, a more or less general hyperplasia of the connective tissue, especially of the capsule of Glisson, leading to a variety of cirrhosis; and, second, the formation of gummy tumours which rapidly undergo degeneration, and by their contraction cause puckering and fissuring of the surface of the organ. Either of these conditions may lead to the development of symptoms identical with those resulting from ordinary cirrhosis. The organs of circulation also are frequently implicated. The muscular tissue of the heart is occasionally the seat of diffuse fibroid infiltration or of more or less extensive gummatous formations, exactly like those involving the voluntary muscles. These induce degeneration of the tissue, induration, adhesion of pericardium, and the ordinary symptoms of progressive cardiac incompetence. There is good reason also to believe that some forms of arterial disease, and especially that form in which the inner coat undergoes a kind of nodular hypertrophy antecedent to the supervention of degenerative changes, are in many cases the result of syphilis. It is at all events certain that arteries frequently assume this condition in those who are the subjects of syphilis, and who are suffering from gummatous tumours in other organs. And it is also certain that some of the lesions observed in the brains of syphilitic patients are essentially due to arterial changes of this kind, involving however not only the internal coat, but the adventitia, and to some extent also the middle coat, and leading to obstruction either directly or by thrombosis.

Syphilitic affections of the lungs (gummata and fibroid infiltration) are described; nevertheless, their recognition is attended with much uncertainty. We have already adverted to the fact that the bronchial tubes, like the larynx, may be distinctly implicated. But, besides bronchial lesions, there are not unfrequently found in the lungs of old syphilitic patients scattered masses of hard greyish or blackish fibroid induration, or caseous masses imbedded in such tracts of induration, which, although in many particulars resembling affections of tubercular or inflammatory origin, are almost certainly gummata. The most grave of all tertiary syphilitic affections are those which involve the nervous centres. Gummatous tumours are developed in connection sometimes with the inner layer of the dura mater, sometimes with the pia mater, or the connective tissue of the brain-substance. In the latter two cases the growths, which may attain the size of a pigeon's egg or even of a hen's egg, are, even if of peripheral origin, for the most part imbedded in the substance of the brain. Their most frequent site is the basal portion. Similar growths occur, though much less frequently, in connection with the spinal cord. The symptoms due to them are those of cerebral or spinal tumours. The cranial nerves and even the brain-substance are occasionally the seats of syphilitic infiltration. Specific affections of the kidneys have been less thoroughly investigated; nevertheless, it is certain that these organs are sometimes affected, sequentially to syphilis, with diffused inflammatory processes, which induce atrophy, and that they are sometimes studded with distinct gummata or with patches of cicatricial tissue, attended with corresponding linear or stellate contractions of the surface and having imbedded in them small caseous masses. The testes are frequent seats of gummata, and also of diffused inflammatory processes. They are apt to become much enlarged; occasionally suppurate; and not unfrequently are associated with hydrocele. The lymphatic glands, as has already been pointed out, get enlarged and indurated secondarily to local syphilitic lesions; but occasionally, here and there in groups, they acquire such enormous dimensions as to simulate the enlargement of these organs due to scrofula, lymphadenoma, or cancer. Among other organs liable to syphilitic disease may be enumerated, the spleen, stomach and bowels, mammæ and organs of special sense.

The effects of syphilis do not end here. The long persistence of tertiary symptoms, with their frequent tendency to relapse, leads gradually but surely to a marked cachectic state of the system, indicated by sallowness and anæmia, with relative increase of white corpuscles, emaciation and loss of strength, and lardaceous or amyloid degeneration of the liver, spleen, kidneys, and other parts, together with the additional symptoms to which such complications give rise. And finally may follow tuberculosis, or insidious but non-specific inflammations of various internal organs.

4. *Inherited syphilis* presents some peculiarities which make it

necessary to give the subject a brief separate consideration. It may be derived from either the father or the mother, or from both. The effects of parental syphilis are not unfrequently manifested in the death of the fœtus, and consequent abortion, at the latter period of pregnancy. The child is born dead, and more or less decomposed, but usually without distinct evidence of specific taint. In some instances, however, the placenta is affected with syphilitic disease. In other cases the infant is born alive, but shrivelled, puny, and unhealthy-looking; and large bullæ appear on the palms, or wrists, or the corresponding parts of the lower extremities. These blebs give rise to unhealthy sores, and the infant almost invariably dies speedily. In the majority of cases, however, the babe appears to be healthy at birth, and first gives evidence of disease after an interval of three or four weeks. The symptoms are mainly those of the secondary period of acquired syphilis; but there are some features which are specially characteristic and important, and to these alone attention will now be drawn. Among the earliest of these are, congestion and swelling of the nasal mucous membrane, with abundant secretion, giving rise to snuffles and other symptoms of chronic coryza; diffuse inflammation of the mouth and fauces, with sores at the angles of the mouth; mucous tubercles about the anus and similarly constituted parts; and a roseolous rash. The rash is generally most abundant on the buttocks, privates, and neighbouring parts of the abdomen and thighs, on the face, and on the palms and soles. It consists in circular patches from a line to half an inch in diameter, which either form lenticular elevations, or are slightly concave or cupped, and present therefore a more or less tumid marginal ring. They vary in colour, are sometimes dusky red, sometimes brown or yellow, sometimes of a more or less coppery tint. They may be smooth or scaly, or may present superficial excoriation or erosion. The eruption on the palms and soles assumes a scaly character, and is attended with a tendency to crack and exfoliate. In association with the above phenomena the child becomes emaciated, its face assumes an old and weird character, its complexion grows sallow and unhealthy-looking, its skin dry and shrivelled, its hair scanty and thin, and not unfrequently it suffers from diarrhœa. These symptoms last probably for a few months, and have generally disappeared by the end of the first year. Somewhat later, generally from the age of four or five up to that of puberty, the tertiary series of symptoms manifest themselves. These differ little from those which characterise the common tertiary stage. There are two or three however of peculiar and special interest, which now become apparent or develop themselves for the first time. One of them is flattening of the bridge of the nose, from sinking in of the subjacent cartilages. Another is enlargement of the lower end of the humerus between the epiphysis and shaft. A third is a peculiar form of atrophy of the permanent incisor and canine teeth. This is generally most marked in the upper two central incisors, and is often

limited to them; they are atrophied, peg-like, and present towards their free edge a reniform or cordate character—the notch occupying the centre of this edge. This condition is traceable to the effect, on the tooth-germs, of the stomatitis from which the children have previously suffered. The last of them is interstitial keratitis, that is, an interstitial inflammation of the cornea, marked by increasing cloudiness and opacity of the part, and attended with a vascular zone in the sclerotic, and more or less intolerance of light. There is no vesication or ulceration, and the opacity speedily diminishes under appropriate treatment. The recognition of the last two affections is due to Mr. J. Hutchinson.

It would be impossible in a brief space to discuss the differential diagnosis of syphilis. It must be sufficient to say that the manifestations of syphilis simulate a vast range of different diseases; and further that syphilis necessarily often occurs in persons who are the subjects of skin-affections and various other disorders, and often exerts a modifying influence over these; and that hence it is frequently quite impossible to form an exact diagnosis, without going fully into the history of the patient's case, and taking into careful consideration all the facts of his past history and present condition.

Morbid anatomy and pathology.—In the foregoing description of the phenomena and sequelæ of syphilis we have necessarily, to a large extent, discussed the pathological processes of the disease and its morbid anatomy. It remains, however, to give a brief connected account of these subjects. The morbid poison which enters the system at the time of inoculation is doubtless a living entity or contagium, which imparts specific properties, primarily to the growth which it directly induces, and secondarily to the vital constituents of the enlarging lymphatic glands situated next above that growth. Possibly from the primary sore, more probably, however, from the group of morbid lymphatic glands, as a centre, is shed into the blood-stream newly manufactured contagious matter (probably particulate); which in its turn infects in different proportions and in different order the various organs and tissues of the body, producing in them specific processes which have a more or less close resemblance to those out of which they arose, and which like them are infectious certainly to other persons, and probably like carcinoma to the individual. The early series of general phenomena (those for the most part which belong to the period of secondary symptoms) differ scarcely at all in their anatomical characters and in their local results from simple inflammatory processes. There is congestion, proliferation of connective-tissue imitative of granulation-tissue, and a tendency in the new formation, after a temporary persistence, to subside altogether so as to leave no trace whatever behind, or to merge into the tissues, in relation with which it appears—if in relation with connective-tissue into connective-tissue, if in relation with bone into bone, if in the matrix of the liver, kidney,

lung, testicle or brain, into nucleated fibrous tissue and to produce therefore in these organs induration, contraction, and atrophy. The later phenomena (those which belong chiefly to the tertiary stage) consist in the formation of adventitious growths, termed gummata, which are identical in structure with primary chancres and the primarily indurated glands. They consist like them of cell-growth, differing little microscopically from ordinary granulation-tissue, and in this respect therefore little from the secondary lesions, but presenting certain special features. Thus they do not so much displace as infiltrate or involve the tissues among which they arise; they have a remarkable tendency to undergo speedy caseous degeneration, and to cause molecular or fatty disintegration of the higher elements which are mixed up with them; if developed in internal organs, they acquire for the most part permanence as either caseous lumps, earthy concretions, indurated fibrous patches, or morbid tracts in which all of these conditions are variously combined; and if they be developed in superficial parts, such as the skin, mucous membranes or superficial bones, their degeneration results in the formation of crusts, ulcers, abscesses or sloughs, with more or less serious destruction of tissue. Gummatous tumours, while in process of development, vary in their physical characters; thus, sometimes they are greyish, firm, and translucent or opaque; sometimes (especially when they form beneath the skin and mucous membranes) they are infiltrated with a mucus-like fluid, which oozes away when they are laid open.

There is undoubtedly a resemblance both anatomically and functionally (at all events as regards their infectiveness) in all the congestive or proliferating lesions which depend for their origin on the syphilitic virus. And although a line may be drawn, both on clinical grounds and for the purposes of description, between secondary and tertiary phenomena, and although it is quite true that the later lesions are far more serious and virulent than those which precede them, there is no doubt that they pass one into the other, that they shade off the one into the other by numerous gradations, and that they are often blended; so that while, on the one hand, gummatous tumours may occur during the secondary period, secondary eruptions may be met with late in the progress of the disease.

Treatment.—For the prophylactic treatment of syphilis, and for the treatment of the primary affection reference must be made to surgical works. The inoculation of syphilis upon healthy persons, which has been so extensively practised by Boeck, and advocated by others, in order that by giving them the disease it might affect them in a mild form and prevent any future attack in a graver form, seems to us, we confess, not only dangerous but altogether unjustifiable. It is now generally admitted that syphilis, like other specific febrile diseases, is incapable of absolute cure, and that it will run a definite course in respect of duration, no matter what steps are taken to arrest its progress.

It is nevertheless certain that we have at least two remedies which exert a remarkable influence over its various localised manifestations, which subdue them almost to zero if they do not absolutely annul them, and which keep the general disease in abeyance even if they fail (as they probably do) to extinguish it altogether. These remedies are mercury and iodine in their various preparations. The value of mercury was early established, and has indeed only lately been regarded with suspicion. But this suspicion arose doubtless out of the injurious influences which the abuse of mercury engendered during the earlier part of this century, and was supported by the recognition of the fact that the free use of mercury failed in many cases to prevent the supervention of secondary symptoms. It is admitted now that mercury does not prevent either secondary or tertiary symptoms from coming on; but nevertheless it is certain that it has a marvellous influence in causing the removal, in turn, of the primary, secondary and tertiary lesions of the disease. The form, the dose, the mode, and the length of time in which the drug should be administered are points on which there is much difference of opinion. Some prefer to introduce it by the inunction of strong mercurial ointment on the inner aspect of the thigh or other parts in which the integuments are thin: in this case from half a drachm to a drachm of the ointment may be rubbed in every night before the fire. Others affect the practice of fumigation by means of volatilising calomel with the heat of boiling water. The drug may by this mean be inhaled, or applied to the general surface, or to particular regions, with little difficulty. For inhalation, not more than four or five grains of calomel should be employed. Others again recommend that the mercury should be administered by the mouth. For this purpose any mercurial preparation in an appropriate dose is applicable. But the most convenient, and possibly the best, is corrosive sublimate or the red iodide of mercury, of which from $\frac{1}{20}$ to $\frac{1}{8}$ gr. may be given three times a day, or equivalent doses of the *liquor hydrargyri perchloridi*. The treatment should be continued until the lesions have disappeared under its influence, and even for a week or two longer, and the quantity should be regulated by its effects on the system, slight soreness of the gums only being maintained. Iodine is almost equally valuable with mercury; but it seems to have a special value during the later periods of the disease, in which (when the lesions prove intractable) it may often be beneficially combined with the mercurial treatment. The usual, and probably on the whole the best, form is the iodide of potassium in from 5 to 10 grain doses three times a day, combined with a tonic. The syrup of the iodide of iron is very valuable in many cases, and especially for young children. Bromine has an antisyphilitic power similar to that of iodine, and is sometimes substituted for it. Among other anti-venereal remedies which have acquired and still enjoy a wide reputation are sarsaparilla, and nitric acid in large doses. They have probably no specific virtues

at all. Tonic medicines—quinine, iron, cod-liver oil and the like—are often of immense value in the treatment of the cachexia which attends the later stages of the disease. The value of local applications to syphilitic lesions is undoubted: the most important of these are the mercurial and the iodic, among which may be enumerated powdered calomel, black-wash, mercurial, citrine or iodine ointment, iodide of starch paste, and localised calomel fumigations—one or other being employed according to the nature of the lesion, and the convenience and relative safety of its applicability.

XXIII. PYÆMIA. (*Septicæmia.*)

Definition.—By the term 'pyæmia' is understood a febrile and generally acute disorder, due to the entrance into the blood of certain poisonous or septic, for the most part inflammatory, products, and usually characterised by the blocking up by clots or emboli of the arterioles of the lungs and other organs, and the consequent occurrence therein of scattered patches of congestion, hemorrhage, inflammation, suppuration or gangrene.

Causation.—The conditions out of which pyæmia arise are very numerous and various. First, it is a frequent sequela of accidental injuries, such as burns, scalds, bruises and lacerations, and compound fractures, especially of the long bones, and of the bones of the head and pelvis. Second, it frequently ensues on surgical operations, especially those which are attended with the formation of extensive raw surfaces, such as amputations of the larger limbs, and also those involving bone, bladder, prostate, urethra or rectum. To these must be added operations on veins, such as phlebotomy and operations for the cure of varicose veins and hemorrhoids. Third, pyæmia occurring after parturition constitutes one of the most common and fatal forms of so-called 'puerperal fever.' Fourth, pyæmia not very unfrequently originates in acute suppurative inflammation taking place at the surface or in the substance of bones—cases in which, as a rule, the periosteum becomes extensively detached, and the bone necrotic. Fifth, many varieties of so-called unhealthy inflammation, such as erysipelas, diffuse cellular inflammation, carbuncle, dissection-wounds, and malignant pustule are often fatal on account of the supervention of this complication. It may be added that when pyæmia manifests itself after injuries or operations, it is generally preceded by some obviously unhealthy condition of the implicated tissues; and also that pyæmia is far more liable to originate in affections of certain organs and tissues than in those of others. Among the former may be included the connective tissue generally, the bones, the pelvic organs both in the male and female, and the veins. That pyæmia is in a large

number of cases imparted by contagion is quite beyond dispute. It is thus that it often spreads in the surgical wards of a hospital, and among the puerperal inmates of a lying-in institution. In all such cases there is good reason to believe that it is transmitted from patient to patient by direct inoculation at the raw surfaces of wounds or of the placental area; or rather that, not so much pyæmia is transmitted directly from patient to patient, as some form of erysipelatous or other unhealthy inflammation is thus transmitted, of which pyæmia is a common accident. It is certain, on the other hand, that even when it complicates the puerperal state and surgical wounds, it often arises, so far as we can discover, *de novo*, and altogether independently of specific influences. There can be little doubt that it is very frequently, if not always, of idiopathic origin when it occurs without breach of surface. It still remains to consider whether there are any special conditions of system and of a patient's surroundings which render him peculiarly liable to become pyæmic. In reference to this point it may be remarked that age and sex have no distinct influence; that patients apparently in the best of health are often struck down with pyæmia; and indeed, that when pyæmia pervades a ward, it by no means selects the weakly and the cachectic in preference to the robust and healthy-looking; and, again, that it does not arise with special frequency in connection with either simple overcrowding, bad ventilation, or common filth.

Morbid anatomy and pathology.—The post-mortem phenomena which characterise the presence of pyæmia are (as stated in our definition of the disease) patches of congestion, hemorrhage, inflammation, suppuration or gangrene, disseminated more or less abundantly throughout the organs and tissues of the body. These are most common in the lungs, and are often confined to them. We find here, irregularly scattered but mostly abutting on the surface, circumscribed patches ranging from the size of a pea to that of a walnut. These are sometimes distinctly apoplectic, in which case they may be reddish black or more or less decolourised, solid or partly broken down into a puriform pulp; sometimes they present the ordinary characters of lobular pneumonia; sometimes they are simple abscesses or gangrenous cavities. These different characters depend in part no doubt on the stage at which death has taken place, but are often due to individual peculiarities. There is usually more or less congestion and œdema of the general lung-tissue, and occasionally diffused pneumonic consolidation and secretion of mucus into the bronchial tubes. There is probably always a deposit of pleural lymph over and around each pyæmic lump which involves the surface of the lung; and not unfrequently general pleurisy ensues. Sub-pleural petechiæ are common. The surface of the heart, like that of the lungs, is often studded with small extravasations of blood; as also are the substance of the cardiac walls, and the sub-endocardial tissue. And sometimes, generally in relation with these extravasations,

small yellowish patches of disintegrating tissue or abscesses may be discovered. When these reach the inner or outer surface of the heart they are apt to provoke inflammation of that surface. Neither pericarditis nor endocarditis is of rare occurrence. Of the abdominal organs, the liver, spleen, and kidneys most frequently suffer. In the liver, generally in connection with patches of congestion or of anæmia, we sometimes find small buff-coloured spots of disintegrated tissue, sometimes abscesses of considerable size full of greenish purulent fluid. The morbid conditions presented by the spleen and kidneys are almost exactly such as are met with in embolism or thrombosis of the vessels of these organs. In the spleen we observe apoplectic or fibrinous blocks of various sizes, which have often undergone more or less disintegration and softening, or even conversion into abscesses. The kidneys are sometimes studded (chiefly in the cortex) with small abscesses, grouped for the most part in lines perpendicular to the surface and surrounded by a halo of congestion. Occasionally no abscesses have formed, but almost the whole of their tissue is mapped out by tracts and bands of deep congestion, which alternate with and surround patches of which the colour is unnaturally pale. Spots of hemorrhage, patches of inflammation, or small abscesses may be present in any other of the abdominal organs, sometimes, for example, in the intestinal wall; and the peritoneum may be affected exactly in the same way as the pleuræ and pericardium. The brain is not very commonly the seat of pyæmic changes; extravasations of blood are generally small in amount and limited to the surface; patches of softening exactly like those due to embolism, excepting that they rarely exceed the size of a horse-bean, may occur in any part of the organ; abscesses containing greenish-yellow glairy pus attain a much larger size. Meningitis also occurs. The bones and joints are frequently involved. The secondarily affected bones rapidly become denuded of periosteum, and fetid pus accumulates upon their surface and probably in their substance, and rapid necrosis ensues. The synovial fringes of the joints get intensely congested, and the synovia increased in quantity or replaced by pus or puriform fluid. The cavities of the joints become distended, and the parts around inflamed. It is important to bear in mind that suppuration may occur in the neighbourhood of joints without involving them, and that pyæmic inflammation of joints is not always suppurative. It must be added to the foregoing account that secondary inflammations, suppurative or not, frequently manifest themselves in the connective tissue and among the muscles; and that, of organs which have not been specially named, the eye, the prostate, and the testis are very apt to suffer. The skin never presents any characteristic change; but it is often slightly jaundiced, and occasionally presents petechiæ; sudamina are common.

The condition of the blood and blood-vessels in pyæmia is a matter of great interest. The bulk of the circulating fluid, and the vessels

in the greater part of their extent, have usually the aspect of perfect health. The coagula, indeed, which are found post mortem in the heart and larger vessels usually differ in nothing from coagula found under other circumstances; very rarely a few soft masses of disintegrated fibrine or of corpuscles resembling pus may be found imbedded in them. But, with this exception, it is only in the arteries which lead to the secondary morbid patches, and in the veins involved in the primary lesion, that visible morbid phenomena are present. The minute arteries distributed to each patch of pulmonary disease are always found filled and obstructed either with ordinary thrombi or with a soft yellowish material, consisting of disintegrated fibrine and corpuscles, or in some cases of these mingled with what appear to be groups of pus-cells. Similar coagula have been detected in the small vessels leading to the diseased patches occurring in the heart, spleen, and kidneys, and doubtless are always present in the arteries which are connected with the generalised pyæmic lesions. The veins which are involved in the primary inflammatory process are in a very large proportion of cases obviously diseased. It is true that, even after careful dissection, they have in some cases appeared to be entirely healthy. But when we bear in mind that in other cases the presence of diseased veins has only been detected after some hours of minute investigation, we shall see reason to suspect their existence in cases where they have been reported to be absent. When diseased, their parietes are thickened and indurated; they may be entire, or may communicate by orifices resulting from ulceration or some other cause with the morbific elements in which they are imbedded; and their interior is occupied by coagula. These are mostly adherent, and more or less decolourised; they may be solid throughout, but more commonly are reduced in their interior into a reddish or yellowish pus-like pulp or fluid. This appears generally to consist of disintegrated fibrine, but is in some cases true pus. It is mostly separated from the venous walls by a layer of fibrine, and shut out from the proximal portion of the venous channel wherein it lies by a continuation of this layer of fibrine, which forms a kind of diaphragm or septum between them. In some cases no mechanical impediment whatever exists to prevent the free admixture of the pus contained in the vein with the general circulation.

We are now in a position to discuss the proximate cause of pyæmia. It was formerly supposed that the secondary inflammatory patches were mere deposits of pus which had been absorbed as such by the veins and carried to the localities in which abscesses were found. But, unfortunately for this view, pus as such is not found to circulate with the blood, and the secondary patches of disease are never in the first instance, and not often at any time, distinctly purulent. The theory of embolism, however, here comes to our aid. There is no doubt that the secondary foci of disease are almost exactly such as would be produced by embolism of the arteries leading to them; and we find, in fact, that these

arteries are really plugged. But we find, further, that these plugs are identical in composition and appearance with the coagulated material which blocks up the veins of the primarily inflamed region. It is reasonable, therefore, to assume that the diseased veins are really the sources of emboli, which, becoming impacted in the pulmonary arteries, induce characteristic changes in the parts beyond, and that the phenomena of pyæmia are, therefore, in large measure due to the dissemination in pellets of the morbid matters—pus, disintegrated clot, and the like—which these veins contain. In favour of this view are the facts that such pellets have been recognised *in transitu*, and that pyæmia is especially liable to occur where veins have been the subject of operation, and where inflammation attacks parts in which the veins are abundant and large, thin-walled, or incapable of collapsing—such parts, for example, as the contents of the pelvis, the uterus after parturition, the cancellous structure of bones, and the meninges of the brain. There can be little doubt indeed of the correctness of the above explanation, so far as it goes, and little doubt also that the quality of the emboli has a marked influence over the quality of the processes which they induce; and that hence whether these latter be gangrenous, suppurative, or simply inflammatory, depends in no small degree on the special nature of the process going on in the primary seats of disease. But will embolism alone explain all the phenomena of pyæmia? To this question Virchow replies in the negative. He considers pyæmia to be a twofold disease, comprising, in the first place, phenomena due to embolism, and, in the second place, phenomena due to the absorption of some more subtle poison. These latter, which he regards as the more important, have been collectively termed '*septicæmia*,' and he considers that these two groups of phenomena may occur independently of one another. A very strong argument in favour of this view is the fact that patients occasionally die with many of the ordinary symptoms of pyæmia, arising from some unhealthy wound, in whom no morbid conditions whatever can be discovered post mortem, save congestion of various internal organs, small extravasations of blood beneath the serous membranes and elsewhere, and tendency to rapid decomposition. Such cases, which are very acute in their progress, are not unfrequently met with in the course of the endemic prevalence of pyæmia, and are regarded by many as cases in which death has supervened before the specific lesions have had time to develop themselves. The discovery of bacteria in the blood of pyæmic patients is a matter of interest, and (although they are also discoverable in the blood of patients who are not pyæmic and never become so) the interest attaching to their presence is greatly enhanced by the observations already referred to (p. 136), which seem to prove that when they swarm in a putrefying surface they evolve a poison which is capable of absorption and of producing symptoms having some resemblance to those of pyæmia. So far, then, as our present knowledge goes, pyæmia (using this term in

its widest sense) seems to be due to the introduction into the circulating blood, through the medium of certain implicated vessels, of showers, so to speak, of septic products, these being partly solid, in part probably fluid, and charged more or less distinctly with the special properties of the local inflammation or process which gave them origin. The presence of these in the blood causes in the first instance, partly by embolism, partly by thrombosis, obstruction of the pulmonary arteries with certain characteristic lesions in the lungs, and at a later period obstruction of various of the systemic arterioles with similar characteristic lesions in the districts which they supply. It imparts also specific poisonous properties to the blood. It is easy to understand from this view how it is that, when pyæmic processes are present, the lungs are, as a rule, affected both earlier and more extensively than other organs.

Symptoms and progress.—The symptoms which usher in an attack of pyæmia are generally well-marked, unless the condition of the patient or the nature of the disease under which he is labouring at the time confuses them. The first symptom to attract attention is almost without exception a sudden, severe, and prolonged rigor, followed by profuse perspiration. The patient may recover from this, and for a time appear to be restored to health. But before long, it may be the next day, or at some earlier period, the rigor returns with its after sweating stage; and again and again, at varying intervals, rigors and sweats recur. In the course of a day or two the conjunctivæ and skin assume a sallow tinge; the patient becomes dull and heavy, or it may be restless, and acquires very much the manner and aspect of a person suffering from some form of continued fever. In company with the above symptoms, or in succession to them, others of more or less importance show themselves. The pulse becomes rapid, weak, and perhaps intermittent. The tongue becomes glazed and fissured, or coated, and after a time dry and brown, the lips parched, the teeth covered with sordes. The patient is thirsty, loses his appetite, suffers often from nausea and vomiting, and not unfrequently from diarrhœa. The respirations become shallow and frequent, and the respiratory acts attended with dilatation of the nares or separation of the lips, and either a sniffing, sipping or sucking sound. Cough often supervenes, attended probably with pains in the chest, and evidences of pleurisy or consolidation of the lungs, and of excessive secretion into the bronchial tubes. The skin, in the intervals between the perspirations and rigors, is often dry and harsh, and may present sudamina. The sallowness generally increases, and often amounts before death to well-marked jaundice. Pain and swelling in or around joints, or in other parts of the connective tissue, often present themselves, and pus may form rapidly in these situations. As the disease advances the patient becomes excessively prostrate, his face shrunken, and for the most part pale, his mental functions disturbed; slight delirium comes on,

sometimes coma, sometimes convulsions; and death ensues usually in from four to ten days. Sometimes pyæmia takes a more chronic course: the symptoms are then altogether less strongly pronounced, the fever assumes the characters of hectic fever, abscesses form in the joints and other superficial parts, and the patient sinks from exhaustion at the end of a few weeks or even a few months, or in rare cases recovers after a protracted convalescence.

We will consider some of the symptoms of pyæmia more in detail.

The patient's aspect may at first be healthy-looking or nearly so, but soon becomes dull and oppressed. The face is sometimes flushed, sometimes pallid, and often these conditions alternate. Towards the close of the disease pallor generally becomes established, and the countenance shrunken and anxious, or of that dull expressionless aspect which is common in the last stages of many febrile disorders. Rigors, though occasionally absent, constitute one of the most striking phenomena of pyæmia. They vary in number and frequency, sometimes recurring at short and irregular intervals, sometimes assuming a quotidian character, and generally ceasing after the first two or three days. Their duration ranges from a few minutes up to half an hour. The temperature of pyæmia has a good deal of resemblance to that of ague; the rigors are always attended with a rapid rise, which is followed by an almost equally rapid fall. During, or after, the first rigor, the temperature may reach 104°, 105°, or even 107°, or more, and the subsequent fall carries it down probably to a little above the normal, occasionally even below it. Subsequently, according to circumstances, the temperature may present a succession of similar elevations and depressions, or maintain a nearly uniform level. Death may be preceded by either a normal, a low, or even a very high temperature. The skin, which is often harsh and dry, perspires profusely after the rigors, and copious perspirations recur from time to time during the progress of the malady, and attend its last stage.

The respiratory acts, as the disease advances, become frequent, independently of the presence of pulmonary complication, and not uncommonly reach 40, 50, or 60 in the minute; and the breath is said to acquire a peculiar sweet odour. The pleuritic exudation, the pulmonary lesions, and the excessive formation of bronchial mucus, may each or all aggravate the symptoms due to the respiratory organs; and may induce dyspnœa, cough with various forms of expectoration, pleuritic stitches, and friction, crepitation, rhonchus, or other auscultatory phenomena.

The feebleness of the pyæmic pulse is remarkable. It is generally rapid from the beginning, or, if not rapid, variable, so that the slightest exertion of body or mind raises it 20, 30, or even 40 beats in the minute. As the disease advances, it frequently rises to 140 or 160 in the minute, and may even reach 200; it then tends to become irre-

gular and almost imperceptible. It is possible that pericardial friction or other signs of cardiac implication may be present.

Abdominal pain and tenderness may be caused by the presence of hepatic or splenic congestion or inflammation, or of circumscribed peritonitis in connection therewith. The jaundice, which is so common in pyæmia, appears to be quite independent of the presence of pyæmic deposits and abscesses in the liver. Frerichs remarks that, 'to all appearance the jaundice is here the result of an impaired consumption of bile in the blood, arising from an abnormal condition of the metamorphic processes which go on in that fluid.'

Urea is largely increased, and often the urine contains a small quantity of albumen.

Arthritic and other superficial abscesses are far more common in the chronic than in the acute form of pyæmia. Their formation is mostly indicated by the usual symptoms which attend such inflammation. Sometimes, however, they come on rapidly and with little or no pain.

The nervous symptoms are much like those which attend typhus and some other specific fevers. They vary, but comprise in the first instance either restlessness or apathy and drowsiness, and later on delirium, which may be violent, but is generally muttering, and often passes into coma. Muscular debility is always well-marked from the beginning, and soon becomes extreme. There are often tremors or subsultus, and sooner or later loss of control over the bladder and rectum.

The time at which pyæmia arises in relation to the morbid condition on which it supervenes varies. In accident and operation cases, and in those of carbuncle and erysipelas, it may come on at any moment from the commencement of suppuration up to the period of complete recovery. In cases of acute suppuration connected with bone, and acute necrosis, pyæmic symptoms are sometimes present almost from the first. In puerperal cases pyæmia usually manifests itself between the third and the tenth or twelfth day after labour. As regards those cases in which infarcts or pyæmic formations are not found after death, in which therefore there is reason to believe that the symptoms during life have been due to the absorption of poisonous fluids only, and to which (if to any) the term septicæmia (as distinguished from that of pyæmia) is specially applicable, it may be observed that the symptoms have a good deal of resemblance to those developed in animals into whose tissues septic fluid has been injected in poisonous doses. There is not generally any rigor, or if there be it is only at the commencement; but among the more striking phenomena are, mental apathy or delirium, vomiting and diarrhœa, great dryness of tongue, feebleness and rapidity of pulse, perspirations, extreme muscular debility, and rise of temperature. The temperature,

however, often sinks as death approaches, and sometimes becomes subnormal.

The prognosis of pyæmia is exceedingly unfavourable. There is little doubt that recovery does occasionally take place; at the same time it rarely happens that this event ensues in cases which, from the severity of their symptoms, are distinctly recognised as pyæmic during life. The cases of recovery are usually those in which the symptoms from the beginning are mild, and which would probably not be recognised as pyæmic but for the fact of their occurrence during the endemic prevalence of the disease in the wards, say, of a lying-in hospital; or they are cases of simple septicæmia.

There is generally not much difficulty in the diagnosis of pyæmia, when it arises after surgical injuries or parturition. There is much more difficulty when it occurs in patients who are already prostrated by acute inflammatory affections, such as carbuncle or erysipelas, the symptoms due to which indeed are not unlike those of pyæmia itself. And it is particularly apt to be misunderstood when it arises out of some deep-seated suppuration. The diseases for which it may be especially mistaken, and for which it has been mistaken over and over again, are typhus and enteric fevers, internal acute inflammations (especially of the lungs), urethral and bladder affections in which the kidneys have become involved, glanders and acute rheumatism. It may be added that it is a good rule, when a case comes under treatment in which typhoid symptoms with great prostration have developed themselves very rapidly, and in which from the absence of any specific symptom the physician hesitates to form a definite diagnosis, to examine the limbs and surface of the body carefully. It has more than once happened to the writer in such cases to recognise, by the increased bulk of a thigh or arm, the source of the symptoms in the existence of a sub-periosteal abscess.

Treatment.—Very little, unfortunately, can be done medically for a case of pyæmia. We cannot cure the complaint; we cannot arrest it; we cannot, so far as we know, eliminate from the system any poisonous matter to which it may be supposed to be due. Quinine has been exhibited with the object both of checking the periodic rigors, and of reducing excessive temperature; cold baths also have been used with the latter object; hot baths have been employed to promote perspiration, purgatives to aid elimination from the bowels, antiseptics of various kinds to obviate the supposed putrefactive tendency of the disease. But all to little purpose. Our main aim must be, on the one hand to support the patient's strength by regulated and suitable diet and the moderate employment of stimulants, in aid of which vegetable tonics in combination with the mineral acids are often useful; on the other hand, to relieve, as far as may be, all distressing symptoms and injurious complications, for which various purposes no drug is so generally useful as opium or morphia. It should be added that, where

symptoms suggestive of pyæmia show themselves, it is of the utmost importance to attend to the condition of the part which is its supposed source; not so much, however, for the purpose of arresting pyæmia in actual progress as of preventing the occurrence of what may perhaps only threaten. Unhealthy wounds should be freely laid open, deep-seated abscesses freely incised, and, if deemed necessary, antiseptic or caustic injections or applications freely employed.

In surgical and obstetrical practice, especially that of hospitals, the question of the prevention of the spread of pyæmia is one of the highest interest. No doubt pyæmia very frequently occurs spontaneously among both surgical and obstetrical patients. But, whenever either pyæmia or erysipelas, no matter how it has originated, appears among groups of such patients, we know that there is a remarkable tendency for it to spread. To obviate this tendency, extreme cleanliness, ample ventilation, scrupulous nicety with respect to the treatment and dressing of raw surfaces, and especially the utmost care not to allow infection to be conveyed from one to another by the fingers of the medical and other attendants, are essential. The extreme value of Professor Lister's antiseptic method of operating and treating raw surfaces is now almost universally acknowledged.

XXIV. LEPROSY. (*Elephantiasis Græcorum.*)

Definition.—A specific disease, endemic in many parts of the world, characterised by the slow development of nodular growths in connection with the skin, mucous membranes, and nerves, and (in the last case) by the supervention of anæsthesia, paralysis, and a tendency to ulcerative destruction and gangrene.

Causation and History.—Leprosy is a disease which has doubtless been largely confounded with other maladies, such as elephantiasis Arabum, syphilis, and various affections of the skin, but has yet been recognised from the earliest times, has been described under various names, and has been regarded with perhaps more general superstitious awe and dread than any other known disease. It was probably not uncommon throughout Europe during the first two-thirds of the Christian era; but there is no doubt that it underwent a marvellous increase during the twelfth and thirteenth centuries. An epidemic wave seems then to have spread slowly from the south-east to the north-west; and it was assumed, indeed (though probably erroneously), that at that time it was imported into Europe by the returning Crusaders. The disease prevailed generally with great severity during the succeeding two or three hundred years, then began to subside, and had finally disappeared from the greater part of Europe by the end of

the seventeenth century. This subsidence of leprosy was closely related in time with the asserted introduction of syphilis; and hence it has been maintained (in spite of the clearest proof to the contrary) that these diseases are co-related, and their manifestations mere modified results of the operation of the same virus. But although the greater part of Europe became thus free at the date above assigned, the disease lingered in the Faroe Isles up to the commencement of the present century, and still prevails in certain parts of Italy, Greece, Spain, Portugal, and Russia, and with especial severity in Norway, Sweden, and Finland. At the present day, however, leprosy is mainly a disease of tropical and sub-tropical climates, and among these its chosen habitats are, perhaps, Central and Southern Africa, India and China, the West Indies, and South America. The etiology of leprosy has been largely discussed. Temperature, climate, soil, race, habits, food, have all been regarded as predisposing, if not exciting, causes. That temperature has no obvious specific influence is manifest from the fact that the disease prevails alike in Norway and in India. That soil and climate are equally inoperative is shown by the fact that it occurs both on marshy soils and at high elevations, both on the sea-coast and in inland regions, both in continents and in islands, and in nearly all latitudes. At the same time it is worthy of note that a large number of the localities which it specially affects are low-lying and marshy, and on the sea-coast or banks of rivers. That race and habits are not specific causes is clear from the prevalence of the disease amongst races of the most diverse kinds, and amongst persons of the most opposite habits. Yet it may probably be admitted, and has been asserted, that of several races living associated together and under many similar conditions, some are more prone to leprosy than others; and also that the disease is on the whole more common among the poor and filthy than among the well-to-do and cleanly. As regards the influence of diet, it may be pointed out that it has been attributed to the use of decomposing fish; but, unfortunately for this theory, the disease is met with where not only fish is never eaten, but where the diet is mainly vegetable. It follows necessarily that if the cause of leprosy reside in any of the conditions which have been enumerated, that condition has at all events as yet escaped recognition. Formerly the disease was regarded as highly contagious, and consequently all communication between the sick and the healthy was rigorously interdicted. At the present day, however, its contagiousness is almost universally denied by scientific medical men; and it is beyond doubt that the attendants on the sick apparently fail to take it, that children live habitually in the same house with leprous members of their family without becoming affected, and that even sexual cohabitation may go on for years without the disease being transmitted from the diseased person to the healthy one. On the other hand, it seems to be clearly established that the disease is to a considerable extent hereditary—hereditary, that is to

say, in the same sense as tubercle and carcinoma are hereditary, but not in the sense in which syphilis is hereditary. In other words, it appears, not that children are ever born with leprosy, but that the children of leprous parents are more likely to become affected than are the children of healthy parents—a fact which probably explains the supposed influence of race. It must indeed be admitted that the causes of leprosy, of its generally endemic character, and of its occasional epidemic prevalence, are alike unknown. That the disease has a specific character is quite clear; and that the tendency to it (if not the disease itself) is transmissible from parent to child is equally clear. But whether it belongs to that class of diseases which is represented by tubercle and carcinoma, or whether, like ague and goitre, it is the result of some obscure telluric condition, or whether, like scurvy and ergotism, it is due to some default or error of diet as yet unrecognised, or whether, like cholera, enteric fever, or syphilis, it is imparted in some special way by the sick to the healthy, are matters in regard to which we have no accurate knowledge. It may be added that certain recent statements in respect to the introduction of leprosy into the Sandwich Islands and into Australia, and its subsequent spread in those countries, (assuming them to be correct) go far to establish its communicability. Dr. Liveing concludes that, if not contagious in the ordinary sense of the word, it is capable of propagation by the imbibition of the excreta of lepers.

Symptoms and progress.—Leprosy is a disease of both sexes and all ages, but commences most commonly in early adult life. It is usually preceded by premonitory symptoms which continue for weeks, months, or even years, before the specific signs of the disease manifest themselves. These consist in the first instance in lassitude and depression, attended with more or less febrile disturbance, rigors, nausea, and loss of appetite. After a time livid blotches make their appearance here and there on the surface of the skin, remain out for a few days or a few weeks, and then subside, to be followed at irregular intervals by other similar outbreaks. They are tender, elevated discs, or rings, or more or less irregular patches, varying perhaps from half an inch to two or three inches in diameter. In the course of time the blotches become more persistent, and their subsidence is followed either by brownish pigmentary stains, or by an unnatural whiteness and opacity of the skin, associated with more or less contraction and depression. The central area of a patch not unfrequently assumes one or other of these conditions, while its periphery is still extending in the form of an elevated livid ring. During the earlier of these stages the affection has often some resemblance to psoriasis, lupus, or acne rosacea, and is sometimes termed *macular leprosy*; during the later of them the condition of skin is sometimes designated *morphœa nigra* or *alba*, according as the cicatricial area is pigmented or colourless.

The specific phenomena of leprosy now begin to develop them-

selves, and these vary according as the skin and mucous membranes on the one hand, or the nerves on the other, are principally affected. Many cases no doubt occur in which all of these tissues are implicated either simultaneously or in succession; but in a large number the specific morbid processes are almost accurately limited to one or other tissue, and the disease hence assumes two distinct and easily recognised types. They are known as '*tubercular*' and '*anæsthetic leprosy*' respectively.

In *tubercular leprosy*, which is relatively most common in temperate climates, nodular elevations slowly develop themselves in the substance of the cutis, and mainly on the site of the macular eruption. These are attached by broad bases, become more and more prominent and sometimes pedunculated, and not unfrequently coalesce with one another so as to form irregular nodulated masses. They vary at length individually from perhaps the size of a hazel nut to that of a walnut. They are for the most part hard and resistent, of a dusky reddish or brownish hue, smooth and sometimes polished on the surface, and often, like those of lupus, present a certain degree of translucency. They are attended with little inherent pain or uneasiness, but are more or less tender, and are remarkable for their permanence and the little tendency which they manifest to undergo degeneration or ulceration. Nevertheless they do occasionally, after a long time, become the seat of some partial fatty change, grow softer and almost fluctuating, and acquire a dirty yellowish hue; and not unfrequently also, when irritated by exposure, filth or injury, they become excoriated or ulcerated, or covered with thin scabs, and exude a serous or ichorous fluid. The growth of the tumours is attended with atrophy of the cutaneous glands and of the hair. The latter first becomes thin and dry and loses its colour, and then disappears entirely. It is important, however, to note that the loss of hair is not, as in syphilis, general, but simply limited to the situations in which there is obvious disease. The tubercles of leprosy occur mainly on those surfaces which are most exposed to the air, namely the face, hands and feet, but they are common also on the extensor aspects of the limbs. On the face they chiefly affect the eyebrows and eyelids, the nose and lips, and the lobes of the ears. The nodulated thickening of the eyebrows and adjacent parts of the forehead gives a peculiar morose character to the expression; and the thickening of the nose and lips with the associated bronzing of the parts imparts to the European the appearance of the mulatto. When the face is thus affected the term *leontiasis* is sometimes applied to the disease. In the hands and feet the back or dorsum is chiefly involved. In addition to the cutaneous growths which have just been described, nodules of the same kind appear in the subcutaneous tissue. The morbid process is limited to the skin and subjacent tissues for a longer or shorter time; but at length certain of the mucous membranes become implicated, especially those of the nose, mouth and larynx. The

affection here is of the same kind as that in the skin; it consists in the formation of nodules which increase in size, run together, and sometimes form flattened elevations. The growths, however, are softer, more readily ulcerate, and on healing leave deep and dense cicatrices. In the progress of the disease the cartilages of the nose not unfrequently become exposed, the tongue large, nodulated, and seamed with cicatrices, and the different parts of the larynx thickened and stiff, and its channel contracted. In association with the affection of the larynx a peculiar cough and hoarseness of voice become developed which are very characteristic of the disease. According to Danielssen and Boeck the trachea and bronchial tubes may undergo the same changes as the larynx. The conjunctivæ are apt to be similarly affected, and inflammation, suppuration, and perforation of the corneæ to ensue.

In *anæsthetic leprosy*, which is specially common in hot climates, it not unfrequently happens that no tubercles are ever developed. And the cutaneous affection may either be that which has been described as among the prodromal phenomema of leprosy, or it may be so slight that attention is first called to it by some impairment or change of sensibility. There may even be no structural change whatever. We will first consider the nervous phenomena, and afterwards the local processes going on in the skin and subjacent parts. In the first instance there may be a combination of hyperæsthesia and anæsthesia, some parts being numb or insensible while others burn or tingle and are excessively tender, and not unfrequently areæ of numbness are surrounded by rings of increased sensibility; these conditions, moreover, replace one another, so that parts which were hyperæsthetic become anæsthetic; and, further, they may occupy numerous scattered spots or pervade separately or in combination extensive tracts of skin. They are often connected, though by no means necessarily so, with the cutaneous maculæ. The affection of the sensory nerves is generally associated with affection of the motor nerves, and indeed the latter (although it seems to come on later) occasionally preponderates. Thus, there are often tremblings and jerkings of the limbs; but especially there soon supervenes muscular paralysis. The anæsthetic and paralysed regions gradually shrink, the fat, the muscles, and even the bones waste, and the skin contracts over them, becoming white or pigmented, and assuming more or less of a cicatricial character. The parts which are generally first affected, and which suffer most severely are the hands and forearms, feet and legs—in the upper extremity mainly those parts which are supplied by the ulnar nerve, and in the lower extremity the regions correspondingly situated on the outer side of the leg and foot. It will be recollected that the ulnar nerve, besides giving sensory branches to the inner side of the lower part of the arm, to the inner side of the hand, and to the ring and little fingers, supplies motor nerves to the flexor carpi ulnaris, the inner half of the flexor profundus, the muscles of the ball of the little finger, the interossei, the adductor muscles of the

thumb, and the palmaris brevis. And the consequence of their wasting and loss of function is that the palm becomes flattened, the thumb separated from the other fingers, and these powerfully extended at their first joints, and flexed at their second and third joints—conditions which impart to the hand the well-known claw-like form which always results from paralysis of the ulnar nerve. Bullæ not unfrequently form and burst, sometimes healing quickly and well, at other times leading to obstinate ulcers, which leave hard depressed cicatrices behind. After a time gangrene is apt to occur in the affected parts, more especially in the hands and feet. This sometimes begins from the surface, and gradually deepens, until the bones are exposed; sometimes begins among the deeper tissues, and involves the skin secondarily. It often ends in the separation of the bones, in the loss of fingers or toes, or even of a hand or foot. It is remarkable, however, how rapidly and perfectly wounds thus made heal up.

The duration of leprosy is very uncertain: that of the anæsthetic variety is, on the average, sixteen or seventeen years, that of the tuberculated form eight or nine. Death is due partly to gradual impairment of nutrition, but mainly to the supervention of complications, especially phthisis, dysentery, and kidney affections.

Morbid anatomy and pathology.—The morbid process on which the chief phenomena of leprosy depend consists in the infiltration of the affected tissues with innumerable small cells containing comparatively large nuclei. These, in accordance with Virchow's views, are probably due to proliferation of the connective-tissue corpuscles; and collectively form more or less extensive masses of new growth which are almost identical microscopically with granulation-tissue and with the tissue of syphilitic gummata, or of lupus. The leprous growth differs, however, from the latter two especially, by its permanence and comparatively little tendency to undergo degenerative changes. The new growths present, at all events during their earlier progress, a greyish, yellowish or brownish tint, are firm, translucent and homogeneous in texture, and contain few blood-vessels and little blood.

In tubercular leprosy the tumours commence in the skin around the hair follicles and glands, which in their progress they gradually compress and destroy, together with the majority of the other textures which they involve; the epidermis, however, remains for the most part normal, and the muscles of the hairs, in the beginning at all events, become hypertrophied. The tubercles do not usually admit of being enucleated, but are connected by processes with the subcutaneous connective tissue. Their formation beneath the skin and in connection with the mucous membranes essentially accords with the above description. It must be added that both in the macular stage and in the anæsthetic form, the cutis, however slightly it may appear to be affected, is still the seat of specific proliferation.

In anæsthetic leprosy the nerves are always implicated to a greater

or less extent—the smaller branches being mainly involved, and of the nerve-trunks those portions which are most superficial and most obnoxious to injury. They swell to several times their normal bulk, sometimes uniformly, but more frequently irregularly, so as to present something of a beaded character. They become at the same time firm, greyish, and translucent. The change is due to a proliferation of the cells of the connective tissue of the nerve-bundles (mainly of that which separates the individual nerves from one another, and of that which bounds and isolates their different strands), and exactly resembles what occurs in the skin and mucous membranes. At first the essential elements of the nerves suffer but little from the adventitious growth which surrounds them; eventually, however, they undergo degeneration.

Dr. Vandyke Carter[1] shows that, in anæsthetic leprosy, the affected muscles become converted into fibrous tissue; and that the bones especially of the hands and feet waste; that the carpal and tarsal bones suffer thus to some extent, but that the metacarpal, metatársal and phalangeal bones are chiefly affected, and in an increasing order from the first of these to the last phalanges; that their shafts become attenuated, and their distal extremities disappear. He shows also that these conditions involve mainly the fourth and fifth fingers and the corresponding toes, and that here the last phalanx not unfrequently disappears wholly, the skin and nail then shrinking on to the top of the second phalanx.

We have already adverted to the statement of Danielssen and Boeck that leprous patients are liable to the development of specific tubercles throughout the bronchial tubes; they describe them also as occurring in the substance of the lungs, liver and other organs. These statements have not, however, been fully verified by subsequent observers. It is certain, however, that in all forms of leprosy the lymphatic glands become largely hypertrophied, and mainly those which are in immediate connection with diseased districts—the glands which chiefly suffer being those of the groin and those of the neck and submaxillary regions. Distinct leprous infiltration and degeneration of the testicles is recorded by Virchow.

The ulceration, gangrene and other inflammatory processes, which are so common in the course of leprosy, seem to be due, not so much to any special tendency which leprous formations have to pass into such conditions, as to what may be regarded as accidental circumstances. Thus in the case of tubercular leprosy, ulceration seems to result from the effects of exposure, cold, dirt, and other sources of irritation; and, in the case of anæsthetic leprosy, the ulceration and gangrene are probably mainly dependent on the irritative implication of the nerves.

[1] On Leprosy and Elephantiasis. 1874.

Treatment.—By common consent leprosy is an incurable disease; nor does it admit of alleviation or arrest by medicinal treatment; but it is doubtless well, when the case admits of it, to remove the patient from a locality in which the disease is endemic, to protect his surface as far as possible from injurious influences of all kinds, and to maintain his strength by appropriate food and various tonic adjuvants.

XXV. AGUE. (*Intermittent and Remittent Fever.*)

Definition.—A specific non-contagious fever, produced by malaria; characterised by enlargement of the spleen and recurring attacks of fever attended each with a cold, a hot, and a sweating stage; and having an indefinite duration, and a tendency to recur which may last for many years or during the whole of life.

Causation and history.—Ague is undoubtedly not contagious. It is not communicable from man to man, nor does it spread from a centre, successively invading town after town and country after country. It is strictly an endemic affection, belonging to certain districts and induced in them by some poisonous influence which pervades them. Ague-districts are scattered more or less irregularly over the whole non-aqueous surface of the globe, excepting apparently that of the frigid zone. And the virulence of the poison which they yield increases for the most part as they approach the equator. They generally present certain common features: they are tracts of low-lying marshy ground, often situated upon rivers or lakes or in the vicinity of the sea, often presenting a luxuriant vegetation, and always a porous soil which is commonly composed to a large extent of decaying vegetable matter. But, however fever-stricken such places may be, the malaria which they breed is evolved at certain seasons only. In our own country, and probably in all temperate climates, the dangerous periods are spring and autumn, especially autumn; in the tropics, the season of heat and drought which follows upon the periodical rains; and in all cases, it would seem that the poison is produced only or with special intensity, not when the marshy ground is thoroughly soaked, but when, after it has been thus soaked, the surface to a little depth has undergone a rapid process of drying. What, it may be asked, is the condition common to all the variously situated aguish regions which causes ague? Is it high temperature? Clearly not: for many of the hottest regions of the earth are completely blameless. Is it the presence of water? The answer must be No; for, if aqueous vapour could cause ague, all who frequent the sea, or live in the vicinity of rivers, should contract ague; and especially, aguish districts should be most dangerous at those very times when they are now most free. Is it the presence of decaying

vegetable matter? Again the answer must be No. Decaying vegetable matter exists abundantly in places where ague never occurs; and moreover, as Sir Thomas Watson remarks, if such matter could cause ague, Londoners ought at least to be occasionally infected by the contents of their dust-bins and by the neighbourhood of Covent Garden market. But the specific influence of decaying vegetables in the causation of ague is disproved by the fact that ague prevails in certain places where no such matter exists. 'In August 1794, after a very hot and dry summer, our army in Holland encamped at Rosendaal and Oosterhout. The soil in both places was a level plain of sand, with a perfectly dry surface, where no vegetation existed, or *could* exist, save stunted heath plants. It was universally percolated to within a few inches of the surface with water, which, so far from being putrid, was perfectly potable. Here fevers of the intermittent and remittent type appeared among the troops in great abundance.' (Watson.) Again, the soil of Hong Kong consists of disintegrated granite, containing, according to Dr. Parkes, less than 2 per cent. of organic matter; yet ague, which had not previously prevailed, became rife and fatal at a time when the soil was being extensively excavated for building purposes. The last quotation illustrates another point of considerable importance in relation to the causation of ague, namely, the influence in this respect of upturning of the soil, of soil at any rate which has long been untouched. The malarious affections which prevailed in the armies before Sebastopol are referred by Trousseau to this cause; and he also points out that in Paris, where ague is almost unknown, epidemics of limited duration have on several occasions been distinctly traced to the formation of extensive excavations.

It would seem, therefore, that neither heat, water, nor decomposing organic matter is alone capable of evolving the malarious poison; but that for its production there must be a certain porous character of soil, a certain degree of saturation of this soil with water—the surface having recently undergone desiccation—and a certain elevation of temperature. It may be added that nothing is more certain than that aguish districts may be rendered perfectly healthy by drainage. In London, most of which is built on land which was formerly marshy, and where ague was once largely prevalent, the disease is now rarely if ever met with unless it be imported.

The malarious poison appears to be manufactured in the soil of the malarious district, and to be exhaled from the surface in company with the moisture which rises from it, and at night-time far more abundantly than in the day. It forms over the infective area a kind of invisible mist, which is denser and more potent in proportion to its proximity to the ground, and which extends to no great height above it. Indeed, it is well known that the ground-floors of houses in aguish districts are more dangerous to sleep in than are the higher storeys; and that the miasm rarely ascends to any great height the sides of mountains

which adjoin such districts. Dr. Parkes considers that the upward limit in temperate climates is 500 feet, in tropical climates from 1,000 to 1,500 feet. As might be supposed, the miasm may be carried by the wind and atmospheric currents beyond the limits of the area in which it is produced; and thus, under certain circumstances, places which are miles away, and in all other respects healthy, not unfrequently become affected. It seems, however, that the miasm is absorbed in its passage across water, so that the intervention of a river three-quarters of a mile or a mile broad, or of a similar breadth of sea, gives perfect safety. Even a belt of trees, acting probably as a kind of filter, will often form an efficient barrier. For the latter reason it is especially dangerous to sleep under trees in malarious places. It is also dangerous, according to some, to drink the water, however pure it may seem to be, which is furnished by the soil of such localities.

What, then, is this miasm? Is it a gas, is it some decomposing organic substance, is it a living thing? No direct proof has yet been adduced of the truth of either of these alternatives. There is, however, much, both in the behaviour of the miasm and in its effects on the human body, to indicate a generic relationship with the contagia of infectious fevers, and to render it probable therefore that the last of the alternatives above expressed is entitled to acceptance. Dr. Salisbury, of Cleveland, indeed, believes that he has discovered the specific cause in the sporules of certain algæ, species of palmellæ.

There are certain facts in reference to the causation of ague, besides those which have been considered, to which attention should be drawn. It seems to be well ascertained that the denizens of malarious districts tend to become, in a greater or less degree, acclimatised, and that hence they less readily contract ague than persons newly arrived. It is remarkable how little the negroes suffer in districts which are fatal to Europeans. Another well-ascertained fact is that persons suffering from fatigue or privation are much more liable to take the disease than those who are well-fed, strong, and in robust health. Again, contrary to all we know of most other fevers, especially of the exanthemata, one attack of ague, so far from being protective, renders its subject more than ever liable to be attacked with it on exposure to its exciting cause.

Symptoms and progress.—The period of latency of miasmatic affections varies within wide limits. Authentic cases are recorded in which persons who have been exposed to the paludal poison have manifested the first symptoms of fever within the ensuing four-and-twenty hours. On the other hand, it by no means unfrequently happens that persons who have been residing in aguish districts at the time of year when ague chiefly prevails have their first attack of ague many months after they have removed thence to some perfectly salubrious locality. Thus we frequently meet with persons, residing in healthy parts of London, who are attacked during the spring or summer with symptoms of ague,

the poison of which was taken into the system during the previous autumn, in Essex or Kent, and had lain dormant during the whole of the intermediate period.

Ague presents itself clinically in two well-marked extreme forms, which, however, pass one into the other by insensible gradations. The first of these is the intermittent fever, which is especially common in temperate climates, and comparatively mild; the second is the remittent fever, which occurs chiefly in the tropics, and is of great severity and danger. We will describe first the phenomena of intermittent, and then those of remittent fever.

A. *Intermittent fever* is characterised by the occurrence of febrile attacks of some hours' duration, separated from one another by periods of apparently, or at all events, comparatively, good health. The patient is attacked suddenly, or after having complained for some indefinite time of lassitude, headache, and general malaise, with a sense of chilliness, and weariness, headache, muscular pains, and epigastric discomfort. The chilliness rapidly increases until the patient feels and looks as if he were suffering from intense cold. He begins to shiver—the sensation of shivering commencing in the back and extending thence to the rest of the body. The shivering is speedily converted into a severe rigor, attended with violent chattering of the teeth and convulsive tremblings of the trunk and limbs. At the same time the skin is dry, and assumes the papular condition known as 'goose's skin;' the face and the hands and feet acquire a dusky hue, the face also looking pinched, the hands and feet shrunken and wrinkled. Whilst this condition lasts the pulse is small, frequent, and often irregular; the respirations are quick and sighing; there is loss of appetite, thirst, and epigastric oppression, not unfrequently associated with sickness; the tongue is perhaps bluish, and slightly furred; headache and pains in the back and limbs are often present, and sometimes torpor or drowsiness; and the urine is pale, abundant, and passed frequently. The length of this, which is termed the '*cold stage*,' presents great variety. In some cases it is represented by a slight sensation of chilliness of a few minutes' duration only. It more commonly lasts from half an hour to one or two hours, and is occasionally prolonged to three or four hours, or even more. During the whole of this stage the temperature of the patient is above the normal, and rises rapidly. The elevation begins in fact before the patient himself recognises the commencement of his attack, and rises quickly and uniformly until towards the close of the stage; at which time, even though he be still trembling violently with the feeling of intense cold, the thermometer placed in his axilla probably marks 105°, 106°, or even 106·3°.

After a time the cold stage subsides, and the next, the *hot stage*, commences. The rigors and aspect of chilliness gradually disappear—slight flushes at first alternating with the diminishing rigors, and then by degrees replacing them. The patient begins to feel comfortably

warm, and the shrunken and livid surface assumes the smoothness and hue of health. But gradually the feeling of heat gets intense; the patient looks excited and flushed; the skin feels dry, harsh, and pungently hot; the pulse becomes fuller, stronger, and soft, but maintains its frequency; the respirations get more rapid and deeper, and the thirst more severe; anorexia continues; the urine is still abundant, but of a darker colour and higher specific gravity; and the headache, which differs in character from that previously complained of, becomes extreme; mental confusion is not uncommon, and occasionally there is slight delirium. During this stage the temperature continues high; sometimes during the early part attaining a higher elevation than was reached during the cold stage, sometimes, on the other hand, falling somewhat below it. The hot stage lasts from one or two hours up to four or five, but is occasionally prolonged to eight or ten hours.

The hot stage is succeeded by the third, or *sweating stage*. The approach of this is indicated by the supervention of a general feeling of comparative comfort; the intense heat of skin diminishes somewhat, and moisture appears on the face, and rapidly involves the whole surface of the body; soon the patient is bathed in profuse sweats; the temperature rapidly falls; the pulse becomes less frequent and softer; the respirations resume their normal rate; the headache disappears; the loss of appetite and the thirst abate; the urine gets scanty, but of variable colour, and deposits a sediment on cooling; and not unfrequently the patient falls into a gentle sleep. The duration of this stage is very various, but is generally shorter than either of the other two. On emerging from it, the patient may still be languid and listless, but on the whole appears to be restored to more or less perfect health.

The duration of the febrile paroxysms and that of their different stages present considerable variety. The whole paroxysm may be completed in an hour or two, or may be prolonged to eight or ten, or even twelve hours. The cold stage, as has been pointed out, may last from a few minutes to some hours, and not unfrequently the shorter cold stage is followed by the longer and more intense hot stage. Again, the hot stage, which is often of some hours' duration, is occasionally absent—the sweating stage in such cases following immediately upon the cold stage. And lastly, the sweating stage may be so slight as almost to escape notice, or may be protracted for many hours.

The period which intervenes between the cessation of one attack and the commencement of the attack next following is called the '*intermission*.' In it the patient seems not unfrequently to be in the best of health. Sometimes, however, he suffers from more or less malaise, the degree and character of which depend on various circumstances which need not be specially considered.

The period which elapses between the commencement of one attack and that of the attack which is next in sequence is termed the '*interval*.'

And it is mainly in accordance with the length of this interval that we determine the different varieties of ague. In one variety the interval is twenty-four hours, or thereabouts, and there is consequently a daily febrile paroxysm. This is termed '*quotidian ague.*' In another variety the interval is forty-eight hours, more or less, and the paroxysm occurs every other day. This should strictly be called 'secundan ague;' but those who framed its name chose to reckon the day of the first attack as one day, the day of freedom as another day, and the day of the next attack as the third day, and consequently attached to it the inaccurate, but now well-known name of '*tertian ague.*' In another variety the febrile paroxysms occur every third day; and this, which should strictly be named tertian ague, has received the designation of '*quartan ague.*' In addition to these three principal varieties, others which are much rarer are occasionally met with. Thus, in some cases the fits recur every fourth, fifth, or even sixth day. And in some cases we have what are termed '*double tertians*' or '*double quartans.*' In the double tertian the patient has febrile paroxysms occurring every day; but, while those of the odd days correspond with one another, in time of commencement, duration, and probably also other features, those of the even days, though presenting a like agreement among themselves, differ markedly from those of the other series. In the double quartan the patient suffers, as it were, from two series of quartan attacks, the first series of similar paroxysms occurring, say, on the first, fourth, and seventh days; the second series occurring on the second, fifth, and eighth days.

In quotidian ague the febrile paroxysm usually commences earlier and lasts longer than in either of the other common varieties—often persisting for ten or twelve hours. In the tertian variety its duration is usually six or eight hours; in the quartan four or six. On the other hand, the cold stage is shortest in quotidian, longest in quartan ague. The interval, as has been pointed out, is rarely exactly twenty-four, forty-eight, or seventy-two hours; when it falls short of either of these periods each successive febrile attack commences earlier in the day than that which immediately preceded it, and is said to *anticipate*; when the interval is prolonged, the periodical paroxysms become later and later, and are said to *postpone.* In the former case the disease is generally becoming more severe; in the latter case there is usually a tendency towards improvement. Tertian ague is at any rate in Europe more common than either of the other varieties; none of them, however, is rare, and they readily and not unfrequently pass the one into the other.

B. *Remittent fever*—the form of ague most common in tropical climates—is much more serious and dangerous to life than the intermittent forms of ague which have just been considered. Its distinguishing feature is that the febrile paroxysms, which come on once or twice a day, are not separated from one another by intermissions of

complete apyrexia, but are rather to be regarded as exacerbations of an abiding febrile state. Further, the cold stage of each exacerbation is always of short duration, sometimes indicated by a few minutes only of shivering or chilliness, and sometimes escaping recognition; the hot stage is much prolonged, lasting from six to twelve hours; and the sweating stage is imperfectly developed, and merges into the period of remission from which it is undistinguishable. The attack of remittent fever is sometimes sudden, but is more commonly preceded by premonitory symptoms, such as chilliness, lassitude, loss of appetite, nausea, epigastric uneasiness, and pains in the head and limbs. The actual febrile paroxysm begins with a rigor or slight chilliness, to which the hot stage speedily succeeds, and after some hours ends in perspiration and the period of remission—the remission, like the hot stage, varying in length from two or three to twelve hours. The paroxysms usually increase in intensity day by day for a few days. The symptoms which the patient presents are for the most part like those which attend intermittent fever, but some of them are much more severe. The temperature attains no greater height, but it never falls during the remissions to the normal standard; there is no difference as regards the respirations and pulse, except perhaps that the latter with the progress of the disease tends to become quicker and weaker. Sickness is much more severe during the hot stage of remittent fever than in the corresponding period of intermittent fever, is often very distressing, and sometimes attended with hæmatemesis (black vomit); the tongue is drier, and occasionally there is slight jaundice; headache and pains in the limbs are more intense; confusion of intellect is more common, and drowsiness, delirium, and coma are not unfrequent. The patient often passes into a distinct typhoid condition, with dry brown tongue, sordes on teeth, muttering delirium, subsultus tendinum, and other symptoms of the kind.

Remittent fever presents at first sight an almost closer relationship, in the type of its fever, with enteric fever and hectic (which usually also are distinctly remittent), than with the varieties of intermittent fever. And indeed enteric fever and hectic were formerly, in many of their forms, termed remittent, and regarded as of malarious origin. It is certain, however, that the so-called remittent fevers of temperate climates have no affinity with ague. And, on the other hand, it is equally certain that there is no essential difference between the remittent and intermittent forms of ague. For not only do they arise from the operation of the same miasm, and present symptoms essentially alike, but their varieties shade into one another by insensible gradations, and they alternate with one another or replace one another in the same individual.

The effects of the ague-poison are not always in accordance with the above description; thus there are described some cases in which

the paroxysm consists in a violent and prolonged cold stage only, during which the temperature is actually lowered—the patient suffering from extreme anxiety and intense thirst, and looking like a corpse; or in which, independently of any other peculiarity, he falls into a condition of exhaustion, and lies torpid, motionless, and as if asleep, for many hours; others, in which the sweating comes on early, is exceedingly profuse and of long duration, and during which the temperature falls rapidly, and the patient lies in a condition of extreme collapse; others, in which the patient presents coma or delirium, or has epileptiform or tetanoid convulsions coming on in the cold or hot stage, and continuing until the establishment of the sweating stage; and again others, in which hemorrhage takes place from the nose, stomach, bowels, bladder, or into the substance of organs. Further, there are various neuroses which are distinctly forms of ague, the more important of them being neuralgic affections of one or other of the branches of the fifth pair. That involving the supra-orbital constitutes one form of the malady known as 'brow-ague.' These may be recognised as being malarious, partly by their periodic character, partly by their occasional supervention on a more or less distinct cold stage, partly by their occurrence in a malarious district, partly by the fact that the patient has already been the subject of ague.

There are one or two points in reference to the paroxysms of ague to which we have hitherto only very briefly alluded, but which are nevertheless of considerable importance. In intermittent fever, during the cold and hot stages the urine is usually secreted in considerable abundance, is pale and of low specific gravity, and the patient generally has very frequent desire to micturate. He passes an excess of urea, uric acid and chloride of sodium, while phosphoric acid is diminished. During the sweating stage the urine becomes scanty and darker coloured, and the amount of the excreted solids which was previously in excess undergoes diminution. In the intermission urea and uric acid fall below the normal standard. In the remittent form of ague the same peculiarities exist, but are necessarily somewhat less marked. In both forms there is occasionally albuminuria or hæmaturia, with renal casts. The spleen is invariably enlarged during the paroxysms, becomes especially swollen during the cold stage, and may generally be easily recognised by palpation or percussion; it then subsides, and during the intermission may return to its normal bulk. If, however, the ague persists, the splenic enlargement becomes more or less permanent.

The duration of ague presents great differences. An attack will probably always subside (unless death supervenes) after some indeterminate period; especially it will subside if the patient be removed from the district in which he contracted it. But this subsidence is rarely final. In the great majority of cases the patient remains for months or years, or for his life-time, liable to fresh attacks of ague,

even if he never again ventures into a malarious district. The attacks then recur at irregular intervals, and are generally determined by some accidental circumstance, such as over-fatigue, an attack of catarrh or indigestion, or the supervention of other ailments, whether mild or serious. In other words, the malarious poison becomes a portion of his being, and seems to tinge and qualify any morbid condition which happens to arise. Death from the ordinary intermittent fevers is very rare; but remittent fever, unless it be promptly treated, is a very fatal disease. The patient dies for the most part in the typhoid condition, and rarely (according to Dr. Maclean) before the eighth day.

If ague assumes a chronic form, and especially if the patient has been long resident in an aguish climate, or has had periodical attacks for many years, organic changes take place in the liver and spleen; their functions become impaired or perverted, and chronic conditions of disease are sooner or later developed. Among the more important of these are various forms of cachexia and dropsy. In some cases the patients simply pass into a condition of debility and anæmia, on which general dropsy may supervene after a time; in some cases jaundice becomes associated with this anæmia, and from the same affection of the liver as causes this, ascites or hæmatemesis and melæna may eventually come on; in some cases, again, degeneration of blood-corpuscles takes place in the spleen, and their conversion there into brown or black pigment-granules, and the diffusion of this pigment thence throughout the system, give a peculiar dirty or bronzed hue to the complexion. Some degree of such discolouration indeed is of common occurrence in persons who have had repeated attacks of ague.

It may be added that it is not uncommon for the denizens of malarious regions to become the subjects of the visceral lesions and cachexia which supervene on ague without ever having experienced a distinct attack of ague—the malarious poison appearing to affect the system slowly and insidiously, and without even the warning which an occasional febrile paroxysm might afford.

Morbid anatomy and pathology.—The pathology of ague is very obscure; and morbid anatomy throws little light upon it. We know that a poison (probably living) is taken into the system, and that this remains incorporated with it for an indefinite period, giving rise at irregular intervals to more or less distinctly periodical attacks of well-marked fever, attended with rapid destruction of tissue, high temperature, and congestion of internal organs, but more especially of the spleen. But where the poison lurks, why it acts periodically, and on what organ or organs it acts chiefly, are matters concerning which we do not positively know anything. There is, however, good reason to believe, on the one hand, that it is not discharged from any surface, and on the other that (whether it acts directly or indirectly thereon) its main effects are wrought through the agency of the sympathetic system of nerves. It

is scarcely probable that the enlargement of the spleen and associated changes in the liver, important though they be in many respects, are anything more than secondary phenomena. The only constant lesion discoverable after death is enlargement of the spleen. This organ becomes distended with blood, and often to many times its normal size; and if the patient die when the attack of ague is recent, it is found large and congested. The liver, too, is commonly to some extent engorged and increased in bulk. Congestion of the neighbouring parts of the alimentary canal has also been observed; and it may be added that in hemorrhagic cases traces of hemorrhage at mucous surfaces and beneath the serous membranes may be discovered.

Enlargement and induration of the spleen and liver are among the common results of long-continued or repeated attacks of ague, or of long residence in malarious districts. Another change to which these organs are liable is a peculiar dark or slaty discolouration, due to disintegration of blood-corpuscles and their conversion into pigment-granules. In the liver, this is generally referrible to the changes which occur in minute extravasations of blood into the capsule of Glisson and the hepatic parenchyma; in the spleen, to similar changes going on in the blood which occupies the intermediate blood-passages. The pigment is apt to escape from the spleen, to enter the general circulation, and to become arrested in the capillaries of different organs, more especially the liver, brain and kidneys, and thus not only causes them to be pigmented, but interferes more or less with their nutrition, and induces various organic changes and functional disturbances.

Treatment.—What the prophylactic treatment of ague should be may be surmised from the foregoing account of the disease. 1st, when practicable, malarious districts should be thoroughly drained and cleared of underwood or jungle; 2nd, those who are compelled to remain in them should take ample precautions; should not go out in the evening, the night, or the early morning; should sleep in the higher rooms of the houses they occupy; should not drink the water of the locality unless it be well filtered or boiled; especially should not expose themselves to the malarious influences when they are ill or fatigued; and on going out should, as Sir T. Watson suggests, wear charcoal respirators, and also regularly take such remedies as are efficacious in curing ague; and, 3rd, persons who are actually attacked with the disease should be removed to some healthy locality.

In treating ague medicinally we have to consider, first, the treatment of the paroxysms, and next that of the disease. It is reasonable to suppose that the ague-patient will experience some actual benefit if we assuage some of his discomforts, and hence that he will be benefited, during the cold stage by the application of warmth, either by packing, warm bottles, hot-air baths, or warm-water baths; during the hot stage by the maintenance of a cool atmosphere, by the use of light clothing, and by tepid or cold sponging; and, during both, by

the administration of diluents. Little or nothing, in fact, is necessary beyond such simple measures. Other remedies, however, have been employed, and some reputedly with considerable success. Thus emetics have sometimes been given previous to the fit; and bleeding has been much lauded as a means of relief during the cold stage. The most valuable, however, of such special modes of treatment seems to be the exhibition of opium in largish doses (about thirty minims of the tincture) during the cold or hot stage.

It is of infinitely greater importance to attack the disease itself, and fortunately ague is one of those maladies for which we have almost unfailing remedies. Cinchona, indeed, its alkaloids, and arsenic are true specifics. There is no difference of opinion as to their efficacy; the only difference which can exist is as to the mode of their administration and the dose. Of the several cinchona alkaloids, quinine, in the form of the sulphate, is undoubtedly the most efficacious, and it is certainly much more convenient of administration than cinchona itself. There are two principal modes in which quinine is administered; by some physicians it is given in a single large dose before each paroxysm is expected, by others in smaller doses at comparatively short intervals.

According to the former mode from twenty to thirty grains of quinine may be given for a dose to an adult. The time of its administration here, however, becomes important. By some it is thought best to give it just before the paroxysm, by others just after it and even during the sweating stage. The immediate object being prevention, it certainly seems most reasonable that the quinine should be given so long before the expected occurrence of the paroxysm as to allow of its being fully absorbed into the system; and hence, of these two alternatives, the latter should be preferred. The plan, indeed, of giving the larger dose during the sweating stage can scarcely be improved upon either in the case of remittent or in that of quotidian ague. When, however, the paroxysms are separated by longer intervals, it is probably best either to divide the large dose into two smaller doses and to give them at intervals, or to give the full dose between six and twelve hours previously to the expected attack.

The other method, which is frequently pursued, is that of giving the quinine in smaller doses—three, four, or five grains—three or four times a day without reference to the times of occurrence of the paroxysms; and, indeed, it may be given freely even while a paroxysm is in progress. In some cases, owing to extreme irritability of the stomach, the quinine (in proportionately increased doses) must be given in the form of enema, or (in proportionately diminished doses) by subcutaneous injection. The time during which the administration of the remedy should be persisted in must necessarily vary with the case. It should be given for at least a week or two after all symptoms have disappeared; and should be at once renewed if a tendency to recurrence manifests itself. It is important, however, to observe that quinine (and

the same is true of arsenic) does not, by continuous use, even for many months, necessarily eradicate the disease. Arsenic is equally efficacious with quinine in the treatment of ague, and indeed sometimes effects a cure when quinine has failed. The liquor arsenicalis may be given in doses of from five to ten minims three or four times a day.

It may be well to observe that it is generally considered advantageous to keep the bowels freely open, and that indeed (so at least it is asserted) quinine and arsenic seem occasionally to be quite inefficacious until a purgative has been administered; that the complications and sequelæ of ague must be treated according to their nature; and that the diet (in regard to which no special rules need be laid down) must be regulated according to the condition, and the tastes or desires, of the patient.

CHAP. II.—DISEASES OF THE SKIN.

1. INTRODUCTORY REMARKS.

MORBID conditions of the skin are of great interest and importance, partly because they are very common, partly because they are in many cases a valuable aid to us in the determination of the nature of internal maladies, partly because their presence so largely affects, not only the health, but the comfort and happiness of those who suffer from them. Further, their position renders them comparatively easy of observation. For all these reasons they have been repeatedly investigated and described with extreme care, and have been distinguished with a degree of minuteness, and classified with an amount of ingenuity, which have been surpassed only in the distinction and classification of the members of the vegetable kingdom. The result has undoubtedly been largely to increase the range and exactness of our knowledge of skin diseases; but it may be questioned whether this result has not been to a great extent counterbalanced by the confusion which the introduction of a large number of names to designate trivial and often fanciful varieties of disease, and the pains taken to discriminate between conditions which are essentially identical, have tended to create.

But there is considerable excuse for minuteness of description and complexity of nomenclature of skin diseases, in the facts—that the skin is an extremely complicated organism, any one or all of the constituents of which may become the seat of almost any of the various morbid processes which have been considered in a former section of this work; that it differs greatly in character in different parts of the body, and is hence not equally liable everywhere to the same affections, or even to present identical appearances under the influence of the same disease; and, lastly, that it subserves various important functions, all of which are liable to modification or impairment in the presence of morbid processes.

m. Individually
A. *Classification and Definition of Ter* head [illegible]

We shall not classify skin diseases either according to the anatomical elements of the skin which are involved, as has been done by Erasmus Wilson; or according to their visible features, which constitutes the essence of Willan and Bateman's system; nor indeed shall we follow any strictly logical scheme of classification. But we shall group them mainly in accordance with their mutual pathological affinities, not hesitating, however, to depart from this arrangement whenever it seems of practical utility, or convenient on any other grounds to do so.

There are certain terms in common use in the description of skin diseases, which we proceed *in limine* to enumerate and explain; and the more especially as there will thus be afforded a suitable opportunity for indicating the principles of Willan's artificial, but nevertheless very simple and useful classification.

1. *Macula.*—By this term is generally meant a spot or patch of discolouration which does not fade on pressure, and in which, therefore, there is some obvious and more or less persistent deposit or change of texture. Freckles, moles, and port-wine marks are good examples of maculæ. Under the same term may be included the circumscribed discolourations due to escape of blood into the tissue of the skin. But these are better known by special names. Extravasations, from about a line in diameter downwards, are (from their supposed resemblance to flea-bites) designated '*petechiæ*'; larger effusions, such as may result from the coalescence of several petechiæ, are called '*vibices*'; and such as present the ordinary characters of bruises are known as '*bruises*' or '*ecchymoses.*' It may be added, that the term '*stigma*' is sometimes employed to indicate small patches or spots of vivid but readily effaceable redness, due merely to congestion, which appear suddenly, and often precede the development of vesicles, papules, or the pocks of vaccinia or small-pox; and that the term '*areola*' or '*halo*' is applied to the ring, more or less broad, of redness which so often surrounds a definite spot of inflammation.

Willan's eighth order of skin diseases was that of the *maculæ*, and included, amongst other affections, freckles, and the various forms of birth-mark.

2. *Exanthema*, or *rash.*—These words are employed, in reference not to individual spots of disease, but to a more or less general eruption of spots or patches, which are inflammatory, and variously grouped, and in the first instance at any rate, red, fading on pressure, and but little elevated above the general surface of the skin. The *exanthemata* formed the third of Willan's orders; and he included in it measles, scarlet-fever, nettle-rash, roseola, purpura, erythema, and erysipelas. It is obvious, however, that he has here grouped together affections of the skin some of which have little in common with the others, and that he has excluded several which should really be regarded as

[illegible]ous purpura is in no sense an exanthem, and erysipelas and erythema have no more right to that name than has acute eczema or impetigo. On the other hand, the eruptions of varicella and small-pox, and especially that of typhus, should certainly be regarded as exanthems. The term 'exanthem' should, indeed, be applied exclusively to the several eruptions which attend and characterise the infectious fevers.

3. *Papula or pimple.*—This is a small elevation at the surface of the skin, generally acuminated or pointed, but sometimes rounded, and rarely exceeding the size of a large pin's head. It is very commonly congested, but by no means invariably so, and often attended with much itching. Papules are produced in various ways. In the condition known as 'goose's skin' there is a temporary production of them at the orifices of the hair-follicles in consequence of the contraction of the arrectores pili; and in the same situation papules often arise from the concentric accumulation of epidermis and sebaceous matter entangling young hairs. The pearly concretions so common in the sebaceous glands of the eyelids constitute another form of papulæ. Typical papules, however, originate either in enlargement of the normal papillæ of the skin, or in inflammatory exudation into the substance of the cutis.

The *papulæ* constituted Willan's first order, and comprised the various diseases known as strophulus, lichen, and prurigo.

4. *Tubercles* are solid elevations of the cutis, ranging roughly from the size of a hazel-nut to that of a papule, varying considerably in form and texture, and presenting more or less permanence. In form, they may be hemispherical, spheroidal and attached by comparatively narrow bases, conical, lobulated or warty; and not unfrequently neighbouring tubercles coalesce, and thus extensive surfaces may become irregularly thickened and lobulated. As to texture, it is sufficient, perhaps, to say that tubercles are sometimes cancerous, sometimes syphilitic, sometimes lupoid, sometimes due to inflammatory changes in sebaceous glands, sometimes simple warts.

The *tubercula* formed Willan's seventh order, and included boils, warts, molluscum, vitiligo, acne, sycosis, lupus, elephantiasis, and frambœsia.

A *wheal* may be regarded as a species of tubercle. Its special peculiarities are that it is of very transient duration, and that it forms a flat, generally circular, elevation, rarely exceeding a quarter or third of an inch in diameter. It sometimes presents a more or less vivid rosy tint, but is frequently pale, and in either case generally surrounded by a halo of congestion. It is usually attended with much itching. A wheal represents an early stage of inflammation; and the swelling which characterises it is due to effusion from the vessels of the part. Wheals may run together, and thus form bands or patches of considerable extent.

5. *Vesicles* are small accumulations of fluid, generally between the

horny layer of the epidermis and the rete mucosum. Individually they vary, for the most part, from the size of a pin's head downwards; but they may be larger than that, and by mutual coalescence may form more or less continuous tracts of considerable extent. They generally stand out prominently from the surface; but where the horny layer of the cuticle is thick, as on the palm and sole, they often present no elevation whatever, are imbedded, and can be recognised only by the peculiar greyish or bluish tint which they present. The amount of fluid relatively to the solid constituents of vesicles varies very much; and especially this is so if the vesicles are of inflammatory origin, inasmuch as the fluid effusion is then often associated with manifest thickening of the subjacent cutis and with overgrowth of the involved epidermis. Indeed, owing to this circumstance, the distinction between vesicles and certain forms of papules becomes purely arbitrary. Certain vesicles (*sudamina*) appear to be simply due to accumulation of sweat between the layers of the epidermis, and their contents are pellucid and acid. Generally, however, vesicles are the result of inflammation, spring up on a congested surface, and present contents which are alkaline, and, according to their age or other circumstances transparent, milky, or tinged with the colouring matter of the blood.

The *vesiculæ* formed Willan's sixth order, and were made to embrace varicella, vaccinia, herpes, rupia, miliaria, eczema, and aphthæ.

6. *Bullæ* or *bleb*s may be regarded as having the same relationship to vesicles that tubercles have to papules. The line of separation between vesicles and bullæ is quite artificial; generally speaking, however, a vesicle the size of a split pea would be termed a bulla. Bullæ usually vary between this size and that of half a walnut. Occasionally they attain the bulk of an orange. But when thus large they are very often elongated and even sinuous as to their base, and their elevation is proportionately reduced. Their contents are identical with those of vesicles.

The *bullæ* were Willan's fourth order, and comprised pemphigus and pompholyx—affections which are now regarded as identical.

7. *Pustules* are accumulations of pus within or beneath the epidermis. They vary in size and form, and also in the degree in which they involve the deeper tissues of the skin. They sometimes commence as vesicles, the contents of which gradually suppurate; but very frequently they are purulent from the beginning. They are generally covered, as vesicles are, by the horny layer only, sometimes, however, by the whole thickness of the epidermis. The inflammation attending the formation of a pustule is much more intense than that which causes a vesicle or bulla, and consequently we find, as a rule, much more marked congestion, thickening, and induration of the surrounding and subjacent parts in the former than in the latter case.

The *pustulæ* were Willan's fifth order, and included impetigo, porrigo, ecthyma, variola, and scabies.

8. *Furfura* or *scurf* is the name given to the thin bran-like scales,

which separate from the surface of the skin on the subsidence of many of the exanthems, and which so commonly form upon the scalp. Scurf consists either of thin plates of epidermis or of a mixture of epidermis and sebaceous matter.

9. *Squamæ* or *scales* only differ from scurf in the fact that the plates of detached epidermis which constitute them are of larger size. They vary considerably, however, in size, thickness, colour, and consistence. Thus, they may be as much as a square inch in area, or even larger; they may be as thin as flakes of scurf or several lines in thickness, in the latter case being always more or less distinctly laminated; they may have the colour of the skin, or present various tints of yellow or brown; and they may be soft or hard, friable or tough. Some of these peculiarities depend on the amount of fluid which has been diffused amongst the epidermic laminæ during the process of their formation. The detachment of scurf or scales is called 'desquamation.'

Willan's second order was that of *squamæ*, and comprised lepra, psoriasis, pityriasis, and ichthyosis.

10. *A scab* or *crust* is a concretion formed upon some diseased surface by the drying up of the exudation which has taken place from it, and generally comprises therefore some of the normal elements of that surface, namely, epidermis and sebaceous matter. The exudation may be either serum, pus, or blood, alone or combined in various proportions; and it is obvious that, according as these occur singly, or intermixed or combined with sebum or epidermis, will the colour and other physical characteristics of the resulting scabs vary. Serum alone dries into thin yellowish or brown translucent flakes, pus alone into greenish scabs of some thickness, and blood into crusts which are black or nearly so. The admixture of sebaceous matter with serum or pus imparts to the resulting scab the colour and general aspect of gum or honey, and that with blood a brown or red tint. When many particles of epidermis are mixed with simple serous exudation, as in cases of acute eczema, the concreted product often assumes a powdery character and the colour of brimstone. Crusts vary much in thickness, and are occasionally of conical form.

It is needless to discuss the meaning of the terms 'excoriation,' 'fissure,' 'ulcer,' 'cicatrix,' and many others which are in common use and generally understood.

B. *Tendency of spots and patches of skin disease to assume a circular form.*

Before proceeding to the description of the different diseases it may be worth while to point out that, while eruptions present great varieties of grouping or arrangement, the individual spots or patches almost invariably have at first a rounded shape, and that as they grow

they maintain that shape unless the form of the surface on which they are situated, or the direction of its grooves, or the union of neighbouring patches with one another, interferes with their regular development. Thus a vesicle, a bleb, a pustule, a papule, or a tubercle is almost invariably circular in the first instance; so is a patch of erythema, lepra, or pityriasis; and so also are the vegetable parasitic affections. In many cases, moreover, there is a tendency for the central part of the inflamed patch to undergo resolution whilst its periphery is extending; and then it not unfrequently happens that the enlarging ring breaks up into fragments, and that some of these form the starting-points of other circles or segments of circles. It is easy to understand from this statement how the sinuous, serpentine and other curious forms which skin diseases frequently assume are produced.

II. ERYSIPELAS.

Definition.—An acute inflammation of the skin, originating for the most part in the neighbourhood of wounds or sores, attended with much redness and infiltration and severe febrile disturbance, and characterised by a marked tendency to spread over the surface, and (especially in the presence of wounds) to become contagious.

Causation.—Erysipelas is either traumatic or idiopathic; that is, it either occurs in connection with wounds, or arises apparently spontaneously on surfaces which were previously sound. The former variety may be developed, therefore, on any part of the body on which wounds have been inflicted, or wherever conditions equivalent to wounds exist, as, for example, in connection with other forms of cutaneous disease, and about the umbilicus in newly-born children; further, erysipelatous inflammation, or a modification of it, may attack parturient women. Idiopathic erysipelas occurs most frequently on the face. That erysipelas is highly contagious among surgical patients, and that its presence in a lying-in hospital induces a rapidly fatal form of puerperal fever among the mothers, and erysipelas of the newly-born infants, are facts now entirely beyond dispute. It is obvious that in these cases the disease is propagated by the transmission from the sick to the healthy of some poisonous matter capable of reproducing it; and from the circumstance that the inflammation always begins at the very spot where a wound or rawness exists, it is reasonable to assume that the poison has been inoculated at that spot. It is by no means clear that erysipelas spreads in the same way to those whose skin and mucous involutions are sound. No doubt many, and apparently very striking examples of such spread are recorded, but, on the other hand, good authorities deny its occurrence, and certainly it is far from common.

In close relation with the subject which has just been considered is the question, whether erysipelas is to be regarded as a specific fever or as a mere local inflammation. The former view is generally entertained, at all events in this country; and the chief grounds on which it rests are: first, the manifest contagiousness of the disease under certain conditions; second, the existence, which is obvious in idiopathic cases, of a distinct, though short, stage of incubation; third, the affirmed enlargement and tenderness of lymphatic glands prior to the appearance of the skin affection, indicating that the erysipelatous inflammation is secondary to constitutional disturbance; fourth, the discovery of bacteria in great abundance in the inflamed tissues and in the lymphatic spaces and vessels connected with them, and the fact that these bacteria may be propagated, with the inflammation which they accompany, by inoculation upon the lower animals; and, lastly, the close resemblance which exists between the general morbid anatomy and symptoms of this disease and those of the specific fevers. The arguments in favour of its being a non-specific and local disease are chiefly the following:—first, the fact that the disease appears to arise constantly from exposure to cold and various other non-specific causes; second, that a previous attack, so far from precluding subsequent attacks, as is generally the case with the infectious fevers, encourages them, as is the common rule with non-specific inflammations; third, that contagiousness is not an attribute of the specific fevers only, for many varieties of simple inflammation—catarrh, ophthalmia, and the like—are apt to spread by contagion; and lastly, that the symptoms and morbid processes which attend erysipelas can be fully accounted for as being the consequences of the local inflammation. We agree with Hebra in the belief that erysipelas is not a specific fever, but a local disease; that is, a local disease in the same sense as inflammations of the lungs, kidneys, and other organs are local diseases.

Apart from contagion, to which, as we have shown, erysipelas is largely due, the causes of the disease seem to be identical with those of other forms of inflammation, especially exposure to cold and atmospheric changes generally, and local irritations of various kinds. The causes which predispose to it are partly breaches of surface, partly constitutional conditions, such as may result from long-continued indulgence in drink, and poor living.

Morbid anatomy.—The earliest local changes consist in a circumscribed blush of more or less vivid redness, which fades on pressure, and the accumulation of inflammatory products—lymph and corpuscles—in the substance of the cutis and subcutaneous connective tissue. The inflamed patch becomes consequently thickened, hard, and brawny. Its margin is well-defined, and obvious to both eye and touch. The character of its surface varies according to the part affected. If the skin be originally smooth and delicate, it becomes yet

smoother, and shining; if it be coarse, all its markings are apt to get magnified and its coarseness therefore exaggerated. The inflammation gradually spreads by continuity to the surrounding healthy parts, and thus extending may ultimately involve a very large area—the entire surface of a limb for example, or that of the head and face, and occasionally (it is said) that of the whole body. As it spreads, however, the parts first affected undergo changes, their tension diminishes, their redness becomes less vivid and assumes a yellowish or brownish tint, and resolution, preceded by desquamation, presently takes place. Thus all stages of the disease may be present at the same time. Occasionally erysipelas (which is then termed '*erratic*') disappears in one part and breaks out elsewhere, and may thus be prolonged by successive outbreaks.

The intensity and results of the inflammatory process vary considerably in different cases. In some the degree of inflammation present is no greater than that attending the affections which we shall shortly describe under the name of erythema. In some the effusion is so abundant that it infiltrates the subcutaneous connective tissue, and well-marked œdema becomes developed. This is common wherever the cutis is thin and the subcutaneous connective tissue lax, as they are in the eyelids and scrotum. In some cases the inflammation goes on to the formation of pus, which, like the œdema, occupies mainly the subcutaneous tissue. The suppuration is frequently diffused; but sometimes, and especially in the eyelids and elsewhere about the face and head, forms circumscribed abscesses. In some cases again, and mainly in connection with suppuration, the connective tissue sloughs; and sometimes the skin itself becomes gangrenous. When œdema, suppuration, or sloughing is present, the inflamed surface becomes paler and duller, perhaps more or less livid, and acquires a soft 'boggy' feel, or pits on pressure. Vesicles and bullæ not unfrequently form on erysipelatous surfaces, and may become converted into pustules. Subsequently excoriations and scales or crusts necessarily make their appearance. Bullæ also, containing sanious fluid, attend the progress of superficial gangrene and subcutaneous sloughing.

Although erysipelas is commonly limited in depth by the fasciæ, it is not invariably thus limited; and hence subjacent organs are apt to get involved. Thus erysipelas of the trunk may produce inflammation of the peritoneum, pleuræ, or pericardium; erysipelas of the neck œdema of the larynx; and erysipelas of the head meningeal inflammation. Again, it not unfrequently creeps from the skin into the mucous orifices—into the auditory meatus, causing inflammation of the ear, or into the nose or mouth and thence to the fauces and larynx. On the other hand, cutaneous erysipelas may result from extension of faucial, aural, and other such inflammations.

There is a marked tendency in erysipelas for the veins, and especially for the absorbents, to become affected. As regards the absor-

bents, indeed, it is not only common to trace red lines from the seat of inflammation to the nearest glands, which get enlarged and tender; but some authors go so far as to maintain that a patch of erysipelatous inflammation is always preceded by inflammatory enlargement of the neighbouring lymphatic glands. Phlebitis, again, with suppuration in or around the veins, occasionally takes place, and occasionally pyæmia.

Repeated attacks of erysipelas lead to permanent thickening and induration, and sometimes to very considerable overgrowth, of the skin and subjacent connective tissue. Indeed, according to Virchow, it is to such attacks frequently repeated that the hypertrophy of these parts in elephantiasis is mainly due.

There is no special affection of internal organs in erysipelas. In the early stages of the disease the blood contains an excess of fibrine and white corpuscles; but subsequently it tends to assume the characters commonly observed in the later stages of febrile disorders. Post mortem it is generally found dark, and fluid or pitchy, with little tendency to coagulate, and still less to the separation of fibrine. It stains the inner surface of the heart and vessels. The organs are generally soft, and the lungs, liver, kidneys, and especially the spleen, congested. Pneumonia is not uncommon. Decomposition is rapid.

Symptoms and progress.—The symptoms of erysipelas are mainly those of the local process and of inflammatory fever; but they are often complicated with those of intercurrent lesions; and they vary in their severity, both actually and relatively, according to the intensity of the inflammation, its extent, and its situation. In idiopathic erysipelas the local signs are generally preceded by an interval, varying from a few hours to two or three days, in which the patient experiences slight febrile symptoms, sometimes rigors; and in which, according to certain authors, some swelling and tenderness of lymphatic glands may be detected. At the end of this time an inflammatory blush appears, generally on some part of the face, attended with heat and tingling, and tenderness on pressure. With the appearance and extension of this, the febrile symptoms increase; there are headache and pains in the limbs, rise of temperature with dryness of skin, rigors, increased rapidity of pulse, furring of the tongue with thirst, loss of appetite and nausea or sickness, generally some constipation, occasionally, however, diarrhœa, and scanty highly-coloured urine. There may be some degree of drowsiness, but sleep is restless and disturbed with dreams. If the case be mild, the symptoms may subside and the patient become convalescent in the course of two or three days. But if the inflammation continue to spread, or in any way to increase in severity, the pulse gets rapid and feeble, the respirations hurried, the tongue more thickly coated and dry; and delirium, at first only when the patient is dropping to sleep or waking, but subsequently constant, comes on. Sometimes at this period diarrhœa occurs; and the patient's

evacuations may be passed into the bed. At this point also (that is, at the end of six or seven days) the patient may begin to amend. When, however, from the inherent severity of the attack or other circumstances, the case takes an unfavourable course, the symptoms assume a more distinctly typhoid character—marked mainly by great failure of muscular power, tremulousness of limbs, dry black tongue, entire want of control over the evacuations, and delirium, which is generally low and muttering, sometimes busy, like that of delirium tremens, and occasionally violent and maniacal. As the fatal end approaches, the temperature often rises, the skin becomes bathed in sweat, the pulse rapid, perhaps irregular, and almost imperceptible, the respirations quick and noisy, and the delirium passes into coma.

The temperature in erysipelas is always above the normal, but rarely exceeds 106°; and although it is liable to considerable variation, there is a general tendency to an evening rise and a morning fall. The urine is always scanty, presents an excess of urea and diminution of chlorides, and often contains small quantities of albumen between the fourth and seventh or eighth day of the disease. The motions are generally dark-coloured, watery, and fetid. The course and event of the disease are often modified by the association with it of some one of the various complications which have been previously enumerated. Thus, œdema of the larynx and congestion of the lungs will each add symptoms and dangers of its own. And similarly inflammation of the membranes of the brain, phlebitis and pyæmia, will each bring its characteristic indications. Further, the health and circumstances of the patient at the time of seizure for the most part largely modify the character and severity of his attack.

Erysipelas which seems to affect only the cutis is termed '*simple erysipelas*'; when the subcutaneous connective tissue is largely involved as well, the affection is called '*phlegmonous erysipelas*;' when œdema, suppuration, or sloughing supervenes, the erysipelas is often termed '*œdematous*,' '*suppurative*,' or '*gangrenous*,' as the case may be. But these distinctions are essentially artificial, for the various forms of erysipelas run into one another, and several, or all of them, may be present at the same time in the same case.

Treatment.—Having regard to the tendency which erysipelas has to become contagious, it is always important that erysipelatous patients should be removed from the neighbourhood of those who are especially liable to take it; and that, in fact, all such precautionary measures should be adopted as have been already recommended in relation to the infectious fevers. The local treatment in mild cases is of little importance; and, even in severe cases, has perhaps little influence. Collodion, nitrate of silver in saturated solution, solution of sulphate of iron, tincture of iodine, and mercurial ointment, have each been strongly advocated. Flour, dusted thickly over the surface, is also recommended. There is an obvious disadvantage in employing anything which con-

ceals or masks the diseased surface; for which reason several of the above applications are objectionable, even if useful on other grounds. Mild astringent lotions and ointments, such as those of lead, zinc, and iron, are probably as useful and convenient as any. Cold-water dressing, which has commonly been discountenanced in this country, is strongly recommended by Hebra, and is undoubtedly useful. Warm applications and poultices are not generally desirable. It is rarely needful to abstract blood locally, or to make incisions, except for the purpose of letting out matter, or relieving tension. In reference to the internal treatment of the disease, we must recollect that mild cases get well spontaneously, and that more serious cases very soon present symptoms indicative of great debility and of blood-poisoning. For these reasons it seems obvious that depletion can never be necessary; but that, as a rule, the strength of the patient should be sustained, and the free action of his excretory organs encouraged. To support strength such nourishment as he can take should be administered frequently and in small quantities; milk, eggs, beef-tea, arrowroot, sago, and the like, are most suitable for the purpose; to which, if the pulse be failing and the tongue dry, brandy, wine, or ale (if the patient prefer it) should be added. To promote the action of the emunctories, purgatives should, if necessary, be from time to time administered, and the patient may be put on a course of mild diuretics or diaphoretics. Ammonia, camphor, iron, quinine, have all been employed in the treatment of erysipelas. It is questionable, however, whether any one of them is of material use in the early stages of the disease. But stimulant medicines are clearly indicated when typhoid symptoms are present; and tonics are, of course, highly valuable during convalescence. Hyoscyamus and opium are not generally indicated, and must always be given with caution. But in cases where there is great irritability, or persistent want of sleep, they, chloral hydrate, or other sedatives are valuable.

III. CARBUNCLE. (*Anthrax.*) BOIL. (*Furunculus.*)

Definition.—A boil or carbuncle is an intense inflammation occupying, within a well-defined area, the entire thickness of the skin (inclusive of the subcutaneous connective tissue), and attended almost always with circumscribed suppuration and the formation of a slough.

Causation.—Boils and carbuncles are usually considered to be constitutional disorders; and undoubtedly they are common in persons of broken-down constitutions, and in those who are recovering from diseases of various kinds. Diabetic patients are said to be specially liable to them. But, on the other hand, they are common in those who appear to be otherwise in perfect general health; often occurring

in connection with acne and other forms of skin disease, or induced by local irritation, such as arises from friction, poulticing, the contact of unhealthy discharges, and (as pathologists know to their cost) the soakage of dead bodies. It cannot be denied that there is, in many cases, a predisposition to boils and carbuncles, and that this predisposition may be induced. We are disposed, however, to regard the disease as essentially local, and due to the operation of local causes; and to believe that, like acne, it is mainly an affection of the sebaceous glands and their surroundings.

Morbid anatomy.—The morbid process commences with circumscribed thickening and induration of the deeper tissues of the skin, attended from the beginning, or soon followed by a little elevation and redness of surface. The resulting nodule increases more or less rapidly in area and thickness and consequently in prominence until, at the end of a few days, it has attained its full development. It then presents a more or less circular base, varying in diameter from half an inch to three or four inches or more; is intensely congested, and surrounded with an areola of congestion and often much œdema; and forms a considerable elevation, which is conical or flat, according as the area involved is small or large, and presents on its summit a vesicle or group of vesicles, containing serous or sanious fluid or pus. Each vesicle soon bursts, discharges its contents, and exposes in its floor a small round orifice, from which, even at this time, an ash-coloured slough protrudes. When there are more vesicles than one, they generally run speedily together; and then by sloughing of the intervening papillary layer of the cutis the subjacent orifices coalesce, so as to form a more or less extensive irregular excavation, the floor of which is formed as are the floors of the primary orifices by underlying sloughy tissue. The slough thus exposed has been gradually forming during the progress of the disease, and involves the deeper structures of the skin and sometimes subjacent parts; mainly, however, it consists of connective tissue saturated with pus, and presenting a yellowish or greyish colour, and a resemblance to wash-leather. It now gradually becomes detached from its bed, and is at length discharged through the orifice which has formed over it. After its separation the excavation which it leaves granulates, the inflammatory thickening of the surrounding tissues subsides, and the parts gradually return to their normal condition, except that a permanent scar remains. The distinction between a boil and a carbuncle is arbitrary; a boil is comparatively small, generally conical in shape, and opens by a single orifice; a carbuncle is characterised by its size and flatness, and particularly by the formation of more orifices than one, and the presence of superficial gangrene. Carbuncles very often arise in the median line of the trunk behind, and especially in the nape of the neck. They sometimes attack the lips (more particularly the upper lip) and are then characterised by great malignancy. The lymphatics and veins

are very apt to get inflamed in these affections; and, of carbuncle especially, pyæmia is a very common sequel.

Symptoms.—The local symptoms are heat, tingling, and aching, with throbbing and great tenderness, which are often followed by pain and redness of the lymphatic vessels and glands in relation with them. There is generally, even with a boil, some amount of febrile disturbance; and with a carbuncle the febrile symptoms may be very severe. Indeed, in the latter case, the general symptoms are almost exactly like those which attend the progress of erysipelas, and may be at least as serious as those of the worst forms of that disease, and the consequences may be fully as grave and fatal.

Treatment.—The general treatment of carbuncle is identical with that of erysipelas. For local treatment free incisions are generally recommended, which, if the carbuncle be large, should be crucial. Pain and tension are greatly relieved by them; but it is doubtful if they check the progress of the disease or materially modify its course. Caustic applications, and especially the free use of caustic potash, are recommended by some. Poultices and warm-water dressings are generally of service. Hebra strongly advocates the employment of cold in the form of compresses saturated with ice-cold water, to be applied so long as they are not disagreeable to the patient.

In the treatment of boils, which often show a tendency to recur, many internal medicines—among others yeast, quinine, and mineral acids—have been recommended, with the object of preventing that recurrence. But it is more than doubtful whether any of them has a specific influence. It is, of course, always desirable to treat any associated malady which may tend to keep up a condition of system favourable to the development of boils. Boils may be dealt with locally on the same principle as carbuncles; and some authorities believe they may be made to abort by the early application to them of strong ammonia, caustic potash, acid nitrate of mercury, or some other such agent.

IV. ERYTHEMA. ROSEOLA. URTICARIA. PITYRIASIS.

Causation and description.—The above affections embrace a considerable number of morbid states of the skin which resemble one another in the facts, that they are for the most part slight, superficial, and essentially short-lived inflammations; that they have little or no tendency to suppuration, ulceration, or gangrene, but end usually in furfuraceous desquamation; and that they are often variously figured and distributed, and are never contagious.

There is great confusion amongst dermatologists as to the distinctions between erythema and roseola. Dr. Willan describes the former as a

nearly continuous redness of some portion of the skin, and the latter as a rose-coloured efflorescence variously figured. But even he includes under the head of 'erythema' affections which, according to his definition, should be varieties of roseola; while, on the other hand, several conditions are now universally termed roseola which, according to the same definition, ought to be regarded as erythema—we refer to so-called 'roseola cholerica' and 'roseola vaccinia.' The formation of wheals is the special characteristic of urticaria; but wheals arise under so many different conditions, and so closely resemble some of the eruptions which are termed erythema, that it is impossible to draw any sharp line between them. For these reasons we propose to discuss erythema, roseola, and urticaria together; and, although we shall preserve the names, we shall regard them as indicating trivial, and in some cases imaginary, distinctions between things which are essentially the same. Pityriasis we look upon as simply the desquamating stage of the different forms of erythema.

Some of these affections are of local origin, due to the action of direct irritants; but many of them, as is shown by attendant circumstances, and by their simultaneous development in different parts, are distinctly traceable to causes acting from within. The former, if extensive, may be attended with febrile disturbance. The latter are generally so attended; and, indeed, not unfrequently appear in the course of some rheumatic, gouty, or other inflammatory or febrile attack. The local symptoms are, for the most part, more or less intense itching, burning, stinging, and occasionally aching.

A. *Erythema simplex* is a pretty uniformly diffused redness, occupying an area of very irregular size and form. The redness is generally bright, disappearing on pressure; and is attended with slight thickening and elevation of the skin; and presents a fairly well-defined margin. It often spreads from its primary seat over the neighbouring skin, and is not unfrequently erratic. There is a very close resemblance between certain varieties of erythema and the simplest form of erysipelas; between which, indeed, it is impossible in many cases to distinguish. One variety of erythema is produced by the direct operation of local irritants, as by the application of a mustard plaister, or by the constant flow of catarrhal secretions from the nostrils or of saliva from the mouth, and in children, when from want of cleanliness the urine is allowed to fret the thighs, groins, and other neighbouring parts. Intimately related to this is the condition known as '*e. intertrigo*,' in which inflammation is induced, either by the attrition of opposed surfaces of skin, or by the effect on such surfaces of the decomposing and fetid sweat which accumulates between them. This is common, in children and fat adults, in the groins and between the upper parts of the thighs and the external genital organs; and in fat women between the pendulous mamma and the surface with which it lies in contact. The persistence of the cause in erythema intertrigo

tends to keep up and intensify the irritation; and consequently excoriation and ulceration are apt to supervene. Another variety of erythema is termed '*e. lævе.*' This is the superficial inflammatory blush which often appears in limbs, and especially in legs, which are the seat of anasarca. The redness is generally somewhat unevenly distributed, and is attended with tenderness and itching, tingling or aching. Vesicles, which rupture and allow of the escape of the dropsical fluid, are very apt to form on the surface; and not unfrequently the inflammation passes into distinct erysipelas, or superficial gangrene ensues. A further variety of erythema is known by the name of '*pityriasis simplex.*' This occurs on various parts of the body, but is especially common in the form of circular or oval patches on the lips, chin, and other parts of the face, in children and persons of delicate skin. The patches present a slight degree of redness, and are very early covered with thin branny scales, or scurf, whence the name pityriasis has been given to them. This affection is also of common occurrence in the hairy scalp, when it is usually called '*p. capitis*' or '*dandriff.*' In this case the branny scales, which form pretty abundantly and, owing to the presence of hair, tend to accumulate, contain, as might be expected from their soil, a large admixture of sebaceous matter.

B. *Erythema multiforme.*—Under this term, which we owe to Hebra, are included *e. papulatum* (in the sense in which Hebra employs that term), *e. circinatum*, *e. iris*, *e. marginatum*, and *e. gyratum*. The earliest stage of the affection is characterised by the appearance of small, flat, circular, congested elevations of the cutis, attended with itching, and differing little if at all from wheals (*e. papulatum*). Their development may cease at this point; but in most cases they pass on to a second stage: the wheal gradually increases in area until perhaps it measures half an inch or an inch in diameter; and while thus increasing, its central portion probably subsides, its periphery forming a congested tumid ring (*e. circinatum*); or the enlargement of the inflamed patch is effected by the development of successive concentric rings of inflammation, separated by zones of fairly healthy skin, and the affection known as '*e. iris*' results. Further, the spots of e. papulatum, and the patches of the circinate form of the affection, which may attain much larger dimensions than have been above assigned to them, tend in the course of their development to coalesce with one another, and thus to cover with more or less uniformity round, oval, or sinuously-margined areæ of several square inches, which, like the spots from which they sprung, are still for the most part characterised by a tendency to central subsidence, and marginal extension by a broad band of congestion. In their progress these 'fairy rings' not unfrequently break up into segments, and hence after a while curved or sinuous erythematous bands alone remain. These latter forms of the affection constitute *e. marginatum* and *e. gyratum* respectively. The several varieties of

erythema above described occur on different parts of the body and are sometimes very extensively distributed; they are most common, however, on the backs of the hands and wrists and corresponding parts of the lower extremities. They are generally attended with febrile symptoms, which, if the eruption be extensive, may run high—the temperature rising temporarily to 104° or more; and they are very apt to be associated with rheumatism or gout. Individually the inflamed patches seldom last more than a week or ten days, sometimes not longer than two or three days, and terminate in desquamation. But the eruption may be continued by successive crops for several weeks. Occasionally it assumes a chronic form, or the patient remains for years liable to more or less frequent outbreaks. It sometimes happens, that the wheals or rings become the seat of intra-cutaneous hemorrhage (*purpura urticans* ?) which generally occurs in the form of minute coalescing points and is for the most part limited to their central arcæ; and that this leads to the death of the involved cutis and separation of eschars, or to the development of sanguinolent blebs, and unhealthy ulcers. Further, vesicles or bullæ, containing limpid fluid, not unfrequently arise in more or less abundance upon the surface of the erythematous patches, constituting varieties of *herpes* and *pemphigus*.

C. *Erythema nodosum* is characterised by the appearance of round or oval red patches varying, roughly speaking, from ½ inch to 1½ inch in diameter. They rise in a lenticular form above the surface in relation with which they are developed, and are consequently most elevated at the centre, where also their redness is most intense; and in both of these respects they fade away gradually at the margins. They are hot, hard and tense to the touch, and to the patient tender and attended with aching. They occur chiefly scattered over the anterior aspect of the leg, between the ankle and knee, but sometimes on the lower part of the thigh. They occasionally appear also on the corresponding parts of the arms; and in very rare cases stud the whole surface of the body, including fingers, toes, and face. The patches generally increase in number for a few days—each one lasting perhaps a week. They get dusky in colour after a day or two, and generally acquire a bluish aspect when exposed to cool air; they present successively the greenish and yellowish tints of fading bruises, and end with desquamation. E. nodosum is for the most part preceded by and attended with febrile symptoms, and not unfrequently associated either with rheumatic pains or distinct rheumatism. It is most common in young persons, especially females, above the age of puberty. The affection described by Willan under the name of *e. tuberculatum* is a modification only of e. nodosum. We believe, too, that the *roseola autumnalis* of the same author is essentially the same disease; and may add that there is little, if any, difference between a chilblain (pernio) and a patch of e. nodosum.

D. *Erythema fugax* is the name given to the evanescent patches of redness which appear on the face, neck, chest, and other parts, in

hysterical and dyspeptic patients. This is closely related to the patches of redness, termed '*roseola*,' which are sometimes observed in cholera, small-pox, and other fevers, and may be held to include those which are so commonly associated with the vesicular and other inflammatory skin diseases of young children.

E. *Roseola*, as has been already explained, is a name of common and somewhat indefinite application. This, or still better perhaps the name '*r. rubeoloides*,' may properly be applied to an affection of the skin, of which Willan seems unnecessarily to make two varieties, namely *r. æstiva* and *r. infantilis*. This rash seems generally to be preceded for a day or two by slight febrile disturbance, and, like so many other rashes, to make its appearance first on the face and neck, whence it quickly spreads over the general surface of the skin. It consists of rose-coloured flatly elevated circles, fading at the periphery into the surrounding healthy skin, and disappearing on pressure, varying perhaps from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter, and often running together over extensive tracts so as to form an imperfect network with scalloped interstices. There is often some general but slight tumefaction of the surface, and a passing chill is apt to render the rash temporarily of a peculiar violet tint. Itching is frequently complained of; and the fauces are sometimes implicated. The affection generally disappears within four or five days after the first appearance of rash. It is quite unattended with danger; and of little importance, but for its resemblance to measles and rötheln or epidemic roseola on the one hand, and to urticaria on the other.

F. *Urticaria* or *nettle-rash* has been subdivided by dermatologists into numerous varieties. It seems unnecessary, however, to make more than two, namely, *u. acuta* or *febrilis*, and *u. chronica* or *evanida*.

The more common form of the disease is *u. febrilis*. In it the appearance of the eruption is often, if not always, preceded by more or less febrile disturbance, with probably some degree of gastro-intestinal derangement; and these symptoms continue during the prevalence of the eruption, which rarely exceeds a few days or a week. The eruption, which is attended with much local heat and itching, generally comes out in the evening or night, and disappears in the morning, and is continued for a few days by successive nocturnal outbreaks. In many cases, however, it appears at irregular intervals both night and day. The wheals arise quickly, seldom remain out longer than a few hours, and on subsiding sometimes leave behind them a slight yellowness of skin and a tendency to desquamate. They may appear simultaneously or in successive crops on any or all parts of the body; but are most common on the face, back, front of the chest, and flexures of the joints. The lips, tongue, and interior of the mouth are occasionally affected. The wheals are sometimes scattered; but are more generally clustered and running together, and may then cover large tracts. Their presence is often attended with subcutaneous œdema and stiffness of parts. Scratching and other forms of local

irritation tend to increase their size, number and duration. Occasionally febrile urticaria is due to the use of certain alimentary substances, such as shell-fish and pork, which, either from some acquired poisonous quality, or from some idiosyncrasy in the subject, act in a special way on the system. In severe cases the symptoms come on rapidly, are very grave, and indeed may prove fatal. They are mainly rigors, failure of circulation, fainting, precordial oppression, vomiting, and difficulty of breathing. They generally subside, however, in the course of a few hours.

Chronic urticaria, which supervenes in some cases on the acute form, is generally unattended with marked fever. It shows itself for the most part, like that, in successive crops of eruption, which come out daily or at irregular intervals, for weeks, or months, sometimes for many years. A curious sub-variety of chronic urticaria is that which Sir W. Gull has termed '*factitious urticaria*.' Here the eruption, although it may come out as in other cases in successive crops, is also really produced by pressure or irritation. And thus the application of a ligature, or the passage of the finger-nail, is followed in a few seconds by the appearance of a line of confluent wheals, with an areola of congestion, which remains out for a minute and then disappears.

Wheals, more or less exactly resembling those of urticaria, are very often the result of the operation of local irritants. They are common in prurigo, scabies, and phthiriasis. They result from the prick of the ordinary stinging-nettle, and from the action of some species of jelly-fish. And they follow the bites of many insects, such as gnats, fleas, and bugs. In this last case, however, the wheals are persistent, and often last for a week or ten days. They probably constitute Willan's '*urticaria perstans*.'

The causes of urticaria are not well understood. Some of its severer forms are caused by poisonous matters received into the stomach, and acting through the medium of the circulatory system. It is natural, therefore, to assume that other forms of urticaria must be due to gastro-intestinal disturbance. That in many cases it really is so is probably beyond doubt. But it is equally certain that, in a large number of instances, especially of the chronic variety of the disease, there is no indication whatever that the digestive functions are at fault. Mental emotion, hysteria, and uterine affections are sometimes assigned as causes of urticaria.

Treatment.—Most of the erythematous inflammations which have just been described need little or no special treatment, either local or general. Many of them must be regarded as parts, and indeed trivial parts, of more serious diseases—such as rheumatism; and their treatment must merge in that of the more general malady with which they are associated. For most of them cooling or astringent lotions, such as cold water or lead-wash, are serviceable and agreeable; but for some of them, more especially e. intertrigo and pityriasis, careful

local treatment is generally essential. In e. intertrigo the affected parts should be kept perfectly clean and free from acrid moisture, and opposed surfaces separated, if necessary, by a piece of lint anointed with some appropriate ointment. Dusting the surface with starch, oxide of zinc, fuller's earth, lycopodium or violet powder, or applying astringent lotions or ointments, are often valuable measures. In pityriasis cleanliness is equally essential, and the cure is often aided by the use of mild mercurial ointments. When the lower extremities are affected with e. nodosum or e. læve, the patient should keep the recumbent position, with the legs elevated. The general treatment of these various affections should be mildly antiphlogistic and comprise cooling drinks and gentle laxatives. In e. nodosum, however, it is frequently necessary to have recourse to tonics. And in urticaria, if it be either severe or chronic, special measures must be adopted. If, for example, there be reason to suspect its dependence on poisonous substances taken into the stomach, an emetic or a purgative may be necessary; if there be much abdominal pain, opiates; if collapse, either ammonia, brandy, or other stimulants. In the chronic form of the disease few remedies have been found useful, but arsenic, mineral acids, alkalies, tonics, and change of air have often been recommended.

V. PSORIASIS. (*Lepra.*) PITYRIASIS RUBRA.

Causation and description.—We have shown that one of the events of the different forms of erythema is the formation of scurf; we pass, therefore, naturally from their consideration to that of psoriasis, which is essentially also a superficial inflammation of the skin, attended with the development of scales. It is thus closely related to pityriasis, and cannot always be separated from it. Willan and his followers have distinguished psoriasis from lepra, but their distinctions are artificial; and we shall, therefore, with Hebra and others, combine them in a common description.

A. *Psoriasis* is characterised by the presence of defined, mostly circular tracts, in which the cutis is somewhat congested and raised; while the epidermis over it is thickened and opaque, and tends to come away in large flakes. These, on their separation, leave behind a congested, irritable, and sometimes slightly excoriated surface, on which squamæ are speedily reproduced.

Psoriasis commences with spots or discs of slight congestion, over which, almost from the earliest moment, the cuticle assumes a scaly character; but at first, and while they are in process of enlargement, the area of congestion usually extends beyond that of desquamation.

The patches vary in size and shape. In some cases they are mere

papules, a line or less in diameter; in some they have a discoid form, measuring between ¼ and ½ inch across; in some they form rings between (say) the size of a shilling and that of a crown-piece, enclosing a central area of comparatively healthy skin, which (especially if they become large) tend to break up into segments; in some cases, again, partly by coalescence of adjoining patches, partly by innate irregularity of growth, they form patches of large size and irregular outline, covering, it may be, an entire limb or even the whole surface of the body.

The squamæ also vary in colour, consistence, thickness and form. These peculiarities are mainly due to the different degrees of rapidity with which they are developed, and to the fact that they result from an excessive formation and exfoliation of epidermis, among the cells of which inflammatory exudation and even the contents of the involved cutaneous glands are diffused in various proportions. The scales are sometimes white and glistening, like mother-of-pearl; sometimes yellow, and more or less waxy in appearance; sometimes brown or black; sometimes close and dense in texture; sometimes friable and flaky, or even powdery. In some cases they form an extremely thin layer, in others they are a quarter of an inch or more in thickness; and occasionally, where a virgin patch has been slowly enlarging, the accumulated scales on its surface assume the form of a limpet-shell. The general outline of the crust will necessarily be determined by that of the patch on which it is produced.

The subjacent skin is always more or less distinctly congested and thickened; and generally, when the disease is in an aggravated form and has existed for some time, tends to get excoriated and fissured, and then to exude serum and blood, which, mingling with the squamæ, form distinct scabs.

The eruption of psoriasis is peculiarly liable to attack the extensor surfaces of the knees and elbows. But it may occur on any part of the person, though it is comparatively rare on the face, and still more rare on the palms and soles. The hairy scalp is a common seat of the disease. The nails also are not unfrequently involved, becoming thick, rough, and coarse in texture. It is very apt to be symmetrical.

Psoriasis presents, as may be supposed from the above account, many varieties of character, some of which it may be useful to remember, if only for descriptive purposes. Thus, when it consists of an eruption of numerous small spots, it is called '*p. guttata*'; when of small discs covered thickly with white scales, '*lepra alphoides*' or '*alphos*'; when of rings, '*l. vulgaris*'; when of segments of circles which have coalesced with similar segments of adjoining circles, '*l. gyrata*'; and when of irregular patches occupying a large area, '*p. diffusa.*'

The progress of psoriasis is occasionally remarkably acute; thus, it will sometimes come out and become general in the course of a week,

and disappear with almost equal suddenness at the end of two or three weeks. At other times, and much more commonly, it is a chronic malady; sometimes persisting for years in two or three situations, as, for example, on the knee, or point of the elbow, and presenting periodical exacerbations in the spring or autumn; sometimes occupying large tracts of surface persistently (*p. inveterata*) for many years, or for life.

The general health of patients suffering from psoriasis is rarely materially or even obviously impaired. Occasionally, however, febrile symptoms attend its acuter manifestations, and sometimes debility and emaciation supervene in the course of long-continued severe attacks. Yet the remarkable tendency of the eruption to break out simultaneously in corresponding situations on both sides of the body, and its undoubtedly hereditary character, together with the fact that an almost identical eruption attends the constitutional operation of the syphilitic virus, point very strongly to the dependence of psoriasis on constitutional causes. It may be added that its development and disappearance are often very manifestly influenced by constitutional modifications. Thus it occasionally shows itself only during pregnancy, disappearing with the birth of the child; and, on the other hand, those who are subject to it, may lose it entirely during the period of child-bearing. It is remarkable how little local discomfort, comparatively, psoriasis produces; a little stiffness and a little itching are often the only inconveniences complained of.

B. *Pityriasis rubra.*—This term was applied by Willan to a variety of that form of pityriasis already briefly considered under the head of erythema. Hebra, and in this respect we follow him, employs it to designate a specific form of skin disease, of rare occurrence, and having a close affinity with psoriasis. So far as is known, it appears to commence with universal congestion of the skin, soon followed by general tendency in the epidermic layer to separate in scales. Its progress is slow, and it is doubtful whether a cure is ever effected. The redness of the cutis, when once established, persists, but is attended with little thickening or discomfort; and the epidermis continues to desquamate, the scales, however, sometimes accumulating in considerable quantity. When fully developed, there is nothing except the history and progress of the malady to distinguish it from universally diffused psoriasis. Patients suffering from it remain apparently healthy in other respects for a long time; but (according to Hebra) they ultimately emaciate, become cachectic, and sink from exhaustion.

Treatment.—The local treatment of psoriasis consists, first of all, in the removal of the scales, which may be effected by warm baths or poultices, or by the thorough inunction of oil or ointments of various kinds; and then in the application of special remedies, among which may be included iodine paint, nitrate of silver, strong solution or ointment of subacetate of lead, and especially tar-ointment, or other equivalent empyreumatic preparations. The persistent use of warm baths

for several hours daily is often of great value. The constitutional treatment most generally resorted to is the exhibition of arsenic in small repeated doses. Tar is often administered internally with the same object; as also are tincture of cantharides, copaiba, iodide of potassium, and phosphorus. Tonics and cod-liver oil are occasionally useful. The disease, however, is very apt to resist all treatment; and even when a cure seems to be effected it is very often only apparent, and the result of the normal periodic retrogression of the malady. The treatment of pityriasis rubra may be conducted on the same principles as that of psoriasis.

VI. ICHTHYOSIS.

Description.—Under this term are included certain affections of the skin, characterised by dryness of the epidermis, with tendency to crack and scale, deficiency or absence of the sebaceous secretion, and more or less horny conversion of the epithelium of the sebaceous follicles.

A. *Ichthyosis simplex, or xeroderma*, is the commonest variety of the affection. It is for the most part congenital, and its presence is generally first recognised by the parents during the first year or two of life, in consequence of the harshness and dryness of the general surface of the skin, and the difficulty they experience in keeping certain parts of it, such as those covering the elbows and knees, in a cleanly condition. In quite young children, indeed, it only manifests itself by the characters just enumerated, and by the tendency of the epidermis to come away in flakes. As life advances, the condition of the skin becomes more characteristic. The affection is then seen to be general, but differing in severity in different parts. It is least marked on the palms and soles, and on the inner aspects of the wrists, arms, and thighs. Here the skin may be a little dry only, and scarcely differing in appearance from healthy skin. The face is generally rough and dry, and slightly furfuraceous. But the greater part of the rest of the surface of the limbs and trunk is mapped out into irregular polygonal areæ, the limits of which are, for the most part, determined by the normal creases and folds; and the epidermis of these areæ—dry, hard, brittle, and somewhat nacreous, becoming partially separated at the edges, and sometimes undergoing complete separation—gives that scaly character to the surface which allies this disease anatomically to psoriasis. But the places in which ichthyosis involves the most striking results are the knees, elbows, and those other parts of the surface which are naturally apt to get thickened under the influence of pressure or friction. Here the epidermis becomes extremely thick and hard, generally brown, or black from impregnation with dirt, and

divided even more manifestly than elsewhere into polygonal areæ. Wilson states that in this affection many of the sebaceous glands are filled with a dry hard substance, which often projects from their orifices.

A condition of skin very closely resembling ichthyosis is often met with in the course of chronic wasting diseases, such as phthisis, and is sometimes developed with advancing years.

Persons who suffer from ichthyosis are said to be, for the most part, feeble and emaciated. But that is certainly not a universal rule. They are often peculiarly liable to eczema and impetigo.

B. *Ichthyosis cornea* is a much rarer affection than the last, and often arises at a later period of life. It is seldom general, but usually appears in scattered patches, which have a tendency to spread. It is characterised by the development of prominent hard, dry, horny processes of epidermis, which very often have an exact resemblance to those occupying the surface of the knee in the simple variety of the disease. These are usually grouped together, and hence individually often assume an irregular prismatic form; and they project sometimes a quarter of an inch or more above the general surface. They are partly due to a mere overgrowth of epidermis in patches, corresponding to the normal polygonal areæ of the skin; but are mainly, we believe, connected with the horny conversion of the epidermic lining of the sebaceous follicles. In the latter case the horny outgrowth first appears as a comedo-like body, which distends the orifice of the follicle, and then rises above it in form not unlike a caraway seed. Presently this gets detached or broken, but the horny matter, still growing upwards and in breadth, distends the sebaceous follicle and its orifice more and more, until they form a mere shallow pit, surrounded by a tumid ring. With the progress of the disease, the pit is effaced; what was the inner aspect of the follicle becomes level with the surface of the skin or projects above it, and still produces (but now from a larger area) its horny growth. Finally, the tendency to horny development extends from the follicle to the epidermis immediately surrounding it. These bodies absorb dirt, and consequently become more or less opaque and black. They are often shed, and then occasionally leave the surface from which they sprang tolerably healthy.

Treatment.—The simple form of ichthyosis is incurable; but it may be much benefited and rendered tolerable by cleanliness, frequent baths, and keeping the surface anointed with oil or grease—olive oil, neat's-foot oil, and the like. The horny variety also is uninfluenced by medicine. But it sometimes dies out in certain situations while it advances in others, and hence it is conceivable that it might occasionally subside altogether. But, although a cure is not to be expected, the horny growths may generally be removed, and the chief discomfort and offensiveness of the disease kept in abeyance, by the frequent use of warm baths, and application of poultices or oil.

VII. ECZEMA. (*Lichen. Strophulus.*)

Causation and description.—The first of these affections is vesicular, that is, characterised by the development of vesicles upon an inflamed base; the second of them is generally regarded as papular—in other words, as due to the formation of solid pimples on an inflamed surface; the last is simply the lichen of children. Many modern authorities, however, now regard the various forms of eczema and lichen as merely varieties of the same disease, and strophulus *a fortiori* as a variety of eczema. We adopt this view, and combine them in a common description under the general name of eczema.

Eczema is an inflammation of the skin, for the most part much more acute in its phenomena than psoriasis, and attended with much more violent local irritation. It often commences with itching; but this is soon followed by the appearance of minute acuminated papules, which are more or less red from congestion, which may be either grouped in patches, or scattered, and which sometimes (but not by any means invariably) originate at the points from which hairs emerge. The papules gradually increase in size, sometimes retaining the solid form, sometimes being obviously vesicular almost from their first appearance. In the former case they may attain a line or more in diameter, when their acuminated character probably disappears; but more frequently perhaps they reach the average size of a millet-seed; and then, after they have remained out for a few days or a week or two, their redness fades, their surface desquamates, and they gradually subside. When the eruption is essentially vesicular, each papule (which is generally intensely inflamed) is occupied or crowned by a circumscribed accumulation of serum between the horny and the mucous layers of the epidermis. The vesicles are rarely larger than a poppy-seed, excepting when they are closely aggregated and neighbouring ones coalesce; under which circumstances a considerable area may get covered with a low undulating bleb, pinned down, as it were, here and there to the subjacent surface by the remains of the party-walls between adjoining vesicles. In this case, also, the eruption may subside at the end of a few days; but the appearances which attend its subsidence vary. Sometimes the contents of the vesicles become absorbed, and simple desquamation follows. More commonly the vesicles burst; and the exuded serum, mingling with the separating epidermis, coagulates into a scab, the character of which depends on a variety of circumstances—such as the part of the skin affected, the cessation or persistence of exudation, the entanglement in it of dirt or other foreign matters, and the admixture of blood or pus due to the effects of scratching or other local violence. In the simplest case the scab is often of a sulphur-yellow hue, and more or less powdery. More commonly perhaps it

is of a dark colour, scaly or gummy, and adherent to the surface. On the scalp the crusts are apt to accumulate and to form thick dirty laminæ.

Eczema is liable to become chronic. In some cases, especially in the papular form, the eruption then loses its vivid redness, and the surface gets thickened, rough, scurfy, and fissured. In some cases, and mainly such as are vesicular, large tracts of skin become red, excoriated and moist, and, on close examination, may be found to be covered in patches with a thin, opaque, soft, epidermic layer which is studded more or less abundantly, especially at the edges, with pits (very much like the perforations by which postage-stamps are separated from one another), at the bottom of which a red weeping surface is visible. These pits are excoriations and correspond to vesicles; and in such cases are probably the only representatives of vesicles which can be recognised. Again, even in vesicular cases, the inflamed surface often after a time loses its vesicular character, becomes uniformly inflamed, brittle and scaly, and assumes characters which, apart from the history of the case, are identical with those of chronic psoriasis or pityriasis rubra.

The vesicles or papules of eczema may be either scattered and discrete, or collected into circular or oval groups of small size, or aggregated in larger irregular clusters, which tend to run together—the intervening skin being at the same time studded with isolated spots. In the first of these cases the papular form of the disease constitutes *lichen simplex* or *strophulus intertinctus* (*red gown* or *red gum*); in the second, *lichen circumscriptus* or *strophulus volaticus*; and in the third, *lichen agrius*, or *strophulus confertus* (*rank red gum*). Eczema may be *acute* or *chronic*—the former lasting for a week or ten days, or more; the latter often consisting in successive outbreaks of the acute disease, but including those cases in which the skin assumes the features of psoriasis diffusa, and also the form commonly known as '*eczema rubrum*.' In the last there is general excoriation with intense redness, abundant exudation of serum, and the formation of numerous red oozing points in place of distinct vesicles. It is most frequently seen in typical completeness on the lower extremities of elderly persons.

No part of the surface of the body is free from liability to eczema. It attacks some parts preferentially, however, and then often receives a local epithet. Thus it frequently occurs upon the hairy scalp (*e. capitis*), constituting a very troublesome and chronic affection; on and in the ears (*e. aurium*); at the edges of the eyelids (*e. palpebrarum*); and on the cheeks (*e. faciei*). It is common too in the axilla and bend of the elbow, about the anus, pubes, and outer part of the thigh, and in the bend of the knee. The nipples of suckling women and the umbilicus of the newly-born babe are frequently affected. And it is not uncommon on and between the fingers. The affections known as '*grocers*'

itch,' '*bakers' itch*,' and '*warehousemen's itch*' are all of them eczema or lichen agrius of the backs of the hands and wrists.

Not unfrequently, when the eczematous inflammation is severe, spots of suppuration appear, intermingled with the original vesicles and papules; and the scales which result are thicker and darker than those of simple eczema. Eczema then approximates in its characters to impetigo, and consequently is often termed '*e. impetiginodes*.'

Eczema, in its various forms, is the most common of all skin diseases. It is of frequent occurrence in babes and young children; but no age is exempt; and it may break out for the first time in extreme old age. It is not an unfrequent attendant on pregnancy and lactation. It is sometimes distinctly hereditary; and a previous attack generally predisposes to subsequent attacks. Its causes are not very obvious; occasionally, however, it is clearly produced by local irritation—in the head by the constant use of hard brushes; in the nipples by the irritation of sucking; between the thighs and buttocks and analogous parts by the effects of the local secretions and by attrition; and in bakers and others by the irritating substances among which they work. Eczema is also frequently induced by the presence of scabies or pediculi. These, however, are not the only causes. It is often idiopathic, and is then not unfrequently preceded for a day or two by febrile symptoms. It is often ascribed to gout, dyspepsia, uterine complaints, teething, and the influence of weather and of climate.

Excepting in the case of the extensive diffusion of the acute disease, eczema is rarely attended with constitutional symptoms. Locally it is characterised by the presence of more or less itching, tingling, or burning. The itching in some cases, indeed, is unbearable.

Treatment.—There is no specific treatment for eczema; it is therefore especially important in every case to ascertain if possible the cause on which it depends, or whether or not the patient have any associated malady affecting the general health; and to treat it. Thus the constitutional treatment of eczema may resolve itself into the treatment of gout or indigestion; the local treatment into the destruction of insects, or the cessation from certain kinds of manual labour. Alkalies, such as liquor potassæ or the bicarbonate of potash or soda, in combination with vegetable tonics, are often resorted to. But the remedy on which most reliance is placed is arsenic. This is generally given in the same manner as in the treatment of psoriasis, and is by most physicians regarded as being most efficacious in the chronic forms of the disease. When febrile symptoms are present, mild laxatives and cooling medicines are desirable. Tonics are often beneficial in its later stages. It is well to pay attention to the diet. Alcoholic drinks are generally injurious, as also are rich foods and hot condiments. The local treatment must vary with the stage of the affection, its intensity,

and extent. In the acute stage, and always when there is much inflammation, cold-water dressings or evaporating lotions, or even the cold douche continued from ten minutes to half an hour at a time, are very useful. Under the same circumstances lead-wash, and such-like applications, are beneficial. At a later period, when there is much accumulation of scabs, it is important to remove them either by washing with soft soap and water, or poulticing, or the saturation of the part with olive-oil. Then the surface must be kept clean; and mild mercurial ointments, or ointments containing lead or zinc, may be gently applied after each washing. In the dry and scaly condition of eczema which simulates psoriasis, the treatment applicable to the latter affection may be employed. Hebra recommends for some cases the rubbing in of liquor potassæ until it acts chemically on the diseased structures, for the purpose both of removing the morbid surface, and of promoting more healthy action. The caustic is applied once a week, the parts being treated with water-dressing in the intervals. Over limited areæ of disease, the application of the solid nitrate of silver sometimes effects a cure. As a rule, however, we think that soothing local treatment, combined with cleanliness, will be found most efficacious. And although soap may be occasionally employed to aid in the removal of scabs, persistence in its use is generally injurious. The patient should use, instead of it, bran, oatmeal, starch, milk, or yolk of egg.

VIII. IMPETIGO. (*Ecthyma.*)

Causation and description.—The affections comprised under these names are essentially pustular; we regard them as being identical, and shall describe them as varieties of impetigo.

Impetigo is a disease which consists in the formation of pustules at the surface of the skin, either between the cutis vera and epidermis, or between the corneous layer of the epidermis and the rete mucosum. The development of pustules is generally attended with more intense inflammation than that of vesicles or papules; and pustules are, for the most part, surrounded by well-marked congested areolæ, and situated upon more or less distinctly thickened bases. They occasionally commence in vesicles or papules, and thus eczema or lichen may pass into impetigo. Most commonly, however, they originate in spots of inflammation—stigmata, papules, or tubercles—in which suppuration is manifest almost from the beginning. The pustules vary in size from that of a pin's head (or less) to that of a split pea or bean. They are generally round or oval in outline, but sometimes irregular and angular, and project in the form of an oblate hemi-

spheroid. At the end of a day or two they break, or their contents concrete, and scabs are formed, which are generally thicker and darker than those of eczema; but which, nevertheless, vary very much in colour and consistence, being sometimes softish, translucent, and honey-like, sometimes dark, opaque, and tough. If the progress of the pustules be favourable, the scabs separate after a few days, leaving reddish spots behind them, which are soon effaced by the completion of a normal layer of epidermis. Very often, however, the scabs become detached while the subjacent surface is still secreting pus; and not unfrequently, when the scab seems fully formed, suppuration still goes on beneath and around it, leading on the one hand to a deeper erosion of the skin, on the other hand to the lateral extension of the pustule by the gradual undermining of the surrounding epidermis and the incorporation of the successive circles of suppuration thus formed. In the latter cases the local progress of the disease may be maintained for a long time; and in these alone, but rarely even here, is there danger of the production of permanent cicatrices. The long continuance of impetigo sometimes leads to permanent harshness, muddiness, and deterioration of the skin.

The lymphatic glands in relation with the part affected by the disease generally get inflamed, large and tender, and occasionally suppurate.

The pustules of impetigo sometimes come out singly (*i. sparsa*), sometimes in groups (*i. figurata*); and the groups may be of considerable extent. In the former case the pustules are generally larger than those of the grouped variety; and if the subjacent thickening and surrounding inflammation be considerable (as they are very apt to be when the pustules are seated on the buttocks or lower extremities, and in adults), the affection is often termed '*ecthyma*.' In the latter case the congestion connected with the several adjoining pustules blends, and thus forms a common area of inflammation which is often very intense (*i. erysipelatodes*). The scabs also, under such circumstances, are apt to run together and form a continuous mass or lamina (*i. scabida*).

Impetigo occurs on all parts of the surface. It is common on the head and face, especially of young children, and when abundant and confluent in the latter situation is sometimes called '*porrigo larvalis*.' Occasionally it attacks the hairy parts of a man's face, and then constitutes one variety of the affection termed '*sycosis*.' It is then very intractable, owing probably to the fact that the root-sheaths of the hairs are specially involved. It is met with frequently about the buttocks, and indeed on all parts of the trunk and extremities.

Impetigo is liable to spread by inoculation: thus it may be conveyed from the child's head or face to the fingers with which it scratches itself; or from the nursling's face to the mother's bosom or hands; or, again, from child to child in families or schools. Some-

times it appears to originate idiopathically, and to be preceded by feverish symptoms, lasting for a day or two; it may be a subsequent development of lichen; and is very often produced by local irritation, arising from pediculi, acari, and even mechanical causes. It is common during the period of dentition. The duration of impetigo is very various, depending partly on the cause, partly on the health of the patient, and partly upon hygienic conditions. The acute form may subside at the end of a week or two; but the disease is very apt to be chronic, and kept up for months, and even years, by successive acute outbreaks. Those who have had previous attacks are liable to suffer from relapses. The constitutional symptoms are generally trivial; there is often, however, some degree of fever when the affection is extensive and acute, especially if the lymphatic glands are implicated. There is generally some itching and tingling of the parts affected.

Treatment.—The local treatment of impetigo differs but little from that of eczema. In quite the early stage the application of cold or tepid water, or cooling lotions, is useful. When scabs have formed it is always important to effect their removal, and this may be accomplished in the same way as in eczema. After their removal, the application of lead or zinc lotions, combined with glycerine, or of mild mercurial ointments, is generally sufficient. Caustics are rarely beneficial, or even admissible. When the hairy parts are affected it is always well to have the hair cut short; and in the case of sycosis it is generally necessary to resort to epilation. It is always important to treat any associated malady under which the patient is labouring, and which may be affecting his general health. But as a rule, tonics are indicated, especially iron, mineral acids, quinine and other vegetable bitters, and cod-liver oil. Change of air is often of great benefit.

IX. SUDAMINA. MILIARIA.

Description.—These names are employed to designate the minute vesicles which appear scattered over the surface of the chest, back, flanks, and sometimes upper arms and thighs of persons who are perspiring profusely, or more frequently perhaps of those who, having had a dry skin for some time, commence again to perspire. Thus we meet with them in rheumatism, pneumonia, and many fevers at the commencement of convalescence. They form at the orifices of the sweat-glands, and are due mainly to the imprisonment of minute drops of sweat by the horny layer of the cuticle. They are generally about as large as pins' heads, round or irregular in shape, containing a colourless acid fluid with leucocytes, and quite unattended with inflammation. They can be easily felt as small, prominent, hard bodies; but very often escape the eye unless carefully looked for, and then appear like minute

drops of melted white wax. They end in branny desquamation. Occasionally their contents are opaline and of alkaline reaction, and each vesicle is surrounded by a narrow halo of congestion. It is to such lesions presenting these characters that the term '*miliaria*' is sometimes, but unnecessarily applied. No treatment is required.

X. HERPES. PEMPHIGUS. (*Pompholyx.*)

Causation and description.—Herpes and pemphigus are vesicular or bullous affections, yet there is a very close affinity between them and erythema, especially erythema multiforme; and indeed it is questionable whether it might not have been best to discuss them all under the same heading. Both herpes and pemphigus become developed upon erythematous patches; and not unfrequently these patches are papulate, discoid, circinate, gyrate, or marginate, and consequently the vesicular or bullous eruption assumes corresponding characters. Indeed, in no inconsiderable proportion of cases, erythema, herpes, and pemphigus represent simply successive stages of the same affection. Various causes have been assigned for herpes and pemphigus, and among them one which is of great interest—namely, some affection, probably irritative, of the sensory nerves. One species of herpes—herpes zoster—is, as we shall presently show, always limited to the area of distribution of some one or more of the nerves of common sensation, and usually attended with intense neuralgic pain; and, moreover, erythematous, vesicular, and bullous eruptions are shown by various authors, and especially by Charcot, to be common accompaniments of pachymeningitis of the cord and of other conditions causing equivalent irritative effects in the cord or nerves connected with it.

A. *Herpes.*—By this term we understand an affection characterised by the development of clustered vesicles, varying between the size of a small pin's head and that of a split pea, and seated on an erythematous base. A circumscribed area of redness, round, oval, or irregular in shape, first makes its appearance. This soon becomes thickly studded with papules, which speedily acquire a vesicular character, and in the course of twenty-four hours or less attain their full dimensions. The vesicles are very close-set, and not unfrequently run more or less together, so as sometimes to form large bullæ. Their contents are in the first instance limpid and pale; but they often become dark from admixture with blood, or opaque and yellow in consequence of suppuration. After two or three days they begin to dry up, and then form thinnish dark-coloured or gummy scabs, which in a few days more become detached, leaving a whole but slightly reddened surface behind. There is always much heat and tingling or stinging during the earlier

stages of the disease. Its total duration is rarely more than two or three weeks, and often considerably less.

Several forms of herpes are enumerated by dermatologists. We proceed to discuss the more important of them :—

1. *Zona*, or *herpes zoster* (*shingles*).—This is the most important and striking affection of the group. It is characterised by the formation of clusters of vesicles on inflamed patches of various forms, and ranging from the size of the palm of the hand to that perhaps of a split pea. The clusters appear almost simultaneously, and irregularly scattered, over the area of distribution of one of the cutaneous sensory nerves. Hence they always occur within certain definite limits and on one side of the body only. In addition to the general characters of herpetic affections, zona is apt to be attended with certain special peculiarities. Thus it is often associated with severe neuralgic pains in the neighbourhood of the part affected, which sometimes precede, sometimes accompany, and sometimes follow the cutaneous eruption, and often last for many weeks ; and again the inflammation is apt to be intense and to penetrate deeply, and hence to be slow of disappearance and to leave permanent scars, and sometimes (especially in the old and weakly) to become gangrenous.

The most frequent seat of zona is the chest or abdomen, where it takes the course of the cutaneous branches of one of the intercostal nerves. But it is not uncommon elsewhere, though it is very often then not recognised as zona. Von Bärensprung enumerates nine varieties ; and it would be possible, but is not necessary, to enlarge their number; they are as follows :— *z. facialis*, where the parts supplied by the fifth pair are affected, the surface of the conjunctiva being sometimes involved ; *z. occipito-collaris*, following the distribution of the occipitalis minor, auricularis magnus, superficialis colli, and occipitalis major ; *z. cervico-subclavicularis*, corresponding to the descending superficial branches of the cervical plexus (supra-sternal, supra-clavicular, and supra-acromian) ; *z. cervico-brachialis*, affecting surfaces supplied by branches of the brachial plexus—namely, the shoulder, upper arm, fore-arm, and hand ; *z. dorso-pectoralis*, corresponding to the third, fourth, fifth, sixth, and seventh dorsal nerves ; *z. dorso-abdominalis*, corresponding to the eighth, ninth, tenth, eleventh, and twelfth dorsal nerves ; *z. lumbo-inguinalis*, corresponding to the branches of the upper lumbar nerves, and extending from the loin to the linea alba, involving also the pubes and genital organs, the gluteal region and outer aspect of the thigh ; *z. lumbo-femoralis*, corresponding to the cutaneous branches of the second, third, and fourth lumbar nerves, more especially the external cutaneous, genito-crural, anterior crural, and obturator, and affecting therefore mainly the anterior and lateral surfaces of the thigh and the inner aspect of the leg and foot ; and lastly, *z. sacro-ischiaticus*, which follows the cutaneous branches of the sacral plexus.

Zona attacks persons of all ages, but chiefly, it is said, young adults. It is held by some to be most common in spring and autumn, and also to occur only once in a lifetime. It is questionable, however, whether either of these statements is true. Its connection with nervous irritation has been already referred to; but nothing more in reference to its causation is known.

2. *Herpes simplex.*—This name may be conveniently used of those cases in which a group of vesicles or several such groups appear, so to speak, casually in some limited area, which then commonly gives a specific name to the affection. Thus we have *h. labialis*, affecting the lips and neighbouring parts; *h. palpebralis*, the eyelids; *h. auricularis*, the pinna of the ear; and *h. præputialis* and *pudendalis*, respectively the prepuce and the labia. In these cases the patches of disease are identical in appearance and progress with those of zona. But there is nothing to indicate that they have any connection with sensory nerves. Moreover, some of them (especially *h. labialis*) are especially apt to attend an ordinary catarrh, and to come on in the course of acute pneumonia.

3. *Herpes iris* is the designation of an eruption of vesicles which arise in series of concentric rings upon a gradually enlarging erythematous disc. It is most frequently observed on the backs of the hands and wrists, feet and ankles, but is sometimes much more generally distributed.

4. *Herpes circinatus* is the name applied to an inflamed disc, which gradually increases in size, and whose enlargement is accompanied by the formation of a ring of vesicles at the circumference, while the centre for the most part gradually returns to a state of health.

It is obvious, as we have already pointed out, that there is no essential difference between the last two varieties or between them and erythema multiforme; and that h. iris and h. circinatus are simply later phases of e. iris and e. circinatum. It may be added that intermediate papular conditions are sometimes observed, to which the names of lichen iris and l. circinatus might (unnecessarily indeed) be applied.

It is important, however, to bear in mind that the name 'herpes circinatus' is often given to the specific eruptions of favus and ringworm, and that the multiform erythematous and vesicular affections which have just been considered (though not themselves parasitic) are very apt to be simulated by and confounded with these vegetable parasitic diseases.

Lastly, cases are occasionally observed in which erythematous patches, irregular in form and size, appear almost simultaneously over the whole cutaneous surface, and become speedily covered with herpetic vesicles which tend to run together. The patches individually are like those of herpes zoster; and, moreover, like herpetic patches generally, run through all their stages in a week or two; but they differ from them in their wide distribution.

Herpes iris, h. circinatus, and the form of herpes last described resemble in their symptoms the corresponding forms of erythema multiforme. They are usually of trivial importance, but occasionally, when of extensive distribution, are attended with much febrile disturbance.

B. *Pemphigus.*—This term comprises most of those inflammatory affections of the skin which are attended with the formation of bullæ or blebs. These sometimes attain the size of a hen's or duck's egg, and are developed on round, oval, sinuous, or irregular surfaces. But associated with such blebs, we often find single or grouped vesicles, no larger than those of herpes. Hence the blebs of pemphigus may be considered to vary between these limits. There is nothing specific, however, in the formation of a bleb; any patch of erythema, or other forms of inflammation, or of gangrene, may become studded with vesicles, and any number of contiguous vesicles may run together and form a common cavity. It follows almost necessarily that there is nothing specific in the conditions to which the term pemphigus is applied, and that the limits between them and affections receiving other names are to a great extent arbitrary.

Pemphigus is not unfrequently (as has been pointed out above) the fully-developed stage of herpes iris, h. circinatus, and other forms of generally distributed herpes. The stages of the disease are then well marked—the first being the appearance of a disc, ring or irregular patch of erythema; the second, the formation of small vesicles, sometimes in a ring at the circumference, sometimes in the centre, sometimes generally over the surface; and the third, the extension or blending of these vesicles and the evolution of a prominent bulla, the edges of which become, for the most part, conterminous with those of the erythematous redness. Owing to the coalescence of neighbouring patches of erythema, neighbouring bullæ may coalesce into sinuous or gyrate bullous bands several inches in length. Further, the eruption may be sparse or limited in extent, or it may be general and abundant. The full development of the disease may occupy three or four days, or more, but is often much more rapid. In cases of this kind it sometimes happens that extensive tracts of surface become erythematous and remain so for some considerable time, vesicles and bullæ from time to time appearing here and there upon them. In other cases of pemphigus, the formation of bullæ is almost coetaneous with the appearance of the erythema, which may then indeed escape recognition as a separate stage of the affection, both being preceded by violent itching, stinging or burning. The bullæ of pemphigus are generally plump and distended with a pale straw-coloured serum, which, after a while, gets darker in tint or milky and opalescent. After a few days the contents begin to disappear by evaporation and absorption, or the bullæ rupture and they escape. Then a thin dry pellicle, consisting of the epidermis which had been raised up and of coagulated

exudation, forms upon the affected surface, and after a few days more becomes detached, leaving a sound but somewhat reddened area behind. Sometimes, especially if the part have been irritated by scratching or otherwise, or if the general health of the patient be bad, the scab more resembles that of eczema or impetigo, probably re-forms after removal, and convalescence may be very much protracted. Ulceration or even gangrene may ensue.

As will be gathered from the foregoing account, pemphigus presents a good many varieties. Sometimes it is *acute*, its entire duration being comprised within a period of three or four weeks. More frequently it is *chronic*—chronic, however, in the sense in which urticaria evanida is chronic—that is, prolonged by successive acute attacks. It is then often termed *p. vulgaris*. Sometimes a single bulla breaks out suddenly, to be followed on its subsidence by a second, and then by a third, and so on (*p. solitarius*). A form of the disease, termed *p. infantilis*, is occasionally met with in newly-born children; large bullæ form on the neck, behind the ears, on the buttocks, genitals, wrists, and other parts, and for the most part progress unfavourably, ending in suppuration, ulceration, and gangrene. A further variety is that called by Alibert *p. foliaceus*. It is characterised by the successive formation of bullæ of small size, which are generally flat and flaccid, and the contents of which become more or less distinctly purulent, and dry up into thick yellow flaky scabs. These on separation leave a deeply congested weeping surface. P. foliaceus is said to spread gradually until it occupies the entire surface of the body, and never to be cured.

The causes of pemphigus are not clearly known. There is reason, however, to believe that in some cases, especially in that of p. infantilis or when it occurs on the soles or palms, the origin is syphilitic. And, as we have already pointed out, it appears in some instances to be connected with affections of the spinal cord or sensory nerves. The symptoms which attend its progress vary. There is often some degree of fever—sometimes high fever, the temperature reaching 104° or 105°; and when the affection is much prolonged, debility and emaciation may ensue. This latter is especially the case in the foliaceous form. Newly-born children affected with pemphigus generally succumb speedily. In many cases the patient's health remains apparently unimpaired throughout the whole course of the malady.

Treatment.—Whatever its form may be, herpes seldom requires special treatment. Cooling lotions, simple ointments, and protection of the affected parts against rubbing, include all the local measures that are usually necessary. The only important object to aim at in the treatment of zona is the relief of the severe neuralgic pain which is so often associated with it. For this various measures may be tried, such as the local application of blisters or other counter-irritants, the inunction of belladonna, or of aconitia ointment, or the use of leeches; and,

besides these, morphia or other sedatives administered by the mouth or hypodermically.

The bullæ of pemphigus require little local treatment. They may be punctured and their contents permitted to escape; but it is unadvisable to allow the cuticular pellicles covering them to get detached. For this reason, among others, it may be necessary to protect the parts with simple ointments spread on lint. For internal treatment iodide of potassium and mercurial preparations should be employed when syphilis is suspected. Arsenic is much lauded by some. In most cases, however, tonics are sooner or later indicated.

XI. RUPIA.

Causation and description.—Rupia is described as beginning with flat bullæ, rarely, if ever, exceeding half an inch in diameter; first containing clear serum; then producing very thick greenish brown or dark-coloured scabs, and deep destructive ulceration. In some respects, therefore, the disease resembles pemphigus; but it differs from all ordinary forms of pemphigus in the fact that its bullæ are the result, not of superficial, but of deep-seated disease. Rupia, indeed, is to be distinguished less by the occurrence of bullæ than by the character of its post-bullous stages. The rupial bulla slowly increases in size, is surrounded by a halo of congestion, and seated on a slightly thickened base. A scab soon forms, but while it is forming the bulla spreads at its margin, and fresh matter, which also soon coagulates, is produced around and under the first formed scab. In this way the rupial sore increases in diameter, the scab increases in thickness and prominence, and the subjacent ulcer becomes deeper and deeper. The resulting scab is always very thick, but sometimes flat and flaky, something like an oyster-shell (*r. simplex*), sometimes conical, like a limpet-shell (*r. prominens*), sometimes irregular and rocky in form. On its removal, a fresh scab usually forms. Rupial ulcers are always deep and unhealthy-looking, and cause much destruction of tissue and permanent cicatrices. In some cases, and especially in children, the ulceration extends rapidly, assuming a phagedænic character (*r. escharotica*), or becoming distinctly gangrenous, when it is sometimes termed *pemphigus gangrænosus*. Rupial sores are generally scattered and few in number, and are not limited to any particular part of the person. They are, perhaps, most common on the buttocks and lower extremities.

Rupia rarely, if ever, occurs in persons who are not obviously weakly and cachectic, and most frequently in those who have previously had syphilis. Indeed, there is some reason for regarding true rupia as essentially a syphilitic disease.

Treatment. — In the constitutional treatment of rupia, tonics of various kinds, iron, mineral acids, vegetable bitters, cod-liver oil, together with good diet and change of air, are all-important. Anti-venereal remedies must not, however, be forgotten, especially if there be a clear syphilitic history. For local treatment, poultices are necessary to aid in the detachment of the scabs; and the resulting ulcers must be treated not only with poultices but with stimulating or detergent ointments or washes, and even in some cases with undiluted caustics, such as nitrate of silver, nitric acid, acid nitrate of mercury, or other such agents.

XII. SEBORRHŒA. ACNE.

Causation and description.—By *acne* is meant an inflammatory affection of the sebaceous glands, dependent on, or at all events connected with, retention of their secretory products. In most inflammations of the skin the sebaceous glands of the parts affected share in the inflammation; and always in acne there is more or less tendency for inflammation to extend from them to the contiguous structures. Hence, as might be supposed, acne occasionally (and especially in some of its forms) passes into other recognised varieties of inflammation of the skin. Further, inflammation of the sebaceous glands is sometimes attended, not with retention of secretion, but with increased production and flow, so that we may have an inflammation of them which is not acne. This is sometimes named '*seborrhœa.*'

A. *Seborrhœa.*—The secretion of sebum in some persons is naturally excedingly profuse, but it is not therefore morbid, and becomes seriously inconvenient only in the absence of scrupulous personal cleanliness. In some cases, however, an excessive production of sebum occurs over certain limited areæ, attended with distinct hyperæmia of the parts, and more or less obvious hypertrophy of the glands. The increased production is limited in fact to patches of distinct erythema. This affection is not unfrequent in the scalp and on the face, especially in children. The secretion is usually more solid than sebum should be, and with the superficial epidermis concretes into greasy flakes, which adhere to the surface. A condition is thus produced which differs little, and not essentially, from pityriasis of the same parts. More rarely the secretion is quite fluid, and may be seen, after cleansing the surface, to form a minute drop at each glandular orifice. This condition, which is occasionally observed on the cheek and nose, is apt to be chronic, and sometimes becomes permanent.

B. *Acne.*—The unnatural accumulation of sebaceous matter in the sebaceous glands is of extremely common occurrence. It may be met with in glands which are still patent, as well as in those whose mouths are obliterated. In the former case the orifices are dilated and pro-

minent, and occupied by the dirt-blackened superficial portions of the accumulated sebum, the whole of which may, by squeezing, be removed in the form of small, maggot-like bodies (*comedines*). In the latter case no orifices generally are detectable, the sebum retains its normal yellowish hue, and concretes into hard, pearly, laminated masses. This condition was termed by Willan '*strophulus albidus*.' A small incision is generally necessary for their removal. *Sebaceous tumours* or *wens* differ little except in size and the consistence of their contents from the bodies last named.

When such accumulations of sebum are associated with inflammation of the parts immediately surrounding them, we have that condition present to which the term '*acne*' is generally applied. Acne, therefore, may occur in two forms. In the one, there is circumscribed inflammation, attended with induration, prominence, and duskiness of tint, but the cause of inflammation is rendered obvious by the fact that at the most prominent part of the tubercle there is a dilated sebaceous orifice, choked with the secretion of the gland. In the other form the orifice of the gland is undistinguishable, the accumulation is deep-seated, inflammatory products are diffused around, beneath, and superficial to it, and thus an indurated congested prominent tubercle is produced, which yields on inspection no visible proof of its connection with sebaceous accumulation. The tubercles of acne vary in size, and are sometimes as large as a horse-bean. They often suppurate, but, especially in the latter form, suppurate very slowly, leading before they discharge their contents to a good deal of localised disorganisation, and eventually to the production of permanent scars. Their contents are scanty but thick, and consist partly of sebaceous matter, partly of pus.

Different forms of acne are described, of which the majority are mere varieties of the same condition, and are generally combined in various proportions in the same case. The term '*a. punctata*' is often applied to that very common condition in which the sebum simply accumulates in the follicles, and leads by its accumulation to the production of a series of black-tipped papules. By '*a. simplex*' is generally understood a. punctata associated with inflammation and suppuration —the papules being surrounded by congestion, and often going on to the formation of small superficial abscesses, which in a short time discharge their contents, and then after a few days, or a week or two, heal up. The name *a. indurata* is given to those cases which are marked by general enlargement and induration with dusky or livid discolouration, and slow deep-seated suppuration. It must be added that one form of *sycosis* is distinctly a. indurata of the hairy regions of the face.

Any part in which sebaceous glands exist may be the seat of acne. But it is most common on the face, especially the forehead, cheeks, nose, and chin; and on the trunk, mainly between the shoulders and

on the chest. It rarely occurs in young children, excepting in the form of *strophulus albidus*. It is most common in both sexes about the period of puberty, and from that time onwards to two or three and twenty. It is frequently met with, however, and then especially in its indurated form, in persons of middle and even advanced age. The causes of acne are obscure. It is certain, however, that the tendency to it runs in families, and that it has a special connection with the period of development and maturation of the sexual functions.

C. *Acne rosacea*.—The condition to which this name is commonly given has been regarded by most modern authors as a mere variety of acne. Hebra, however, maintains that it is essentially distinct from acne, although frequently associated with it. It generally consists in more or less extensive patches of inflammatory redness, associated with slight infiltration of the affected cutis and visible dilatation of the superficial vessels, and also with the presence here and there upon the inflamed patches and in their neighbourhood of tubercles corresponding precisely to the description already given of those of acne indurata. The affection is really therefore an inflammatory condition of certain parts of the skin, in which there is a special tendency for the sebaceous glands to be implicated. Acne rosacea is limited to the face, affecting sometimes the nose, sometimes the cheeks, sometimes the forehead, sometimes the chin, but generally several of these regions at the same time. It is for the most part symmetrical in its distribution, and tends gradually to extend. It usually begins with circumscribed hyperæmia of the nose or cheeks, often attended with an increased secretion of sebaceous matter, and generally with a more or less obvious development of dusky red tubercles, which may or may not suppurate. This condition, variable at first, soon becomes permanent, the cutis getting infiltrated and thickened, the small veins of the part dilated and tortuous, the tubercles more abundant and larger, and the face consequently much disfigured. In this latter state the disease may remain for many years, or for life, without material change. But in some cases, and more especially in elderly men who have been addicted to alcoholic excess, the affection, which is then almost invariably limited to the nose and its immediate neighbourhood, assumes a hypertrophic character; the parts which were originally affected with a simple form of acne rosacea become swollen and tuberculated, until in some instances the nose forms a huge misshapen, lobulated, pendulous mass. These changes are due to inflammatory hyperplasia of the cutis vera, the tissues subjacent to it being rarely, if ever, implicated. The sebaceous glands, however, are involved and hypertrophied, sometimes still discharging their products through the yet patent ducts, sometimes from obstruction allowing accumulation of sebum, and perhaps undergoing suppuration. The affected parts become deeply congested, and the dilated varicose veins larger and more numerous.

Beyond heat and flushing, which are liable to frequent exacerbations, little local inconvenience or discomfort attends acne rosacea in any of its forms.

Acne rosacea, in its simpler variety, is an affection of adult life, coming on generally between 25 and 30, but sometimes making its appearance for the first time after the age of 40. It is far more common in women than in men. The hypertrophic variety of the disease, on the other hand, is rarely observed in women; and it attacks the opposite sex for the most part in middle age or the decline of life. The causes of hypertrophic acne rosacea are not in all cases obvious; there is no doubt, however, that in large proportion it is traceable to long-continued habits of intemperance, or over-indulgence in spirituous liquors. The difficulty of assigning a cause to the other form of this affection is still greater; nevertheless, it is certain that many of those who suffer from it are dyspeptic or liable to uterine disturbances, and that when any of these complications are temporarily present there almost invariably occurs marked exacerbation of the facial inflammation.

Treatment.—In seborrhœa plentiful ablution with soap and water, and the use of astringent lotions, containing acetate of lead or sulphate of zinc, or of mercurial preparations, are the chief measures to be employed. Constitutional treatment is generally useless.

In treating acne, it is of great importance to insist on frequent and thorough washing with soap and warm water, to be followed by the friction of a rough soft towel, or flesh-brush. These measures, however, are even more important to prevent than to cure. All black spots should be removed, either by squeezing the papules in which they are contained between the nails, or by pressing down upon them a ring, a little larger than the black spot, and including it. The mouth of a watch-key answers the purpose very well. Superficial collections of matter should be punctured, and discharged. The chronic tubercles of acne indurata should be opened with a narrow-bladed knife, and have their contents expressed, or should be touched at the summit with the acid nitrate of mercury, or some other equivalent escharotic. The local inflammation may be allayed to some extent by the use of lead-wash, or lotions containing from two to four grains of sulphate of zinc, or from half a grain to two, three, or even four grains of bichloride of mercury to the ounce. Mild mercurial ointments are sometimes useful. Sulphur, in the form of ointment or lotion, is strongly recommended by most dermatologists. In our general treatment we can aim only at improving the general health, and must be guided, therefore, solely by the general symptoms which the patient presents.

In sycosis it is important to have the hair of the affected parts kept closely cut, and to remove the hairs running through the tubercles or pustules by frequently repeated epilation. Hebra insists on the necessity for keeping the surface constantly shorn, for the application

of sulphur and other stimulating ointments, and for the incision of the inflamed tubercles.

The treatment of acne rosacea differs little from that of simple acne. But it is especially important here to attend to the general health and habits of the patient, to remedy indigestion, to remove anæmia, to prescribe a wholesome unstimulating diet, and to maintain the healthy functions of the skin and other organs. The local treatment is absolutely that of acne simplex; but it generally needs more persistent employment.

XIII. LUPUS. (*Noli me tangere.*)

Causation and description.—The term 'lupus' is applied to a series of affections characterised by a specific overgrowth of the cutis, for the most part of chronic progress, and resulting in the formation of indelible cicatrices, or in more or less extensive destruction of tissue.

Lupus usually commences with more or less distinct congestion and hypertrophy of a limited area, which, in a large proportion of cases, is studded with solitary or grouped lenticular tubercles a line or two in diameter, and presenting a slightly translucent aspect and a dull red or pale salmon colour. The patch of congestion slowly increases in area or the tubercles in number, until in many cases a large extent of surface after a while becomes involved. While this extension is in progress various changes take place. In some instances, the parts first implicated, without attaining any further stage of development, gradually lose their inflamed and hypertrophic character, but instead of simply reverting to the healthy condition, become pale, depressed and contracted, and assume a cicatricial character. In some instances, previously to the attainment of this cicatricial termination, their surface yields adherent scales, or crusts. In some, the tubercles, almost from the beginning, are the seat of suppuration, and become crowned with thick adherent scabs. In some, extensive ulceration ensues, with grievous and irremediable destruction of parts. In its morbid anatomy lupus appears to consist in the development of a kind of tissue, resembling granulation tissue, composed of small cells, imbedded, according to the density of the growth, in a greater or smaller quantity of fibrous material. Lupus is generally regarded as a scrofulous disease; and it not unfrequently occurs in those who are suffering or have suffered from scrofulous suppuration of the cervical glands, or who are otherwise out of health; moreover exacerbations seem not unfrequently to be induced, in those who are already its subjects, by temporary conditions of general ill-health. Females suffer from lupus much more frequently than males, children than adults, and the poor than the well-to-do. The local symptoms which

attend its progress are for the most part trivial; often the patient makes no complaint, or, if he complains at all, complains only of itching or tingling.

In accordance with the different peculiarities of character and progress which have been above referred to, several varieties of lupus have been described, the more important of which we shall now briefly discuss.

A. *Lupus erythematosus*, which was first described and named by Alibert, is the least severe form of the disease. It occurs mainly on the cheeks, nose, forehead, and scalp, but is not limited to these parts; and it makes its appearance there in the form of rounded erythematous patches, which slowly increase in diameter, and may at first be readily mistaken for patches of simple erythema. But sooner or later they get covered with either thin scales or thick crusts, composed largely of sebaceous matter and continuous by their under surfaces with processes of the same material prolonged into the dilated orifices of the subjacent sebaceous glands. In the former case the affection simulates psoriasis; in the latter, that morbid condition of the knuckles caused by dissection, to which Dr. Wilks has given the name of '*verruca necrogenica*.' The progress of lupus erythematosus is very chronic, and scarcely attended with any abnormal sensations, but when it subsides it leaves behind it permanent changes in the condition of the skin. It usually begins in adult life, and affects women more commonly than men.

B. *Lupus exedens* and *non-exedens* (*tubercular lupus*).—Lupus non-exedens, like the last, may occur on any part of the surface of the body, but usually originates on the nose or cheek. It commences with the appearance of small tubercles, such as have been above described; which slowly increase in number, sometimes assuming an annular arrangement, and involve more and more of the contiguous cutaneous surface, sometimes extending to the mucous membranes, and especially to that of the nose. Their course is very uncertain. Sometimes, after making but little progress, they slowly subside. More frequently they advance irregularly, now remaining quiescent for a while, now undergoing comparatively rapid extension, and thus, continuing for years, ultimately involve extensive tracts of skin. These become seamed and puckered, and of a greyish white colour in those parts which have undergone involution, and present groups of reddish tubercles in those which are still extending. In the progress of tubercular lupus, the tubercles not unfrequently become covered with scales or crusts, below which gradual erosion is going on, or undergo actual suppuration or ulceration with the formation of scabs. In some cases the tendency to suppurate or ulcerate, and to scab, forms a special feature in the disease, which then receives the name of '*lupus exedens*.' This leads to more or less rapid and extensive destruction of tissue, and when occurring (as it most frequently does),

in connection with the nose, often involves the gradual loss of more or less of the septum nasi and cartilages which bound the nostrils. The cicatrisation to which lupus non-exedens, and still more that to which the exedent form leads, is not merely in a high degree disfiguring, but often induces serious consequences. The eyelids become retracted, the nose curiously thin and pointed, the alæ contracted and the nostrils altered in shape, the mouth distorted, and the lower lip and chin drawn down upon the chest, as they sometimes are after extensive burns. The forms of lupus here described usually begin in early life, but are often prolonged by successive outbreaks up to an advanced age.

C. *Pustular lupus.*—This variety of the disease simulates impetigo. It is sometimes limited to the face, and has then been termed by Mr. Startin 'impetiginous lupus.' Sometimes, however, the whole surface —head, face, trunk, limbs—becomes more or less thickly covered with it. The eruption consists of tubercles, which are mostly discrete, but are here and there collected into confluent patches, which vary from $\frac{1}{4}$ to $\frac{1}{6}$ inch in diameter, tend to suppurate scantily at their most prominent points, and presently become crowned with small dark, hard scabs, deeply imbedded, and remaining fixed (unless detached by violence) for weeks or months. The detachment of one scab is liable to be followed by the formation of another; but sooner or later each tubercle gets absorbed, leaving behind it a temporary livid discolouration and a permanent depressed cicatrix. Pustular lupus is often associated with the presence of suppurating scrofulous glands.

Treatment.—In the treatment of lupus, constitutional remedies hold an important place. Among these the most efficacious are cod-liver oil, quinine, iron, and other forms of vegetable and mineral tonics, and arsenic. If there be a suspicion of syphilis (and it is often extremely difficult to distinguish non-specific lupus from some forms of tubercular syphilide) the ordinary anti-syphilitic remedies must not be omitted. Change of air is often valuable. Local remedies are very variable in their effects; sometimes they seem to do more harm than good, sometimes their use appears to be followed by rapid amendment. In the tubercular form of the disease, especially if the tubercles be attended with ulceration or any other form of destructive process, the use of solid nitrate of silver, potassa fusa, acid nitrate of mercury, or arsenical paste (made according to Mr. Startin's formula with three parts of arsenious acid, two parts of bisulphuret of mercury, and one part of calomel, together with water) is often highly advantageous. The caustic, however, needs to be repeated from time to time, and previous to its application the surface should be freed from scales and scabs. In the milder cases less severe local applications are usually indicated, such as nitric acid lotion, iodine paint, blistering fluids, or mercurial, lead, or zinc ointment.

XIV. KELOID. (*Kelis.*)

Causation and description.—This affection was first described and named by Alibert. It is characterised by the gradual formation of roundish, elongated, linear, branching, or reticulate patches, which are elevated a line or two, or even more than that, above the general surface, and appear to be mainly a hypertrophic condition of the cutis. The patches vary in colour, but are usually either white and shining, or of a more or less rosy hue, and are often marked with vascular ramifications. They present for the most part a smooth and rounded surface, and generally send out here and there claw-like processes or spurs which gradually lose themselves in the surrounding healthy skin. It is from this peculiarity that their name was derived, and that they acquire their generally recognised resemblance to hypertrophic scars. They are dense and firm in consistence, and never become covered with scales or crusts, or undergo ulceration or other such destructive changes. They are sometimes attended with tingling, itching, or burning, and are often tender to pressure. Their progress is slow; they usually extend gradually for a time, and then are apt to become stationary; occasionally they undergo involution, and disappear. In the early stage of their development they consist largely of fusiform cells, and are by Virchow and others regarded as sarcomatous; at a later stage they become almost entirely fibrous.

Keloid commonly occurs in isolated patches of various sizes on the chest or back; but it may be multiple, and may be met with on any part of the surface, even the face, ears, genital organs, and extremities. Occasionally it involves nearly the whole of the trunk.

The causes of the disease have not been clearly determined. It occurs, however, mainly in adults, and seems not unfrequently to be induced by local irritation or injury. Indeed, one form of it, which is generally termed '*false keloid*,' seems to be clearly due to hypertrophic changes occurring in connection with ordinary scars.

The *treatment* of the disease is unsatisfactory. The growths, when large, have occasionally been removed with the knife, but the results have not been encouraging. Local applications, such as iodine paint, blistering fluid, and various forms of stimulating ointments, have been tried and recommended; but, again, the benefit resulting from them has rarely been very decided.

XV. XANTHOMA. (*Vitiligoidea. Xanthelasma.*)

Causation and description.—This affection was first clearly described by Drs. Addison and Gull under the second of the names given above. It has since been carefully investigated and described

by various dermatologists, and more especially by Dr. Hilton Fagge. It consists mainly in a kind of fatty or atheromatous change in the texture of certain portions of the skin, and in this respect has a very close affinity to atheroma of the arteries. The affected parts appear on section to consist of fibrous tissue (in a greater or less degree the normal fibrous tissue of the part) studded more or less abundantly with groups of oil-globules. It occurs in two forms, namely *x. planum* and *x. tuberosum*. In the former, the affected portions of skin present an opaque, yellow, or buff colour, are distinctly marginated, and although perhaps appearing to be elevated, are actually level with the general surface and undistinguishable from it in consistence and texture. In the latter variety, papules or tubera arise varying from the size of a pin's head to that of a hazel-nut, which sometimes by their aggregation form nodulated masses of considerable extent. These are generally yielding, elastic and but little indurated, are of the normal colour of the skin or of a reddish hue, and frequently studded, especially in their more prominent parts, with opaque yellow spots. Xanthoma is often unattended with local uneasiness; in the tubercular form, however, there is not unfrequently some degree of itching or tingling, and tenderness. Its course is for the most part progressive; but sometimes it becomes stationary and occasionally disappears. It never undergoes ulceration or other such destructive changes.

Xanthoma may occur on almost any part of the surface; on the eyelids, nose, ears, cheeks, head, neck, shoulders, nates, back of elbows, and front of knees, about the wrists and ankles, on the palms and soles, and on the knuckles of the fingers and toes. When occurring in the neighbourhood of joints it seems to be not unfrequently distinctly connected with the tendons. Xanthoma has also been observed in the mucous membrane of the nose, gums, lips, tongue, and larynx. The plane form of the disease is met with mainly in connection with the eyelids, ears, and other parts of the face, and with the mucous membranes. This, if the affection be at all largely distributed, occurs concurrently with the tuberose form; but it is not unfrequently alone present and limited to the face, and more particularly to the eyelids. In the latter case it usually commences in the skin of the upper lid near the internal canthus, and not unfreqently gradually extends thence until it involves the greater part of both lids. The tuberose form may be met with in the same situation, but is more commonly observed upon the extremities. When occurring in the palms or soles, the tubercles are usually of small size, but very numerous, and give a mottled aspect to the affected surface; and on the wrists and ankles the affection may assume a good deal of the typical appearance of keloid.

Although the causes of xanthoma, like those of so many other affections, are obscure, some curious facts have been observed which seem to have some relation with its ætiology. Many recorded cases, probably

half, have laboured during the development of the disease under jaundice, due to organic disease of the liver; and many also, as Mr. Hutchinson has pointed out, appear to have suffered largely from sick headache. In one or two cases the disease has been associated with diabetes.

Treatment.—No efficacious treatment is known.

XVI. LICHEN RUBER.

Description.—This is the name given by Hebra to an affection which is to some extent, no doubt, papular, but has no affinity whatever with the eruptions commonly included under the name of lichen. It begins with small colourless or reddish solid papules, for the most part unattended with itching. These increase in number, but very little in size, and presently coalesce at their margins so as to form smooth patches of uniform thickening and induration, the effect of which is to smooth away the finer furrows or creasings of the skin, to interfere with the free movement of parts, and to render the patient more or less hide-bound. The indurated skin is often thickened apparently to two or three times its normal thickness, and it loses its sensibility in a greater or less degree.

The disease begins symmetrically on different parts of the body, and may remain limited in its range, or may gradually spread over the whole surface. But there are certain situations in which its effects, however wide its distribution, are most obvious: these are the hands, feet, face and neck. The hands are affected mainly on their palmar aspects, but the convex surfaces of the metacarpo-phalangeal and phalangeal joints are also involved, and, in a less degree, the remainder of the backs of the fingers, which are apt to remain papular. The hands get stiff and almost useless, the fingers are kept widely separated and semiflexed, and cracks are apt to appear over the convexities of the joints. The feet and toes are similarly affected. The skin of the face becomes smooth and hard, the delicate wrinkles about the eyelids, forehead, and cheeks undergo more or less complete obliteration, and much of the patient's mobility of features and natural expression is lost. The primary papules and the infiltrated skin are said by Hebra to be red, and to have a tendency to yield thin scales. They are, however, sometimes pale, or of a pale dead-leaf colour, and free, or almost free, from desquamation. Hebra points out, also, that the nails get brittle, and either thin or thick; that the hair is unaffected; that the disease rarely undergoes amendment or cure; and that the patient tends to emaciate, and in the course of years to die from exhaustion.

He further states that the papillæ of the skin have been found after death to be hypertrophied, and the root-sheaths of the hairs thickened.

Treatment.—Arsenic in large doses, and cod-liver oil by inunction, are the only remedies which have been found beneficial.

XVII. SCLERODERMA. (*Scleriasis. Addison's keloid. Morphœa.*)

Causation and description.—Under the above series of designations have been described a number of morbid conditions of the skin, which are now generally admitted to be closely correlated, if not absolutely identical with one another. They are very rare, and consequently, although interesting, do not claim any lengthened consideration.

They are all characterised by the appearance of patches of induration and thickening; which vary in extent and shape; tend gradually to increase in size; are attended often with tingling, sometimes with anæsthesia; are white and ivory-like, or of a pale yellowish or brown hue, sometimes mottled, sometimes surrounded by a halo of congestion or discolouration; are for the most part of long duration, and in their progress apt to become faintly tubercular, or to desquamate, or even to ulcerate, and, when they finally disappear, to leave behind them more or less brownish discolouration, with atrophy and cicatricial seaming of the surface. The affected parts are for the most part smooth, scarcely, if at all, elevated above the general level, and incapable of being pinched up in a fold; and the thickening, although generally limited to the skin, sometimes involves also the subjacent connective tissue. The affection appears to consist anatomically in an overgrowth of dense connective tissue, associated with the accumulation of cells, resembling lymph-cells, in the sheaths of the small vessels.

A. It is comparatively not uncommon to meet with a patch or group of patches of scleroderma on one side of the forehead, in the area of distribution of the fifth pair. The affection then usually remains limited to this region. It commences insidiously, perhaps as a mere discolouration, gradually increases in size, and occasionally spreads to the hairy scalp, where it causes circumscribed alopecia. It is very chronic in its progress.

B. Another variety of the affection—Dr. Fagge's '*circumscribed scleriasis*' (scleroma)—is that which Dr. Addison described under the name of '*true keloid*,' deriving the word keloid from κηλὶς (a spot produced, as it were, by burning). In this, which is also a very chronic affection, the patches commence variously, sometimes as a mere loss or change of colour, sometimes as a mere depressed smoothness, sometimes as a simple induration, attended or unattended with itching or tingling.

The patches differ in shape: are round, oval, band-like, irregularly polygonal or stellate, and not unfrequently send out promontories, as it were, or peninsulas, into the surrounding healthy skin. They vary also in size: are sometimes no larger than a sixpenny-piece or shilling, but tend to increase, and thus sometimes involve ultimately very extensive areæ. They are usually multiple, and new spots are apt to arise from time to time. Beyond the itching and tingling, the main source of discomfort to the patient is the interference with the free use of parts which any considerable extension of the disease involves. He becomes hide-bound, and his fingers, hands, arms, or other parts which are affected, more or less distorted, fixed, and useless. This immobility is increased when (as often happens) the skin becomes adherent to the subjacent tissues, and when (as also occasionally takes place) subjacent muscles waste. The mucous membrane of the tongue, lips, and gums is sometimes involved in the disease.

C. A third form of the disease, which Dr. Faggo designates '*diffused scleriasis*' (scleroma), is that to which the names 'sclerema,' 'scleroma,' 'scleriasis,' and the like, are more particularly given. It appears to have been observed almost exclusively on the Continent, and is mainly characterised by the rapid extension of scleroderma over large parts of the surface of the body. It seems frequently to have begun at the back of the neck, and thence to have spread to the face, back and front of the trunk, arms, and even over the whole surface. The tongue may be involved. The integument becomes thick, hard, ivory-like, and smooth, the arms, hands, and fingers stiff and immovable, the face an expressionless mask. The aspect and feel of the affected regions have been likened to those of a frozen corpse.

None of the above varieties of scleroderma appears to be associated with any indications of constitutional suffering; and the secretion from the kidneys and even that from the affected portions of skin remain normal. They are all more or less chronic in their course—the first two lasting, as a rule, for years, and leaving on their subsidence marked signs of their pre-existence behind, the last, however, often disappearing entirely in the course of a few months. Women appear to suffer much more frequently than men. In some cases (especially of the diffused form), the attack is said to have originated in exposure to cold or wet; but little or nothing further is known with respect to the causation of the disease. There is some obvious resemblance between scleroderma and the later stages of lichen ruber, and still more between it and true leprosy, of which disease some authors regard its circumscribed forms as mere varieties.

Treatment.—No local measures seem to have been useful in the treatment of scleroderma. The constitutional remedies which have been employed include cod-liver oil, quinine, iron, arsenic, and iodide of potassium.

XVIII. ELEPHANTIASIS. (*Elephas. Pachydermia. Barbadoes Leg. E. Arabum.*)

Causation and description.—The condition to which the above names have generally been given is mainly a disease of tropical climates, and more especially of certain parts of India. Its chief characteristic is a more or less enormous hypertrophy of the connective tissue of certain parts of the body, associated with early implication of the lymphatic glands and vessels.

A. *Elephantiasis* commences with an erysipelatoid inflammation of the part about to become permanently affected, attended with febrile symptoms, and indicated by superficial redness, and general and deep infiltration. At the same time the superficial veins and lymphatics generally form red painful indurated cords, and the corresponding lymphatic glands undergo considerable acute tumefaction. If an incision be made at this time, a large quantity of yellowish transparent fluid, coagulating spontaneously, and having all the characters of lymph, escapes. After a few days, probably, the inflammation subsides, but more or less swelling remains. Subsequent attacks of inflammation, excited by various causes, supervene at irregular intervals—each attack adding to the mischief, and leaving behind it a tendency to still further hypertrophic change. The final result is that the affected part becomes largely, sometimes enormously, increased in bulk, and altered in aspect.

In some cases the hypertrophic condition occupies mainly the skin and subcutaneous connective tissue; in some it involves the whole extent of connective tissue between the skin and bone. In either case, but chiefly in the former, the skin is liable to be much modified in texture and form; sometimes it becomes coarsely papular or warty, sometimes studded with nodular elevations, sometimes undergoes ulceration; and the epidermis, though often remaining normal, may desquamate, or get thick or horny, or acquire the characters observed in ichthyosis, or become more or less deeply coloured from deposit of pigment granules in the rete mucosum. The affected surface, moreover, may be anæmic, congested, or livid. When the disease extends deeply, fat, muscles, and nerves get compressed and waste, but the bones undergo hypertrophy—new layers and irregular outgrowths forming, by means of which adjoining bones occasionally become organically united.

Elephantiasis appears to consist primarily in an inflammatory hyperplasia of the cellular elements of the connective tissue, in connection with which (according to Virchow) there is reason to believe that the roots of the lymphatic vessels are specially involved. Inflammatory overgrowth of the elements of the lymphatic glands next ensues, with obstruction to the passage of lymph through them. Then this fluid stagnates in the lymphatic vessels, which sometimes dilate even to their

radicles in the cutaneous papillæ; and it presently accumulates in the interstices of the affected tissues, adding to their bulk and at the same time stimulating them to overgrowth. It is only in the early stage of the disease that the dilated condition of the lymphatics admits of ready detection. At a late period the morbid tissues are characterised mainly by the presence of a dense accumulation of white fibrous tissue. The lymphatic glands also, after a time, become the seat of fibroid change.

The regions most frequently attacked with elephantiasis are the lower extremities and genital organs. But other parts may become affected, and especially the female breast. In the first of these cases the disease may commence in the toes or about the ankle, and gradually involve the whole leg up to the knee. It rarely, however, rises above that point. In extreme cases the form and appearance of the affected member remind one of those of an elephant's leg, whence the common name of the disease. When the scrotum or labia are involved they often reach enormous dimensions; the scrotum which is sometimes also the seat of hydrocele, may attain a weight of 50 or even 100 lbs.

Elephantiasis is a disease mainly of adult life, and is more common in men than women. Its progress is slow, but is largely governed by the conditions under which the patient lives, or the care he takes of himself. Fatigue, exposure to weather, or of the affected part to anything provocative of irritation or inflammation, is apt to aggravate it; while, under opposite conditions, the disease may make but little progress, or remain stationary. There is nothing in it necessarily inimical to life; but want of cleanliness or other accidental circumstances may give rise to ulceration or gangrene, and thus imperil life or cut it short.

Elephantiasis does not appear to be a specific disease. Swellings and indurations of precisely the same kind are apt to occur in the vicinity of old ulcers, and especially in parts which have undergone repeated attacks of erysipelatous inflammation. Only in these cases the hypertrophy rarely, if ever, goes on to that inordinate extent which characterises the endemic elephantiasis of tropical countries.

B. *Elephantiasis lymphangiectodes.*—A condition, closely related to elephantiasis, occasionally arises independently of inflammation, at all events of inflammation of the parts chiefly implicated. It is due sometimes to the continued application of a tight ligature round the upper part of one of the extremities, sometimes to obstructive disease in the lymphatic glands, or lymphatic vessels, either arising during adult life, or of congenital or infantile origin. In all of these cases the morbid condition appears to be chiefly, if not wholly, due to obstruction of lymphatics, with consequent dilatation of those below the seat of obstruction, accumulation of lymph in the textures, and overgrowth of the connective and other tissues. The lesions closely resemble those of elephantiasis Arabum; and the resemblance is not unfrequently enhanced by the occasional supervention of attacks of inflammation. The disease appears to be not uncommon in tropical climates; and is attributed by

Dr. Lewis to the presence of filariæ in the blood, and to obstruction of the lymphatics by these entozoa.

This form of elephantiasis generally first reveals itself by simple increase in bulk of the part affected. This increase goes on more or less insidiously, until it becomes considerable—the tissues getting more or less indurated and dense, and the surface pale, congested, or otherwise modified in colour, and either smooth, papular, or tuberculated. After a time, groups of vesicles generally make their appearance, sometimes widely distributed, sometimes in an irregular patch, sometimes in a linear series, and generally imbedded, as it were, in the solid tissue. These, which are really dilated lymphatic spaces, are apt to rupture from time to time, and then to exude considerable quantities—sometimes several pints—of lymph, which coagulates after its escape, and is either yellowish and transparent, or milky from the presence of molecular fat. This affection is usually limited to one of the lower extremities, or to the upper part of the thigh and contiguous part of the abdomen, or to the genital organs and perinæum; and it may be added that there is good reason to believe (as is elsewhere pointed out) that chyluria is due to a similar condition involving the mucous membrane of the bladder or other parts of the urinary tract. When the lower extremity becomes affected in infancy, not only does the limb increase generally in bulk, but the bones, relatively to those of the opposite member, become manifestly hypertrophied—augmented both in thickness and in length.

Treatment.—The treatment of elephantiasis should be mainly prophylactic; the patient who is suffering from it should be careful to avoid all causes of renewed inflammation; he should keep the affected parts clean and cool, should not expose himself to cold or vicissitudes of temperature, and should avoid all over-fatigue and exposure of the parts to irritation or injury. Moreover, these should not be allowed to be pendulous. During the inflammatory stage antiphlogistic remedies may be had recourse to; fomentations or cold lotions should be applied locally, with the object of preventing hypertrophy, and it may be of promoting absorption; and the affected region should (if its form or position permit) be kept evenly and firmly bandaged. Hebra recommends that the bandage be of cotton, and dipped in water at the time of application. He further recommends that, previous to the use of bandages, scales and crusts be removed by cataplasms, baths, or greasy applications, and that afterwards mercurial ointment be rubbed in.

XIX. MOLLUSCUM CONTAGIOSUM.

Causation and description.—This is an affection occurring mainly among children, and characterised by the development of small globular or sub-globular outgrowths from the skin, usually varying from the size of a pea downwards, but occasionally attaining larger dimensions. They are sessile, though sometimes attached by constricted bases. They differ little if at all in colour from the surrounding skin, but have a slight degree of translucency. They are unattended with pain or itching. Each tumour for the most part presents a distinct central depression, from which can often be expressed a little milky fluid or wax-like substance. On section it is found to consist of a lobulated gland-like body, the crypts of which are lined with columnar epithelium, and filled with rounded cells of large size. All these crypts communicate with a central duct, which for the most part is full of cells containing fatty matter. The growth appears in fact to be in some sense a kind of epithelioma. Molluscum has been supposed to be due to some abnormal development of the sebaceous glands; but both Beale and Virchow regard it rather as taking its origin in the hair-follicles. We believe, however, that we have seen molluscous tumours in the palm of the hand.

Whatever the nature of the disease may be, we consider that it has been clearly proved to be contagious. It frequently occurs simultaneously among the children of a family, and under such circumstances even the adult members occasionally become affected. The parts on which the tumours chiefly appear are the face, head and neck, and trunk; but they occur also on the limbs.

Treatment.—Local measures only are of use. If the tumours are attached by narrow bases they should be snipped off; if by broad bases, they should be effectually cauterised with nitrate of silver, potassa fusa, acid nitrate of mercury, or the like, previous to which it may be well to lay them open with a scalpel.

XX. PHTHIRIASIS. (*Lousiness.*)

Causation and description.—Lice, the presence of which gives rise to the affection sometimes termed phthiriasis, are of common distribution as parasites throughout the animal kingdom. Three varieties affect man, namely the *pediculus capitis*, the *pediculus vestimenti*, and the *phthiriis* (or pediculus) *pubis*. The first of these as a rule inhabits the head only; the second lives in the underclothing and feeds on those parts of the body which are uncovered with hair; the last infests

the hair of the pubes and armpits, and less frequently the eyebrows, eyelashes, whiskers, beard, and moustache.

A. The *pediculus capitis* or *head-louse* is generally of a grey colour like that of the scurf, and hence is very readily overlooked; it has, however, a dark streak (alimentary canal) along the central line of its body, the presence of which may aid in its detection. It lives among the hairs close to the scalp, feeding for the most part on the scurf and even on the hairs, and running along the latter with considerable agility. The female, which is larger than the male, deposits her eggs or *nits* upon the hairs, attaching them thereto by a tough transparent sheath. These, which may be readily mistaken for particles of scurf, are fixed upon the hairs much as are the cocoons of some moths upon the stalks of grass, are furnished with a lid, and measure about half a line in length. The female, according to Küchenmeister, begins to lay eggs at the end of eighteen days, and lays about fifty. They are hatched in six days.

Pediculi always cause more or less itching and consequently a tendency to scratch the head with the nails. This may be all. But in many cases the irritation which they produce leads to the development of eczema or impetigo, and the formation of thick scabs. As Mr. B. Squire has pointed out, impetigo in children limited to the back of the head is often of pedicular origin; and impetigo affecting the nape of the neck in adults (especially females) is also commonly attributable to lice. There is good reason to believe that the affection termed '*plica Polonica*' is nothing more than a combination of filth, lice, and entanglement or felting of the hair. We may add that pediculi (then termed '*p. tabescentium*') are very apt to accumulate in the heads of patients suffering from long and wasting illnesses. But there is no sufficient reason for regarding them as distinct from the common head-lice.

B. The *pediculus vestimenti* or *body-louse* is scarcely distinguishable from the last, excepting by its larger size, and its habits. It lives in the under-clothing, and attaches its eggs to the superficial projecting fibres. It is not always easy to detect its presence, for it is only occasionally discovered crawling upon the skin, or even upon the plane surface of the shirt or chemise. It almost always lies concealed in the folds or pleats; and it is in these situations also that its eggs are deposited. The eggs, moreover, though almost exactly resembling those of the head-louse, have generally so much the colour of the garment to which they adhere that they are seen with considerable difficulty.

Body-lice, like the last, often cause itching only; but often after a time, the constant irritation of their presence leads to the development of an indistinctly papular condition of the skin, and bleeding points and lines, the consequences of violent scratching. This state of skin closely corresponds with the ordinary descriptions of *prurigo*. And

indeed there can be no doubt that the great majority of cases of so-called '*prurigo senilis*' are essentially cases of phthiriasis. The presence of body-lice not unfrequently also causes urticaria, lichen, and eczema.

C. The *pediculus pubis* or *crab-louse* is very different in form from the other species of louse. It presents a much broader thorax and abdomen, and its chitinous claws are much more elongated and massive. It never affects any other parts than those which have already been named as its habitat; always nestling close to the skin, and biting deeply into it. It fixes its eggs, which resemble those of the head-louse, close to the points of emergence of the hairs.

The pediculus pubis causes violent irritation, and frequently induces an impetiginous eruption and the formation of abundant scabs.

Treatment.—It is usually not difficult to get rid of lice. The thorough use of soap and water, and thorough personal cleanliness, are of course essential, but alone are not generally sufficient. Many local applications will destroy them, but none probably is more efficacious than daily washing with decoction of staphisagria seeds, or the inunction of the parts (as recommended by Mr. B. Squire) with oil of stavesacre diluted with olive oil, or the application of mercurial ointments, such as the ammonio-chloride. The remedy must of course be continued until all nits (as well as lice) are removed or dead. It is often desirable, in order to promote certainty and rapidity of cure, to hunt out and destroy the pediculi one by one, to pluck or cut out the nit-bearing hairs, or even to shave the head or other hairy parts. The applications which have been enumerated are useful even in the treatment of the pediculus vestimenti, but the chief treatment here must be directed to the clothes. Not only, however, must these be frequently changed and washed, but the bed-clothes must be similarly treated, as must also the clothes of anyone sharing the same bed.

XXI. SCABIES. (*Itch.*)

Causation and description.—Itch is a skin disease dependent on the presence of the acarus scabiei, and marked by the development of a papular, vesicular, or pustular eruption, with intolerable itching, which is especially violent in the evening and at night.

The *acarus scabiei* is an animal not unlike a cheese mite, both in general form and in colour, and is visible to the naked eye as a minute white ovoid speck. Its body has a short oval form, is convex above, somewhat flattened below, studded with numerous spines and bristles, and furnished (in the adult state) with eight legs. In the female the four front legs end in stalked suckers, the four hind legs in bristles.

In the male the hindermost pair of legs, as well as the four front legs, present suckers. The acarus just escaped from the egg has six legs, the hindermost, or fourth pair, only making their appearance after the first change of skin. The male is little more than half the length and breadth of the female. The egg, which is oval, measures about one-third the length of the adult female.

The acari live for the most part in burrows (cuniculi) which they make for themselves in the substance of the epidermis, beneath its horny layer. According to Hebra, about a fortnight elapses from the time of hatching until the complete development of the animal. At the end of that time the impregnated female penetrates the corneous layer of the skin, and then slowly tunnels beneath it in a straight, zig-zag, or curved line. In its onward progress it deposits eggs, sometimes as many as fifty, in a linear series; and at the end of two or three weeks, or it may be six (Hebra), it dies at the further end of its burrow. This may then have attained the length of half an inch, or an inch, or even more than that. It is generally quite obvious, on careful examination, as an irregular line, studded with sub-cuticular black matter (fæces); presenting at its commencement, in consequence of the gradual desquamation of the skin, a groove with retreating sides—a kind of calamus scriptorius—and at its opposite extremity a minute papule, in which the white body of the animal can generally be pretty readily distinguished. The formation of the burrow and its full development may be unattended with visible signs of inflammation; but not unfrequently papules, vesicles, or pustules rise up in its immediate neighbourhood, the burrow then passing over them, or alongside of them, but very rarely forming any communication with them. Sometimes strings of vesicles, running perhaps together, mark its whole length. The eggs contained within the burrow hatch there and the young speedily migrate. The male acarus is difficult of detection, partly from its minuteness and comparative infrequency, and partly from the fact that it either simply imbeds itself in the skin without burrowing, or rambles over the general surface.

The acari mostly burrow about the wrists and hands, especially on the palmar aspect, and between the fingers, and in the corresponding situations in the lower extremities; they also infest the nipples and organs of generation, the flexures of the elbows and knees, the axillæ and the buttocks. No part can be regarded as necessarily exempt from their ravages. The face and head, however, are rarely attacked. The presence of the acari causes intolerable itching, which increases at night time, and provokes violent scratching. It also gives rise to inflammatory eruptions—papules, vesicles, blebs, or pustules—which are to be looked for especially on those parts of the surface which the acari chiefly affect; and occasionally it induces urticaria, eczema, or impetigo, which are not necessarily limited to the neighbourhood of the burrows, and may become general. The papular and vesicular

forms of eruption are the most common. The pustular variety shows itself for the most part in persons who are out of health or possess peculiarly susceptible skins. Sometimes the inflammation becomes excessive, and produces not only pustules but considerable inflammatory exudation and infiltration. This condition may often be observed in the penis and the nipples. The disease has naturally little or no tendency to spontaneous cure; but can certainly be kept in abeyance by personal cleanliness. Under opposite conditions, however, it is apt to become greatly aggravated. Occasionally the tips of the fingers and toes, with the nails, get destroyed, partly by the direct operation of the acari, partly by the ulceration which they induce. A very severe form of the disease, common in Norway (and hence termed '*scabies Norvegica*'), but not confined to that country, is characterised by the formation of thick tough crusts extending over the palmar surface of the hands and fingers, and the corresponding surface of the feet and toes, the parts beneath being excoriated or ulcerated. The crusts contain innumerable acari and ova, both living and dead.

From the different degrees of severity which it presents, and from the very various eruptions to which it gives rise or with which it may be associated, itch is a disease which, on the one hand, is very apt to be overlooked when present, and, on the other hand, is very liable to be assumed as present when the patient is entirely free from it. The appearance of a papular, vesicular, or pustular itching eruption between the fingers and about the wrists, and in other situations which itch affects, is no doubt an important indication; but similar eruptions, not due to the acarus, occur in these same localities. The transference of the disease to a bed-fellow or to those with whom the patient has similarly close relations, is also a point of great significance; but it must not be forgotten that one member of a household may have itch for months and yet fail to infect any of the other members. The only real proof of its presence is the discovery of the acari, their eggs, or their burrows. The burrows are sometimes marvellously well seen, presenting all the characters which have been already described; but they are often incipient, and very difficult of recognition. When they are distinct the discovery of the female acarus is easy. It can generally be seen, even with the naked eye, at the further extremity of the burrow, or apparently a little beyond that point, as a very minute whitish papule. If the surface of this papule be torn with a pin, the acarus may readily be removed from its bed on the point of the instrument. In performing this operation it is well to avoid wounding any neighbouring vesicle or pustule. Even in cases where no obvious burrow exists, the acari may be occasionally detected in the neighbourhood of some of the itching papules by the presence there of the minute whitish elevations which they cause. Sometimes, even when distinct burrows are present, there is some difficulty in detecting the acari at their extremities; in such cases one of the burrows may be broken down, and

its contents removed on the point of a pin or lancet, or, still better, a whole burrow may be cut out. By these means the ova may be readily obtained. A further plan is to remove the scabs, if there be any, to boil them in a solution of caustic soda until they become limpid, and after allowing the fluid to stand for a time in a conical glass, to examine the deposit with a microscope. Dead acari, including males, and six-legged grubs, and ova, can often be obtained by this process.

Treatment.—The essential object in the treatment of scabies is the destruction of the acari and their ova. For this purpose it is necessary not only to apply an appropriate parasiticide, but to soften the skin and remove its superficial epidermis so as to expose the burrowing mites to its influence. The patient therefore should have daily hot baths, use soap abundantly, and rub the surface thoroughly with a flesh-brush or a rough towel. All scabs should be removed. Then sulphur ointment, either of those of the Pharmacopœia, or that of Helmerich, which contains carbonate of potash, should be rubbed well into the skin, especially into those parts which seem most affected, and should remain upon the skin until the next bath. Treatment of this kind will generally cure itch in the course of a few days or a week, but may not improbably induce eczema or some other form of superficial inflammation, which will need other remedies for its cure. No doubt less active measures will suffice to cure scabies, but the cure will probably then be long delayed. On the whole, however, when a person has itch it is better for himself, in the long run, and better for those with whom he associates, that he should recognise his condition, retire for a few days from public life, and adopt the measures which will most speedily work a cure. Tincture of benzoin and balsam of Peru, far more agreeable applications than sulphur, are said to be more efficacious than it in the cure of itch. They should be rubbed well into the affected parts. The purification of the patient's clothes and bedclothes forms an essential part of the treatment.

XXII. OTHER SKIN-AFFECTIONS CAUSED BY ANIMALCULES.

Causation and description.—Fleas, bugs, and *gnats* do not of course come under the category of parasitic animals. So many persons, however, suffer from their bites, and the effects of their bites are so often misinterpreted, that it seems desirable to make a remark or two in reference to them. A recent flea-bite always exhibits a punctiform subcutaneous extravasation of blood, surrounded by a comparatively broad rosy areola. The latter soon disappears; the former may persist

for several days. People, and especially children, of the lower classes are often thickly covered with such petechial spots in different stages of their progress; and their skin, when seen for the first time (especially if they be suffering from some febrile disturbance), is very apt to suggest the presence of the typhus eruption. The smallness, however, of the spots, their uniformly petechial character, and the probable detection in them on close inspection of the puncture made by the insect, will alone, for the most part, enable a careful observer to distinguish the eruption due to fleas from that of any of the specific fevers. In some persons flea-bites produce considerable irritation and the development of wheals or tubercles, associated sometimes with a large amount of subcutaneous effusion of serum. The effects are then not unlike those which commonly arise from the bites of gnats and bugs. In all these cases there is generally in the first instance violent itching, which is followed presently by the formation of a wheal or tubercle from the size of a split pea downwards, and very often by more or less considerable subcutaneous œdema. The latter pretty soon subsides; but the wheal probably continues for a week or fortnight, and is generally attended with itching during the whole of that time. In its progress (owing in some measure to scratching) punctiform extravasations of blood often take place into its central part, and these are sometimes succeeded by vesication, or the formation of a pustule. Sometimes the wheals gradually subside and disappear; but in many cases their subsidence is attended with the separation of a squama, or the formation and detachment of an eschar, or, when there has been vesication or suppuration, the production of a scab. It need scarcely perhaps be said that gnats generally select exposed parts of the skin, fleas those regions which are protected by clothing, and that bugs are more indiscriminate in their attacks. There is no doubt that the bites of these insects, especially in children of delicate skin, produce eruptions the source and nature of which are apt to be entirely overlooked. Many attacks of so-called 'strophulus,' 'lichen,' and 'impetigo,' ascribed to dentition, dyspepsia, and other causes, are really due to the operations of the above animals. *Mosquito* bites are almost identical with gnat bites in their effects; they are generally, however, much more numerous and individually more venomous.

The *leptus autumnalis*, or *harvest-bug*, which is of common occurrence in the autumn in grass and cornfields and among gooseberry bushes, is very apt to imbed itself in the skin, and to cause much irritation there. The effects pass off in about a week. The mite, which is just visible to the naked eye, is of a red colour and presents six legs. It is probably the immature condition of an unrecognised eight-legged animal.

The *pulex penetrans* (*chigoe*) is a native of South America and the West Indies. It is so small as to be seen with difficulty, and is characterised by the possession of a proboscis as long as its body. Only the

impregnated female attacks man. It penetrates the skin of the feet and toes, generally in the neighbourhood of the nails, where its impregnated body quickly develops itself into a white vesicle the size of a pea. This enlargement is due to the rapid growth of the larvæ, which, if the cyst be ruptured, escape into the surrounding tissues and cause in them severe inflammation with suppuration. The recognised mode of treatment is to dilate with a needle the orifice by which the intruder entered, until it is large enough to allow of its extraction without rupture.

On the Island of Bulama and its neighbourhood, on the West Coast of Africa, a pimple ultimately attaining the dimensions of a boil, and then attended with much pain and surrounding inflammation, and even affection of the neighbouring lymphatic glands, is attributable to the grub of some insect, which is deposited doubtless in the egg beneath the skin, and attains its full growth in that situation. The perfect insect is unknown.

The *acarus* (*demodex*) *folliculorum* resides in the sebaceous follicles, for the most part in the duct or about the spot at which the sebaceous follicle opens into that of the hair. It is sluggish in its habits, and lies imbedded in the sebum with its head pointed towards. The number of acari in a follicle vary from one upwards. As many as thirteen have been discovered at one time (Küchenmeister). They differ in size, and in some degree in form, with age. In the earlier periods of their development they present six, and subsequently eight legs. They are most commonly found in the comedines of persons suffering from acne punctata, but do not cause this affection, or apparently aggravate it. In order to find them the expressed sebum should be diluted with oil, and then submitted to microscopic examination. This parasite causes no distinctive symptoms in man; but is said to produce serious and sometimes fatal consequences in the dog.

XXIII. TINEA TONSURANS. (*Porrigo scutulata. Ringworm.*)

Causation and description.—Ringworm depends upon the presence of a fungus, termed '*tricophyton tonsurans*,' which chiefly affects the roots and shafts of the hairs, but also invades the epidermis and the nails. Its mycelium consists of filamentous jointed branching tubes, which in the hair run in groups parallel with its long diameter, but in the epidermis and nails form an irregular interlacement. The spores are minute oval or rounded bodies, formed, in the first instance, in linear series at the extremities of the mycelial filaments; but soon so abundantly developed that this relation is entirely lost. Spores form both in the epidermis and in the nails, but their chief seat is the shafts

of the hairs within and a little external to the skin. The fungus spreads superficially, as do most fungi, in gradually enlarging circles, which, however, from various accidental circumstances, often expand irregularly, and often, when they are large, break up into irregular segments, and often, moreover, present fits of alternate quiescence and growth.

When ringworm occurs on the non-hairy skin, it reveals itself first as a slightly raised roundish uniformly erythematous patch, a line or two in diameter. This slowly increases in size, becoming at the same time more distinctly circular; and when it attains perhaps half an inch in diameter the inflammation at the centre begins to subside, and the patch thus becomes a ring. In its further progress the ring may enlarge to the size of half-a-crown, or a crown, and still extending (but then for the most part irregularly), may creep, for example, over the whole side of the face or front of the chest. The margin of the patch is always red and elevated, but varies in breadth, and often presents papules or vesicles; and hence the affection has been called indifferently *erythema circinatum*, *lichen circinatus*, and *herpes circinatus*. The central area, even if all inflammation appears to have subsided in it, still retains a yellowish or brownish discolouration, and a tendency to scale. Moreover, fresh spots of inflammation are apt to appear here and there upon it. Occasionally, patches of ringworm present two or three concentric erythematous rings, separated by rings of fairly healthy integument. There is no doubt that this variety has often been termed *erythema*, *lichen*, or *herpes iris*.

When the nails are attacked, which is rare, they become in the affected parts irregular, thick, softer than natural, and at the same time more or less opaque and of a yellowish tint. The fungus penetrates them generally from the root, and not unfrequently the adjoining surfaces of the fingers, and the hands, are at the same time involved.

The most important, if not the most common seat of ringworm, is the head. Here the circular form of the affection and its erythematous or vesicular margin are seldom distinguishable. The patches, however, are generally well-circumscribed, and are indicated—partly by an abundant formation of adherent glistening scurf, which clings around the bases of the hairs, and is continuous with the lining of the hair-sheaths, and, by its peculiar scaly character, has given to ringworm one of the names, *porrigo scutulata*, by which it was formerly known; and partly by the condition of the hairs, which become swollen, dull and opaque, limp and lacerable, so that they break off either at the surface of the scalp, or a line or two above it. This breaking off of the hairs produces a marked resemblance to a stubble-field, and has suggested the common name of the disease, *t. tonsurans* or *tondens*. This stubbly character may be concealed, and the surface rendered apparently bald, by accumulation of scurf. On removal of this many of the broken hairs are removed with it.

Ringworm sometimes in men attacks the beard, moustache, and whiskers, producing one of the varieties of *sycosis*. It there excites (as it does occasionally in the scalp) considerable inflammation, causing deep-seated suppuration about the sebaceous glands and roots of the hairs, and is very intractable.

Ringworm is generally attended with more or less itching, especially if the head be the part affected. It is highly contagious, and is particularly liable to spread amongst children. Adults, however, especially those who are in attendance on affected children, often take it. But in them it is limited for the most part to the nails and fingers, and other non-hairy parts of the skin. Many suppose that it attacks mainly those who are in enfeebled health. But this is doubtful. When confined to the general surface it can, for the most part, be easily cured. In the head, however, or the beard, or nails, its eradication is extremely difficult and apt to be long delayed. Children may suffer from it for several years; and we have known it to persist in the finger nails of an elderly lady for at least seven years, never during that time extending to other parts of her body. Tinea tonsurans affects the horse and some other of the lower animals.

Treatment.—The treatment of tinea tonsurans is purely local, the main object being to destroy or remove the fungus which produces it. Many substances are recommended as parasiticides, the most important being the sulphurous acid of the Pharmacopœia, and empyreumatic substances, such as *unguentum picis liquidum* (diluted or not), *unguentum creasoti*, oil of cade, and the like. In the treatment of ringworm of the head or beard, it is of great importance that the surface be kept close clipped or shaven, and by washing with carbolic soap and water free from scales or other kinds of exudation. Further, it is desirable that all affected hairs be removed from the morbid patches by daily diligent epilation. After each daily washing and epilation the specific medicament should be applied and kept applied: sulphurous acid by means of several folds of lint saturated with the solution and covered with oiled silk or paper; ointment by being rubbed in and then left in a thick coat on the surface. In the case of ringworm of the body, the same measures as to cleanliness and specific applications may be pursued; but here it is often advantageous to destroy the affected surface of the skin with some caustic, such as nitrate of silver, strong acetic acid, iodine paint, or blistering fluid. When the nails are involved, the surface should be removed in slices and sulphurous acid or creasote ointment freely and constantly applied.

Ringworm of the head and beard is very apt to reappear weeks or even months after apparent cure. The reason of this is of course obvious. It is important therefore that the treatment should be prolonged far beyond the period of apparent cure, and that the hairs of affected areas should be from time to time carefully examined. Dr. Duckworth has recently pointed out that if a few drops of chloroform be

dropped on suspected portions of the head, diseased hairs acquire an opaque yellowish-white colour, the healthy hairs remaining unaffected.

XXIV. TINEA FAVOSA. (*Favus. Porrigo Favosa and Lupinosa.*)

Causation and description.—The cause of favus is the growth in the skin of the fungus known as the '*achorion Schönleinii.*' This consists in a jointed mycelium, differing little from that of the tricophyton tonsurans, and like it invading the epidermis, nails, and hairs. It differs essentially, however, from the tricophyton in the seat and character of its fructification. The formation of sporules begins with the development of short rounded joints or sporules at the extremities of certain of the mycelial tubes, and a complex development of other sporules from them by budding. The first evidence of fructification to the naked eye consists in the appearance of minute disc-shaped sulphur-yellow spots beneath the horny layer of the epidermis, or of minute yellow cups at the points of emergence of hairs. These gradually increase in size, until they form yellow cupped discs from $\frac{1}{4}$ to $\frac{1}{3}$ inch in diameter, through the centres of which hairs not unfrequently pass. On breaking these masses up they are found to be white within and brittle, and microscopically to consist of sporules seated in a finely granular matrix.

The early stage of favus, which is commonly overlooked, and is most obvious when the disease attacks the smoother parts of the body, consists, like that of ringworm, in the appearance of small circles of erythema, which soon enlarge and become rings, and may then be studded with papules or vesicles. These rings of herpes or lichen circinatus rarely grow larger than a sixpence or a shilling and are at first absolutely undistinguishable from those of ringworm; but soon there appear here and there at the edges or over the surface of the discs the characteristic yellow points of fructification; and these rapidly attain their full dimensions. The mature favi, if discrete, maintain their characteristic form and appearance; but where many of them are developed in close contiguity with one another they are apt to blend, and before long to form a prominent, irregular, mortary mass crossed superficially by an imperfect network of undermined epidermis, and presenting collectively an appearance not altogether unlike that of a rupial scab. Not unfrequently the progress of favus is attended with considerable inflammation, and even suppuration, the products of which blend with those of the vegetable growth. Under these circumstances the neighbouring lymphatic glands also become inflamed. As a rule, however, favus is attended with little local

irritation, and little itching. It is characterised generally by a peculiar mousy odour.

Favus most frequently attacks the head, and leads to the falling out of the hair, and the growth in its place of thin, colourless, woolly hairs, and often causes subsequently total destruction of the hair-follicles, and permanent baldness. The affected hairs, however, are not rendered brittle, as in tinea tonsurans, and therefore do not break off. Nails attacked with favus do not differ appreciably from those which are the seat of ringworm.

Favus is of rare occurrence in England, but in Scotland appears to be somewhat common. It is limited almost entirely to persons of filthy habits, and generally begins in childhood. When treated in its early stage it is easily cured; but when it has infected a large area it is exceedingly intractable, and will often (notwithstanding careful treatment) persist for many years. That this, like other parasitic diseases, is infectious, is beyond doubt; nevertheless, it is remarkable how rarely (compared with tinea tonsurans) it spreads among children, or from one member of a family to another. Favus is a common and fatal disease in mice. Cats also sometimes suffer from it.

Treatment.—The principles and details of the treatment of favus are as nearly as possible identical with those of the treatment of ringworm. In the first place all the favi should be removed by washing, poulticing, or the employment of oleaginous applications. Then the surface should be kept scrupulously clean, and treated by such parasiticide remedies as are useful in ringworm. Persistent epilation is of essential importance. In severe cases it is necessary to continue the treatment for many months, a year, or longer. Yet even when thus apparently cured, it not unfrequently breaks out again as soon as treatment is discontinued.

XXV. TINEA VERSICOLOR. (*Pityriasis Versicolor. Chloasma.*)

Causation and description.—This disease is caused by the growth among the epidermic cells of a fungus, termed the '*microsporon furfur*. The mycelial tubes are about equal in thickness to those of the fungi which have been above described, but their texture is more delicate. They form an interlacement in the substance of the epidermis, but do not invade the hairs or nails. The spores are developed in microscopic clusters, somewhat resembling bunches of grapes, scattered here and there among the mycelial tubes, and seem to originate within buds springing from the sides or ends of certain of the cells of the mycelium.

Chloasma is characterised by the formation of light-brown or liver-coloured spots, which are slightly elevated above the general surface

of the skin, covered with a more or less abundant branny scurf, and attended with slight itching. The primary spots have a circular outline, and vary perhaps from the third or fourth of an inch in diameter downwards. In the first instance a few such spots appear here and there. These increase in size, and soon other similar spots arise in their vicinity. By degrees neighbouring spots blend, and thus more or less extensive tracts of skin become pretty uniformly covered, the edges still presenting a sinuous character, and the neighbourhood numerous outlying solitary and coalescing islets.

Chloasma seems never to attack children, and very seldom persons of cleanly habits and among the better classes of society. It is a disease of adult life, and not unfrequently appears in those who are consumptive or otherwise out of health. It usually commences on the chest or between the shoulders; and thence may spread, over the abdomen and back, to the shoulders, upper arms and even forearms, and to the buttocks and thighs. But it never affects uncovered parts. This circumstance, together with the fact of its occurring mainly in those who wash little and seldom change their linen, seems to indicate that the disease originates in filth. Like other parasitic diseases it is contagious, but its contagiousness is not well marked.

Treatment.—In the treatment of chloasma perfect cleanliness is necessary. The affected parts should be daily washed with soap and water and well scrubbed with a flesh-brush or a rough towel; after which, one of the parasiticide applications should be well rubbed in. Under these measures the disease soon becomes apparently cured. Its complete cure, however, demands persistence in treatment long after all visible traces of the disease have disappeared.

XXVI. ALOPECIA AREATA.

(*A. circumscripta. Porrigo or Tinea Decalvans.*)

Causation and description.—This is an affection mainly of the hairy scalp, but occasionally also involves the eyebrows and eyelashes, the beard and whiskers, the hair of the armpits and pubes, and, it may be, even the general surface of the skin, and is characterised by the temporary or permanent loss of hair in more or less distinctly circumscribed areæ.

A well-developed patch of alopecia areata of the scalp is usually quite unmistakable. It is a well-defined bald surface of circular or sinuous outline, for the most part clean, smooth, and shining, and free from congestion or scurfiness. The skin indeed appears to be, if anything, thinner than in health, and the orifices whence the hairs should

emerge are atrophied and indistinct. The patch may be perfectly bald in its whole extent, or may present here and there groups of such downy hairs as constitute the lanugo; but not unfrequently a few long hairs still stud its surface at distant intervals; and often in the neighbourhood of these and of the margin may be seen on close inspection short club-shaped hairs, varying from about a line to ¼ or ½ inch in length. These are most obvious in dark-haired persons, from the fact that each clubbed free extremity still presents the natural dark colour; but the portion of shaft between it and the scalp becomes more and more attenuated and more and more devoid of colour as it approaches the latter. They can be pulled out more readily than healthy hairs, but still are generally attached with some degree of firmness. Their presence may be taken as indicative of the extension of the disease. When the alopecia has become arrested, downy hairs begin to show themselves over the bald area; and these may gradually assume all the characters of the surrounding healthy hairs, or become coarse, and white or otherwise modified in colour, or remain weak and scanty. Sometimes new hairs grow up in the centre, while the disease is still spreading circumferentially.

Alopecia areata is for the most part of chronic progress, lasting generally for months, often for years, or even for life. In some cases the patient presents only one or two circular spots, which enlarge up to a certain point and then undergo resolution. In some cases the disease continues to extend indefinitely, partly by the enlargement of old patches, partly by the development of new ones, until the greater part of the scalp or even the whole scalp is involved, and until it may be the eyelashes and eyebrows, one after the other, and finally all other collections of hair disappear. Occasionally the [illegible] disease is acute, the hair falling out rapidly and [illegible] still perhaps more or less patchily. The final issue [illegible] uncertain. In the great majority of cases recovery [illegible] a longer or shorter time; but it is important [illegible] often a tendency for the disease to recur at irregular [illegible], and not necessarily in the part originally affected. In a considerable number of cases, and especially in those in which extensive tracts of surface have suffered, complete restoration of the hair never occurs; and in a few, absolute and permanent general alopecia ensues.

The clubbed hairs above referred to present certain peculiarities of microscopic structure. The clubbed end is usually broken into a brush, and frequently presents in its interior an irregular group of largish cells, which are evidently the cells of the axis of the hair, at that part, modified in character. From this point downwards the hair becomes more and more attenuated, until it ends in a very slightly dilated point, which represents the imperfect root. Occasionally, a little below the clubbed extremity, the dwindling shaft is interrupted by a small knot, within which such a group of cells exists as is

usually found in the clubbed end itself. Looking to the fact of the occurrence in the originally healthy hair, at a point which seems to separate the normal from the attenuated portion, of a spot in which there has been some sudden modification of nutrition and growth which renders the hair at this part brittle and peculiar in structure; and to the fact that the portion of the shaft subsequently formed becomes, in consequence of the gradual wasting of the hair-root, more and more attenuated, until it falls out bodily; it would seem pretty certain that the diseased process, as it affects the hairs, depends on the gradual spreading from some central point or points of a wave of inflammatory or other influence which, as it passes over each hair-papilla, momentarily excites it as it were to unhealthy over-production and then leaves it enfeebled and perishing. This disease is asserted by Bazin and many others to be parasitic, and due to the presence of the *microsporon Audouini*.[1] There can be little doubt, however, that this view is erroneous. It is believed also by many to be contagious; but this, again, is doubtless an error. It is certain, however, that it is apt, like psoriasis, to break out periodically in the same individual, and like that also to affect several members of the same family, and to be transmissible from parent to child. The disease is more common in children than adults, and in females than males. We have seen it in a child ten months old, and it is often met with, still progressing, in persons between forty and fifty. Its presence is neither preceded nor accompanied by any general signs of ill-health; nor is its progress usually attended with any subjective local symptoms. Occasionally its commencement and spread are marked by more or less intense tingling or itching, so that the experienced patient not only knows, before the hair falls out, when a new patch of disease is commencing, but knows also when an old patch is spreading.

Treatment.—The treatment of alopecia areata is very unsatisfactory. Many patients get well who are never subjected to any, and many go on progressively from bad to worse in spite of the most sedulous care. There are no obvious indications for constitutional treatment, but tonics and arsenic are often employed empirically. For local medication it is generally thought best to use stimulants, and especially to blister the affected regions periodically with the *acetum cantharidis* or iodine paint. We do not believe that shaving the head is of any use, excepting for the purpose of facilitating the application of local remedies. It may be added that those who believe in the parasitic nature of the disease would naturally recommend the use of creasote, sulphurous acid, or other parasiticides.

[1] Recently M. Melassez claims to have rediscovered the specific fungus of this disease. He has seen spores of indeterminate character, and in very small numbers, in the horny layer of the epidermis—none in the rete mucosum, none in the hairs. They are doubtless accidental; at all events there is absolutely no ground for regarding them as the cause of the disease.—*Archiv. de Physiologie*, 1874.

XXVII. PRURIGO.

Description.—This name is given to a condition of the skin, attended with more or less violent itching, and usually marked by coarseness of texture, and the presence of scratches produced by the action of the finger nails. It is uncertain whether there is any specific affection to which the name is applicable. Willan obviously included under this term mere pruritus, or itching from various causes, and especially that due to the presence of body-lice. But he also included a papular affection which he regarded as quite distinct from other varieties of papular diseases. Hebra also describes a similar affection, which he considers to be *sui generis*, and to which he limits the use of the name.

According to the latter authority, prurigo is a disease of remarkable intractableness, if not incurable, consisting in the development of flat papules, not differing in colour from the skin, scarcely appreciable by the eye, but readily detectible by the touch, and leading to a general coarseness of texture and more or less pigmental deposit. It may occur upon nearly all parts of the body, though rarely attacking all in the same individual; and it especially affects in an increasing ratio the front and back of the trunk, and the extensor aspects of the upper arms and thighs, fore-arms and legs. The papules are apt to be irritated into inflammation or torn by scratching, and the eruption to be complicated, after a time, with eczema, impetigo, urticaria, and the like. Notwithstanding Hebra's authority, it may still, we think, be a question whether prurigo does not represent a heterogeneous group of ill-developed or ill-defined affections, attended with the common symptom of intense itching, and in which a coarse sub-papular condition of skin is present, in consequence partly of some abnormal nutritive condition of the skin, partly of the influence of constant scratching and other varieties of irritation. According to this view, prurigo may be a legacy left by eczema, impetigo, or erythema; or it may be present in persons liable to these affections during the periods when they seem to be free from them; or it may be referrible to phthiriasis or scabies, to jaundice or uræmia, to want of cleanliness, to the irritation produced in delicate skins by the too abundant and too frequent use of soap, or to excessive friction either by the towel or by the clothes.

Treatment.—For the treatment of prurigo, Hebra especially recommends sulphur, in the form of ointment, baths, or fumigation, tar in its various preparations, creasote, and frequent bathing. Besides these remedies lotions may be employed containing opium, prussic acid, acetate of lead, acetate of ammonia, or vinegar, or else black-wash, or mercurial or plumbic ointments. The constitutional treatment must depend on the patient's general symptoms or state of health,

or on the nature of the ailment to which the pruritus is referrible. When the itching is due to parasitic affections, parasiticide applications must be employed.

XXVIII. CONCLUDING REMARKS.

Besides the various affections of the skin which have just been passed in review, there are many others, of more or less interest, which could not be omitted from a work devoted to skin diseases, yet scarcely call for consideration in a manual of medicine. They are either of no practical importance, or they are extremely rare, or they fall entirely within the domain of the surgeon, or they are mere symptoms of more important disorders, and consequently are considered, so far as is necessary, elsewhere in this volume. We allude more particularly to such hypertrophic affections as horns, corns, warts, nævi, fibromatous and fatty tumours, epithelioma, and other varieties of malignant disease which affect the skin primarily or secondarily, and form either circumscribed tumours or infiltrating growths; to various atrophic conditions of the skin, hair, and nails, inclusive of the condition to which Dr. Wilks has given the name of *linear atrophy*; to increase or diminution of pigment (*ephelis, lentigo, vitiligo, albinism*); to the eruptions characteristic of many specific febrile disorders; and to such rare or ill understood affections as *frambœsia, pellagra* and *acrodynia*.

CHAP. III.—DISEASES OF THE RESPIRATORY ORGANS.

I. INTRODUCTORY REMARKS.

A. *Anatomical Relations.*

1. The *organs of respiration* comprise the larynx, trachea, bronchial tubes, lungs, and pleuræ.

Larynx and trachea.—The larynx is situated in the upper and fore part of the neck, extending from the hyoid bone above to the lower border of the cricoid cartilage below. The trachea commences at the lower border of the larynx, on a level with the upper orifice of the œsophagus and the fifth cervical vertebra, and runs downwards in the mesial line to the level of the third dorsal vertebra, where it divides into the two bronchi. The upper half of it is situated in the neck, the lower half in the chest, behind the sternum. Behind, it lies in contact in its whole length with the œsophagus. In front, it is embraced above, as low down as the fourth, fifth, or sixth ring, by the thyroid body, and below, just above its bifurcation, is crossed by the transverse arch of the aorta. The roots of the lungs are situated in the posterior mediastinum, on the level of the bodies of the fourth and fifth dorsal vertebræ; the right bronchus, which is nearly horizontal, being on the level of the fourth vertebra behind and second costal cartilage in front; the left, which passes down obliquely, reaching as low down as the fifth vertebra behind, and a little below the second costal cartilage in front. The latter passes under the aortic arch and is therefore in contact, above with the transverse arch, behind with its descending portion.

Lungs.—The apex of each lung rises above the first rib into the root of the neck; and the posterior obtuse margin occupies the groove between the ribs and vertebræ as low down as the eleventh rib. The base of the lung varies in position with the varying position of the diaphragm. The vault of the diaphragm rises during expiration on the right side to the level of the fifth rib at the sternum, on the left to the level of the sixth, and of course therefore the liver on the right side and the stomach on the left attain these respective elevations. The outer margin of the base, however, owing to the upward convexity of the diaphragm, reaches to a lower level, and during medium distension of the lungs with air may be traced in nearly a

direct line from the junction of the sixth costal cartilage with the sternum outwards and downwards to the head of the eleventh rib. During deep inspiration the edge may descend considerably between these extreme points. The anterior margin, like the lower one, varies in its position during the respiratory acts. When the lungs are moderately full their anterior borders are separated above by a triangular interval, the base of which corresponds to the sternal notch, the apex to the lower edge of the manubrium. From this point downwards to the interval between the fourth ribs, they continue parallel and nearly in contact. They then separate again, the edge of the right lung still passing vertically downwards, while that of the left retreats, forming a notch of which the apex corresponds to the junction of the fifth costal cartilage and rib, or to a corresponding point in the fifth interspace, and within which the heart becomes superficial. After a deep inspiration the anterior edges of the lungs are usually in contact from above down to the commencement of the cardiac notch; after a deep expiration there may be an interval of an inch or two between them. The extreme apex of the lower lobe of either side is situated behind, and in the adult about three inches below the summit of the lung.

Pleuræ.—The cavities contained by the parietal pleuræ correspond pretty accurately to the forms of the lungs; they are, however, only fully occupied by the lungs when these are largely inflated. During ordinary respiration there is a portion of each pleural cavity beyond the lower margin of the lung, and another beyond the anterior margin, in which opposed portions of the parietal pleura are in contact with one another. It must be added to this statement that the pleuræ do not line the thoracic parietes quite down to the attachment of the diaphragm in front; and that while the anterior margin of the right pleura extends to the mesial line of the sternum from the level of the second rib downwards, that of the left retreats somewhat at about the point at which the notch in the left lung commences.

2. *Regions of chest.*—It is usual and convenient for clinical purposes to map out the chest into regions. The names of those which are generally recognised sufficiently indicate their respective positions. They are as follows.—In front: the *supra-sternal*, situated immediately above the sternal notch; the *upper sternal*, corresponding to the upper half, the *lower sternal* to the lower half, of the sternum; the *supra-clavicular*, placed just above the inner half of the clavicle; the *clavicular*, corresponding to the inner half of the same bone; the *infra-clavicular*, extending from the clavicle downwards to about the level of the third rib; the *mammary*, of which the nipple may be taken as the centre, extending from about the third to the sixth rib; and the *infra-mammary*, comprising the remainder of the front of the chest;—at the side: the *axillary*, bounded by the summit of the axilla above, in front and behind by the axillary folds, and extending half-way down the thorax,

and the *infra-axillary*, occupying the lower half of the lateral aspect of the chest;—at the back: the *upper scapular*, situated above the spine of the scapula; the *lower scapular*, corresponding to the infra-spinatus fossa; the *inter-scapular*, lying between the vertebral border of the scapula and the spinous processes of the vertebræ; and, lastly, the *infra-scapular*, including all that part of the back of the chest situated below the lower angle of the scapula. We have not assigned exact limits to all of these regions, partly because different writers assign different limits to them, partly because, convenient though they are for ordinary purposes, it seems to us preferable, when there is need of exactitude, to define the position and limits of areæ by reference to the ribs and other fixed landmarks, and by measurement.

B. *Pathology of Voice, Respiration, Cough, and Expectoration.*

In the investigation of diseases, and more especially those of the respiratory organs, much information may often be obtained by attention to any peculiarities which the voice or respiratory acts may present, to the presence or absence of cough and to the quality of the cough, and to the character of the expectoration.

1. *Voice.*—The voice may be feeble, tremulous, or absent, its quality or its pitch may be changed, and its register or compass may be contracted or modified.

Mere *feebleness of voice* is so commonly associated with the presence of diseases, whether in the lungs or elsewhere, which cause enfeeblement of the muscular system generally, that it attracts comparatively little notice. It depends essentially on feebleness or imperfection of the expiratory act, however these conditions may be brought about. Hence we meet with it whenever there is much dyspnœa present, especially if at the same time the respirations be hurried and shallow; and it is a notable characteristic of all cases in which, whether from disease of the spinal cord high up, or from any other cause, the diaphragm or the intercostal muscles or the muscles of expiration are paralysed or weakened. Tremulous or bleating voice arises from want of accurate control over the expiratory muscles or over those of the larynx itself. It is met with chiefly in old age and in persons who are hysterical or nervous.

Absence of voice, that is to say, total inability to produce laryngeal intonation, and the capability of evolving only that wheezy sound which forms the basis of all whispered vowels, indicates that the patient is unable to bring the vocal cords into apposition, and that the rima glottidis remains during his attempts at phonation in that patent condition which it affects during ordinary respiration. This condition is due to a paralytic state of the adductors of the vocal cords, which may be either of functional or of organic origin.

The *pitch of the voice* depends on the action of the larynx alone.

There are two widely different diseases in which the voice very frequently becomes markedly high-pitched or squeaky; these are Asiatic cholera and leprosy. It becomes high-pitched also in those who are under the influence of laughing-gas. Trousseau points out that when there is lesion of the superior laryngeal nerves alone, there is, owing to the consequent paralysis of the crico-thyroid muscles, inability to utter the higher notes, and the voice consequently becomes deep-toned; and further that in some forms of laryngeal inflammation, attended with hoarseness, the voice is low-toned on first rising, and becomes higher as the day advances. It is obvious that in the last two cases the *compass of the voice* also must be contracted.

Pathological changes in the *quality of the voice* are largely dependent on conditions external to the larynx. It is thus that it gets altered when the faucial passage is narrowed by the presence of enlarged tonsils, when the soft palate is stiff and sore from inflammation, or paralysed after diphtheria, or when there is cleft palate. Hoarseness or roughness of voice—in other words, loss or impairment of the musical quality of the voice—may depend upon any circumstance which interferes with the regular vibration of one or both of the vocal cords. Thus it may arise from inflammatory or other thickening of the cords, from ulceration, from the presence of warty or other growths, or from the adhesion of mucus or other matters to their surface; and it not unfrequently arises simply from the fact that while one cord acts perfectly, the other cord is paralysed. Hoarseness passes on the one hand into the normal intonation of the voice, on the other into absolute aphonia.

2. *Respiration.*—Ordinary quiet breathing is effected without appreciable effort, and with scarcely audible sound, at the rate, in the adult, of from sixteen to twenty respirations in the minute—their number having to the beats of the pulse a ratio of about one to four or five, and the act of inspiration being probably somewhat longer than that of expiration. The respiratory acts are liable in health, and still more in disease, to many deviations from the above rule; they may be modified in frequency, depth, and strength, and may be attended with more or less noise, discomfort, and effort.

The frequency of respiration is diminished in syncope and collapse and various affections implicating the nervous centres, and occasionally also in cases of dyspnœa dependent on the presence of some mechanical obstacle to the entrance and escape of air. It is generally increased in inflammatory and febrile disorders, in affections of the lungs, pleuræ, and heart, and, above all, in some forms of hysteria, in which indeed the acts have been known to exceed one hundred in the minute. The *depth* of the respiratory acts is usually in inverse proportion to their frequency. Hence when they are rapid, they are also, as a rule, shallow and inefficient; when abnormally slow they are deep and laboured. Under these latter circumstances especially, the relative

duration of inspiration and expiration is frequently considerably altered; in some cases, as in certain forms of gastro-intestinal disturbance and in some varieties of cardiac affections, the inspirations are prolonged and sighing; in others, and more especially in cases of emphysema, asthma, and mechanical obstruction of the larynx or trachea, the duration of expiration becomes relatively very largely increased. The respiratory rhythm is affected in another way in a variety of breathing to which Dr. Stokes has called special attention, which only occurs in a marked form when death is impending, and chiefly, he thinks, in cases of enfeebled heart. It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length until a state of apparent apnœa is established.

The term *dyspnœa* is employed of all cases in which respiration is unusually rapid, and equally of all those in which it is unusually slow, or even of normal rate, but attended with marked exertion. The special muscular efforts which accompany and indicate dyspnœa are in some cases apparently limited to the dilatation of the nares during each inspiration; in some to this act in conjunction with rhythmical opening of the mouth; in other cases the muscles of the neck also act more or less powerfully; and between these conditions and the phenomena of the asthmatic paroxysm, in which breathing is effected with the most agonising efforts, and every ordinary and extraordinary muscle of respiration is called into powerful action, there are all gradations. The abnormal sounds which attend dyspnœa are sometimes a sniffing sound produced in the nares, sometimes a sucking or sipping sound manufactured with the lips, sometimes a panting sound effected in the throat.

Further, whenever the rima glottidis is narrowed, and incapable of enlarging to permit the free passage of the breath, or the trachea is diminished in calibre, as it may be from the presence of a diphtheritic membrane, or the pressure of an aneurysmal tumour, both inspiration and expiration acquire what is called a 'stridulous' character; they become remarkably harsh and rough, presenting in some cases almost a metallic ring. These peculiarities are always greatly increased when respiration is hurried, or during the inspiration which precedes a cough. Closely related acoustically to stridor is wheezing or whistling, which is a common attendant on old bronchitis, and always accompanies the asthmatic paroxysm.

3. *Cough* is a modification of breathing, which is characterised by a deep-drawn inspiration, followed by closure of the glottis and a series of short but violent expiratory acts. It is generally excited by some irritation or abnormal accumulation, either at the glottis, in the trachea, or in the larger bronchial tubes; or it is a simple nervous affection. The act of coughing is generally preceded by tickling or some other uncomfortable sensation referrible either to the larynx or

to some part of the trachea. The cough may be unattended with expectoration or dry, either because there is nothing to be expectorated, or because the offending matter cannot be dislodged; or it may be accompanied by more or less abundant discharge of mucus or other matters. In the first case the cough may be that of the early or dry stage of inflammation, or of hooping-cough, or it may be a nervous disorder. In the second case—that in which the cough is ineffectual—there is probably some mechanical obstacle in the larynx or trachea to the discharge of peccant matter, or clogging of the bronchial tubes with tenacious or even solid material, or limitation of the mucus to some of the smaller tubes. The third case does not call for special remark.

All coughs are from their very nature *spasmodic*; but some, from the entire want of control which patients have over them, and from peculiarities which they present, are especially deserving of that epithet. The most remarkable of these are the paroxysmal coughs which characterise pertussis, obstruction of the trachea, and spasmodic croup. In pertussis a deep inspiration is followed by a rapid succession of spasmodic expiratory efforts, continued until further expiration is mechanically impossible; then follows a long inspiration, effected through the spasmodically closed glottis, and yielding the characteristic whoop. In spasmodic croup there is a series of coughs, the expirations being remarkably harsh and noisy, the inspirations attended with a whistling sound. In tracheal obstruction, the inspirations are prolonged, stridulous, and wheezing, the expirations also wheezy and often unattended with marked laryngeal noise, and these are repeated in rapid succession until the patient appears on the eve of suffocation, when probably he is relieved by the discharge of a little mucus.

The *noises* which attend the acts of coughing have already been partly considered. They may be divided into those of the inspiratory act and those of expiration. As regards the former, if there be spasmodic closure of the glottis, there is either a whoop, as in pertussis, or a whistle, as in spasmodic croup; but if the laryngeal orifice be obstructed by the presence of a false membrane upon it, or if there be an impediment in the trachea, the sound of inspiration becomes wheezy or harsh. In the majority of cases the sound of inspiration is merely that of a deep-drawn breath. The sounds which attend the expiratory element of the cough are due to the condition of the laryngeal orifice and the force with which the expiratory blast bursts through it. Thus, if there be no impediment to the full inflation of the lungs, and the vocal cords be in a normal condition, the expiratory acts will necessarily (if forcible) be more or less noisy and at the same time musical. But the character of the sound will of course be modified according to the degree of tension of the cords, and in some measure in accordance with the degree in which they may have become thickened or have lost elasticity in consequence of inflammatory or other change.

Many of the most noisy coughs are those which occur in hysterical or nervous patients, in whom the vocal cords are healthy in structure, and in those persons in whom they are affected with only slight catarrh. If, on the other hand, the vocal cords be prevented from vibrating freely, as may happen when the soft parts above the rima are greatly swollen, or the cords themselves and other parts of the larynx are invested with diphtheritic membrane, or laryngeal or tracheal obstruction renders the expiratory blast feeble and insufficient, the cough loses its musical or sonorous character, and becomes wheezy and voiceless.

4. *Expectoration.*—The expectoration is often a valuable aid to diagnosis. Many persons, especially those beyond middle age, spit on rising from bed in the morning a small quantity of viscid or tenacious mucus, studded with black particles. This black matter, which is supposed to be of extraneous origin, is nevertheless contained in cells. Such expectoration indicates the presence of a little bronchorrhœa, but is hardly to be regarded as a sign of any actual disease. In inflammation of the respiratory passages, the discharge of mucus becomes augmented, sometimes enormously. This at first is a watery, slightly viscid, colourless fluid, of saline taste and reaction, containing microscopically shed epithelial cells and mucous and granular corpuscles. Later on its viscidity increases (sometimes it is very viscid from the beginning), it becomes difficult to void, and coalesces after expectoration into a coherent mass, which adheres to the vessel into which it is discharged. Such expectoration is sometimes colourless, sometimes greenish or yellowish, and occasionally streaked with blood. At a still later stage the sputa become opaque and yellow or green, less viscid, and acquire either the physical characters of pus or characters between those of pus and mucus. This purulent conversion may be general or partial, and in the latter case the sputa not unfrequently present the so-called 'nummulated' character due to the fact of thick opaque pellets floating in transparent watery mucus. All these varieties of expectoration may arise in the successive stages of acute or chronic bronchitis—the presence of pure mucus alone indicating for the most part acuteness of inflammation; that of pus, the supervention of a chronic condition, or possibly the approach of convalescence. The nummulated character implies that, while the bronchial tubes are partly secreting mucus, they are partly secreting pus, or pus is gaining an entrance into them from other sources. Nummulated expectoration is frequently met with in cases of dilated tubes, of pulmonary cavities, and of empyematic or other abscesses which communicate with the lung and discharge through it. But it is also met with in simple chronic bronchitis. In many cases, when abscesses open into the lungs, the expectoration consists of almost pure pus. The expectoration of ordinary acute pneumonia is characterised by extreme viscidity with more or less transparency, and by the fact that it is uniformly tinged with blood. Its colour

presents numerous gradations between yellow or reddish-brown (rusty) and a bright vermilion. As the disease passes into convalescence, the expectoration loses its peculiar colour and its viscidity and gets muco-purulent, like that of bronchitis. In certain cases it becomes either distinctly purulent, or, while still incorporated with blood, watery. The latter form of expectoration is sometimes likened to plum-juice.

Blood in streaks occurs in bronchitis, blood uniformly diffused in pneumonia; but very often unmixed blood is poured into the bronchial tubes, and is discharged thence, either still unmixed or blended only with a small quantity of mucus. The sources of such pulmonary hemorrhages are the bursting of aneurysms into the air-passages or lung-tissue; the laying open of branches of the pulmonary artery or vein during the progress of tubercle, carcinoma or other destructive morbid processes; intense hyperæmia of bronchial tubes or of the walls of pulmonary cavities; and pulmonary apoplexy. In the last group of cases the hemorrhage is generally scanty; in the others it is often extremely profuse. Copious and sudden hæmoptysis is generally characterised by the arterial character of the expectorated blood, and by its more or less frothy condition; but when the hemorrhage is small in amount, and expectorated at intervals, it is often in the form of dark brownish or blackish-red pellets.

Casts of the air-passages are not unfrequently expectorated. In diphtheria, membranous casts of various parts of the larynx, trachea, or larger bronchial tubes are often thus discharged. More rarely, branching casts of systems of the smaller bronchial tubes are spat up. These are sometimes mere coagulated blood, sometimes simple pneumonic exudation concreted in the smaller bronchial tubes, sometimes casts of laminated texture apparently identical with diphtheritic membrane.

Among the *foreign bodies* which are occasionally expectorated must be mentioned hydatids, either from the lung itself or from the liver, and earthy concretions—the remnants of dried-up tubercular matter in the lungs or bronchial glands. No doubt tubercular, carcinomatous and other such matters are occasionally brought up, but they can very rarely be recognised as such. The progress, however, of destructive processes in the lungs may often be detected or verified by the discovery on microscopic examination of fragments of lung-tissue. A convenient way of finding these is to boil a small quantity of sputum with a strong solution of caustic soda until they form a thin watery fluid, to place this in a conical glass for the purpose of subsidence, and then to examine the sediment microscopically. The matters to be especially looked for are the curved fragments of elastic tissue which bound the orifices of the smaller bronchial tubes, air-passages, and air-cells.

Purulent expectoration often has a faint, sickly, or sweetish odour. The only smell, however, of clinical importance is that which is commonly attributed to the presence of gangrene. This is horribly fetid, difficult to describe, but when once smelt impossible to forget.

It may be readily detected in the sputum itself; but it is evolved most intensely with the patient's breath during the act of coughing. The sputa which yield this odour are generally distinctly purulent, occasionally nummulated, and have usually a more or less discoloured or dirty-looking aspect. They may be intermixed with blood in a more or less altered condition.

C. *Investigation by Sight and Touch.*

The information which may be acquired through the eye by inspection, and through the hand by palpation, as to the condition of those functions of the respiratory system which lie within the scope of such methods of investigation, is obviously very considerable. We will speak of them in relation: first, to the larynx and trachea; second, to the intrathoracic organs.

1. *Larynx and trachea. Laryngoscope.*—The apex of the epiglottis may sometimes be seen, and its condition ascertained, by merely looking into the throat when the mouth is widely opened and the tongue depressed. Its condition and also that of the parts bounding the upper orifice of the larynx may sometimes, especially in children, be roughly yet sufficiently determined by means of the tip of the fore-finger passed back through the mouth into the fauces. The invention of the laryngoscope, however, and the perfection to which its use has been brought, make it now possible for us to determine the condition of the larynx with the utmost nicety and to employ local remedial measures with intelligence and accuracy. The apparatus usually employed for laryngoscopic examination comprise: first, a lamp yielding a steady, bright flame provided with some form of reflector or condenser; second, a circular concave mirror, from 3 to 3½ inches in diameter, and with a focal length of 12 or 14 inches, which should be freely movable in all directions upon its support, and should either be fixed to the forehead immediately above the eye by means of an elastic band, or attached to a spectacle frame and adapted to the right eye—in the latter case it should be provided with a central perforation of an oval form; third, a laryngeal mirror of metal or silvered glass, of circular, oval or quadrilateral form, and varying in diameter from half an inch, for a young child, up to an inch, fixed at an angle of about 120° to a thin metallic stem or shank, which should itself be fastened into an ivory or wooden handle. The entire length of the combined shank and handle should measure from 6 to 8 inches. In making an examination, the patient should be seated in front of the examiner, with his head inclined a little backwards; the lamp should be placed at the side of, and somewhat behind, his head; and the examiner should so arrange himself that his eye, with the mirror adapted, should be at the distance of about a foot in front of the patient's mouth. The mirror should be so adjusted as that the light which it reflects may be brought to a focus

at about the back of the patient's uvula. He should then be directed to open his mouth widely and to protrude his tongue; the point of which should be firmly grasped and firmly but gently drawn forwards by the fore-finger and thumb of the operator's left hand, enveloped in a cambric handkerchief or towel. Then, the area of reflected light being steadily kept upon the point previously indicated, the laryngeal mirror (which has been previously warmed either over a lamp or by immersion in hot water, in order to prevent the condensation of the patient's breath upon it) is to be carefully passed backward until it reaches the base of the uvula, in which situation it must be held, with its surface facing downwards and forwards, at an angle of about 45° with the horizontal plane of the mouth. If the upper orifice of the larynx be not at once seen in the mirror, the direction of the face of the mirror may need a slight alteration, or it may be necessary to pass the mirror a little farther upwards and backwards, or otherwise to modify its position. It is important, in order that the examination be satisfactory: first, that both patient and operator be patient and steady; second, that no needless force be employed to draw the tongue forward, and that it be not injured by undue pressure against the lower teeth; third, that in introducing the mirror, neither the tongue nor the palate be touched by it, excepting of course only that part of the palate against which it has to rest; and fourth, that no single introduction be of long duration. It is best, usually, to repeat the operation several times in the course of a sitting. It need scarcely be added that many difficulties present themselves to interfere with the success of laryngeal inspection, some of which render inspection impossible, while others may be overcome with a little patience and delicacy of manipulation.

Even if the condition of the larynx be healthy, we may in some cases perceive only the epiglottis and the tips of the cornicula laryngis. In more successful observations, however, we may detect not only these bodies but all the other boundaries of the superior orifice of the larynx, including the aryteno-epiglottidean folds, the cartilages of Wrisberg, the posterior commissure, together with the rima glottidis, the true and false vocal cords, and if the rima glottidis be open sometimes the tracheal cartilages, and even the bifurcation of the trachea. All parts of the larynx, except the edge of the epiglottis and the true vocal cords, have a reddish hue, like that of the interior of the mouth, gums, or lips, the redness being usually brightest in the false vocal cords, in the cushion of the epiglottis, and over the cornicula and cartilages of Wrisberg. The vocal cords are pearly-white, the edge of the epiglottis, and, it may be added, the tracheal and the cricoid cartilages distinctly yellowish.

It is always important to observe the movements of the vocal cords, and to examine the larynx both when the rima is fully open and when it is perfectly closed. The rima is always more or less widely open during ordinary quiet respiration; but, in order to have it as widely

open as possible, the patient should be directed to draw a deep breath. In order to effect closure, he should be required to utter a vocal sound. The best for this purpose, as requiring for their pronunciation the greatest expansion of the oral aperture and cavity, are the vowel sound which is sometimes termed '*ur* vocal,' and is uttered in the words 'cur' and 'myrrh,' and the broad sound of 'a' represented by 'ah.'

The morbid conditions for which we should mainly look are swelling, congestion, ulceration, and exudation, such as may be caused by inflammation, diphtheria, syphilis, or other morbid processes; warty or other growths; paralytic or spasmodic affections of the vocal cords; and compression of the trachea by aneurysmal or other tumours.

As regards the examination of the larynx and trachea from without, the chief points which are ascertainable are: first, the presence of tenderness; second, deviation of the trachea from the middle line, which may be due to tumours either in the neck or within the thorax; and, third, infiltration and thickening of the soft parts around. Thus, in inflammatory affections of the larynx, especially in cases in which the cartilages are in a state of necrosis, thickening with induration of the surrounding tissues is often a very remarkable feature; and still more remarkable is the stony induration of parts and the fixation of the larynx which attend some cases of carcinomatous or other malignant infiltration.

2. *Chest.*—The *form* of the chest is often indicative of the presence of disease within. It must not be forgotten, however, that its general form varies widely in different individuals, sometimes from inheritance, sometimes from rickety tendency during early life; and that want of symmetry is often traceable to the preponderating use of the right hand, or to spinal curvature. But such varieties are quite independent of pulmonary affections, and we must be careful not to confuse them with those which are attributable to the latter causes.

General *expansion* of the chest is a common characteristic of patients who have suffered for many years from chronic bronchitis or asthma, especially if there be at the same time pulmonary emphysema. Partly in consequence of long-continued over-exertion of the inspiratory muscles, partly from the difficulty which emphysematous lungs have to get rid of their surplus air, the chest increases in both its antero-posterior and its lateral dimensions, and assumes a rounded or 'barrel-like' form. If such changes begin in early infancy, it is not unusual to find that, while the upper part of the chest becomes generally expanded, the lower zone (owing to the comparative weakness of the ribs in early life) undergoes more or less contraction. It is seldom that the causes here spoken of operate on one side of the chest only. General enlargement of one side may be caused by accumulation of serum, pus, or air in the pleural cavity. In such cases the intercostal spaces get widened, the intercostal depressions effaced, and sometimes (especially if the effusion be inflammatory) replaced by

actual bulging. Under such circumstances, undulation or fluctuation may occasionally be detected. In cases in which a lung is wholly or in the greater part of its extent pneumonic, the affected side remains fixed in the position of full inflation. Localised enlargements, or bulgings, may be the result of localised accumulations of air or fluid, or of the presence of aneurysmal, sarcomatous, or other varieties of intra-thoracic tumours. In cases of empyema it is not uncommon for the pus to find its way between the ribs, to form an accumulation between them and the integuments, and thus to cause a localised swelling.

Contraction of the thoracic walls is exceedingly common; but it is rarely general and symmetrical, unless it be due to natural conformation, or the consequence of rickets, or of diseases like hooping cough attended with long-continued impediment to inspiration. It is of chief clinical interest when it is unilateral or limited to definite regions. All pulmonary diseases, attended with diminution in the size of the lung, are attended with more or less marked contraction corresponding to that diminution. The most remarkable example is furnished by empyema or hydrothorax which has caused complete and permanent collapse of the lung. With the absorption or removal of the fluid the affected side gets reduced in all its dimensions, but especially flattened from before backwards, and the patient's carriage comes to resemble that of a person suffering from lateral curvature of the spine. Atelectasis, apneumatosis, cirrhosis, and the contraction of cavities are all attended with more or less manifest contraction of that area of the chest-walls which corresponds to the portion of the lung involved; but the most frequent, and on the whole the most important, of these localised contractions is that which is so commonly observed beneath one or both clavicles during the progress of phthisis.

The *movements* of the chest are often very significant. The violent muscular efforts, yet little movement of the ribs, which mark the respiratory acts of emphysematous patients with barrel-shaped chests are very characteristic. The entire quiescence or little comparative movement of the affected side in cases of effusion into the pleura or of pneumonic consolidation, and of the apex of the lung in cases of phthisis, is equally matter of interest and clinical importance.

Whenever grave notes are uttered by the voice a distinct vibratile thrill, the *vocal fremitus*, may be felt, not only over the larynx and trachea, but over the face and head, and over the whole of the surface of the chest to which lung-tissue is subjacent. The best mode of detecting this thrill in the chest is to place the palm of the hand flat and firmly on the part selected for examination. The degree in which it may be perceptible varies greatly in different persons, in dependence partly on the pitch and strength of the voice, partly on the quantity of muscle or fat present in the parietes of the chest. It is generally best recognised in male adults with spare frames. For obvious reasons it is more perceptible at the upper part of the chest, in front and between

the scapulæ, than elsewhere; and it is either absent from the area of cardiac dulness, or comparatively feeble there. It is said to be a little more marked on the right than on the left side of the chest, but the difference is at most trivial; and it may be regarded as a general rule that, with the exceptions referred to it is present in an equal degree at corresponding parts of the two sides of the chest. The presence of disease largely modifies the intensity and distribution of the vocal fremitus. Whenever there is fluid effusion into the chest, the thrill becomes greatly enfeebled or absolutely annulled over the surface to which the fluid is subjacent. Whenever, on the other hand, lung-tissue is consolidated by pneumonia, the vocal fremitus over the affected region is much intensified. It must be added that mere thickening of pleura or accumulation of solid lymph in its cavity, acts equally with fluid effusion in damping vocal fremitus; that solid growths, whether in the lung or external to it, have a like effect; and that in rare cases fremitus is diminished even over pneumonic lung. The explanation of the diminution of vocal fremitus in the several cases above enumerated is sufficiently obvious. The intensification which attends most cases of pneumonia is due apparently to the concurrence of two conditions—the one, consolidation of the vesicular tissue which increases its capability of conducting sounds; the other the permeation of the solid mass by pervious tubes along which the vocal vibrations are carried into its midst.

In support or correction of the judgments formed from the results of visual or manual examination, it is always well to have recourse to actual measurement of the chest, or of portions of it, and of the amount of expansion or movement which they undergo. It is needless to describe in detail how all such measurements are to be effected; it is sufficient, probably, to name the chief instruments which may be used for the purpose, namely, the measuring-tape and calipers, and the cyrtometer.

The last name is applied to a metal wire, or specially devised band, which admits of close adaptation to the surface of the chest, and retains its form after removal, so as to allow of a tracing being made from it.

Spirometer.—It is sometimes useful to ascertain what Dr. Hutchinson calls the 'vital capacity' of the chest by means of an instrument made on the principle of the gasometer, which he terms the 'spirometer.' He measures this capacity by the amount of air which a person who has distended his chest to the utmost is able to discharge by voluntary expiratory effort. This amount appears to be very constant in relation to stature. Thus, the average vital capacity of a man five feet seven inches high is about 225 cubic inches, and for each inch of stature above this there is an increase, and for each inch below it a decrease of about eight cubic inches. It is often difficult to make persons under examination exert a sufficient effort to manifest their true vital

capacity; but if after having done so there is any wide departure from the scale above given there is good reason to suspect the existence of some morbid condition, either involving the lungs, or interfering with the due performance of the respiratory acts. The vital capacity of women is much less than that of men.

D. *Investigation by Percussion and Auscultation.*

Of all aids to the recognition of the morbid processes which are going on within the thorax none is so important as the employment of *percussion* and *auscultation*—both, methods of investigation scarcely thought of prior to the commencement of the present century, but which, within the last fifty years, have been largely cultivated and have furnished the most valuable results both to the physiologist and to the physician.

1. By *percussion* is meant the investigation of the condition of internal organs by the sounds which are yielded by sharply striking the surfaces over them. There are three principal methods by which this may be effected, namely: 1st, by striking the surface directly either with the fist, the knuckles, or the tips of two or three fingers brought together into the form of a hammer, or by simply filliping with the nail of the forefinger; 2nd, by the use of the hammer and plexi-meter—the plexi-meter is a small thin ivory disc which, for the purpose of receiving the blow of the hammer, has to be laid firmly and flat upon the surface of the part to be percussed; the hammer, which usually has a comparatively heavy metallic head, is furnished at its striking extremity with an india-rubber pad, which alone comes in contact with the plexi-meter, and prevents the development of any sound special to the instrument; 3rd, by the employment of the fore or middle finger of the left hand as a plexi-meter, and the tips of one, two, or three fingers of the right hand as a hammer; in this case the finger of the left hand should be laid firmly and flat, with its palmar surface downwards, on the surface to be examined, and the tips of the striking fingers of the opposite hand should be brought down perpendicularly and sharply upon it. The first of these three methods of percussion has fallen into almost entire disuse, chiefly because of the needless pain which it is apt to inflict; still it may sometimes be employed with great advantage—and especially the method of filliping with the forefinger—when, as in the chest of young children, and in the exploration of the abdomen, even slight pressure of the plexi-meter is liable to displace air or fluid, the presence or absence of which at some particular point it is important to determine. The second method is a valuable one, especially for clinical teaching, because the sounds which it evolves are loud and readily distinguishable by a class of students. The third method is that which is in general use, partly

because of its great convenience, and partly, because, although the sounds which are elicited by it are comparatively feeble, they are perfectly appreciable. It may be noted here that, whenever it is sought to compare by percussion the corresponding parts of opposite sides of the trunk, it is most important that for each pair of examinations the pleximeter, whether the finger or the disc, should be symmetrically placed, and the force and direction of the blows should correspond; and, further, that it is important as far as possible to prevent any sound due to the instrument itself from interfering with that elicited from the part percussed.

a. Normal percussion phenomena.—The sounds which are yielded to percussion by the healthy chest are of two kinds, *resonant* and *dull*. These words are by no means well-chosen, but they are sanctified by long and general usage, and would be difficult to replace. By resonance we mean to imply the presence of more or less musical quality, by dulness the absence of such quality.

i. *Resonance.*—A resonant sound is yielded by all those parts of the chest-walls which are by their deep aspect in contact with lung, and by that part of the left half of the chest to which the stomach is subjacent. The quality of the resonant sonnd which is evolved on percussing the pulmonary regions of the thorax is difficult to describe, but sufficiently characteristic to be easy of recognition when once it has been heard. It is somewhat deep in tone, short in duration, and vaguely musical. It differs, however, in some degree in quality in different parts of the chest, and considerably in different individuals. Hence it is important, in judging of the significance of percussion sounds, not to assume the existence of a normal standard sound with which all others must be compared, not to compare the resonance of one person's chest with that of another, nor, indeed, to compare indiscriminately the resonance of different parts of the same individual's chest. But we should carefully compare the sounds yielded by the corresponding points of the two sides of the chest. The chief cause of the resonant quality of the percussion note is the vibration of the struck walls which is permitted by the fact that an elastic medium—the air—is situated on either side of them. It is obvious, however, that the elasticity of the inflated lungs is less than that of the free atmosphere outside, and that hence the vibration of the thoracic walls must be to some extent less perfect than it would be were the air on both sides equally free to move. The sound, we repeat, is mainly due to the vibration of the thoracic walls alone; but it is difficult (owing to the somewhat irregular form and structure of these walls, and to the interference with their vibration caused, on the one hand, by the solid organs which lie here and there beneath them, and, on the other hand, by the junctions of the chest with the upper extremities) to determine to what extent and in what manner these vibrations are effected. It seems reasonable, however, to assume that so much of each half of the thorax as bounds lung-tissue vibrates bell-like when any part of that half is struck, and that

the impure musical sound which is elicited comprises a fundamental note due to the vibration of the whole or a large portion of the side, and harmonic tones due to the vibration of aliquot parts of it. This view is entirely compatible with the fact that percussion notes of somewhat different quality are yielded on striking different parts of the surface, and, if correct, makes it obvious that the sound elicited by the percussion of any spot is by no means necessarily indicative of the condition of the lung-tissue immediately beneath it. It must be added that some, though a very variable, quantity, of thoracic resonance is independent of the presence of air beneath the chest-walls. Thin and elastic bones, even if they be imbedded in solid tissue, vibrate sensibly when percussed. A sound which is not absolutely dull may be obtained by percussing the bones of the skull; and some degree of resonance may always be elicited over the sternum even when no lung-tissue is subjacent to it. The ribs, also, especially if the patient be thin, usually yield a somewhat resonant sound.

The stomachal resonance may always be recognised (though variable in extent, distinctness and quality according to the degree of distension of the organ with gas and to the level which it attains within the cavity of the thorax) at the lower part of the left side of the chest, both posteriorly, laterally, and in front, but chiefly in the last two situations. It may readily be distinguished from the normal lung-resonance by its much more distinctly musical character, by its purer tone and generally higher pitch. The sound is often termed *tympanitic*, or drum-like.

ii. *Dulness.*—Absence of resonance, or dulness, is observable on percussing the præcordial region, and that part of the right side of the chest between which and the liver no lung-tissue intrudes. This sound, again, can be better appreciated by a single experiment than by any description. It may be described as short, somewhat sharp, and unattended with any appreciable ring or tone. The feeble sound elicited by the percussion of the thigh is often referred to as the very type of a so-called 'dull' sound. It differs, however, materially from that which is yielded by the præcordial region. And, indeed, the quality of dulness, in the clinical sense, presents many varieties, and passes by insensible gradations into that of resonance. Many so-called dull sounds become obviously musical when tested stethoscopically.

b. Abnormal percussion phenomena.—Percussion in cases of pulmonary disease is mainly of use in enabling us to ascertain the presence and define the limits of consolidation, pleural effusion, and morbid growths, and of conditions causing extension or modification of resonance.

i. *Dulness.*—Whenever any considerable mass of lung-tissue is rendered solid, either by tubercular infiltration, by inflammatory deposit, by effusion of blood, by carcinomatous growth, or in any other way, all that area of the chest-wall on which it abuts loses its normal

resonance and becomes more or less dull. The presence of fluid in the pleura causes dulness in even a more marked degree up to the level of the effusion. The recognition of the cause of dulness must depend partly on the situation, extent, and form of the area of dulness, partly on a variety of considerations, the collective significance of which will be more conveniently discussed hereafter. It may be mentioned, however, that pneumonic consolidation usually occurs in the lower part of the lung, tubercular infiltration at the apex, and that pleuritic effusion, unless it be circumscribed by adhesions, or so abundant as entirely to compress the lung, may often be recognised by the changing level of the upper limit of dulness in accordance with the different positions which the patient's trunk is made to assume. But although marked dulness is always present when consolidation is extensive and continuous, it is often absent, or at all events scarcely appreciable, when either an extensive tract of lung-tissue uniformly contains more solid matter or fluid and less air than natural, or miliary or larger nodules of solid tissue, separated from one another by a network of crepitant tissue, are even thickly distributed. Thus congested or œdematous lungs, and lungs in the early stage of inflammation, on the one hand, and lungs which are the seat of disseminated tubercles or of lobular pneumonia on the other, are not unfrequently so strikingly resonant as utterly to deceive the too confiding percusser.

ii. *Resonance.*—It is obvious that, whenever there is any extension of the area of dulness, there must be a corresponding diminution in the area of resonance. On the other hand, the normal areæ of thoracic dulness are not very unfrequently reduced or effaced by the extension of resonance. In association with such changes, and sometimes indeed apart from them, the resonance of the resonant area is altered in intensity, quality, or pitch. To denote different varieties and degrees of resonance many terms have been employed—such, for example, as wooden, leather-trunk-like, tubular, cavernous, tympanitic, high-pitched, and the like. Some of these are obviously fanciful, some indicative of a foregone conclusion with regard to the case under examination; but others do, to some extent, explain themselves, are applicable and convenient. Augmentation of resonance (to which condition the epithet '*tympanitic*' is sometimes given) may often be heard over emphysematous lungs, or lungs distended (as they sometimes are in cases of acute bronchitis) with air, but especially over a pleural cavity the seat of pneumothorax. It should be added, however, that in such cases the augmentation of resonance is for the most part attended with the production of a purer note, and frequently a note of somewhat higher pitch, than characterises the normal chest-resonance of the patient. But augmented resonance, with change of quality and pitch, is often heard under very different conditions from those which have just been considered. It is frequently observed, for example, that in cases of extensive pulmonary consolidation, or pleural

effusion, of one side, the crepitant remnant of lung-tissue evolves under percussion a much purer note than the corresponding part of the opposite lung. The sound is sometimes described as being more resonant than that yielded by the opposite side; possibly it may be so, but it is at all events more distinctly musical, and always of considerably heightened pitch. Not unfrequently indeed the sound is almost exactly like that produced by percussing a portion of small intestine or striking one of the treble keys of the piano. This modified resonance is most frequently observed over the apex of the lung when the rest of the organ is consolidated or compressed, but by no means necessarily occurs only in that region; and may sometimes be distinctly heard over portions of pneumonic lung which are not yet completely solidified. Various explanations of the phenomena here described have been given. With respect to the increased resonance which attends pneumothorax, emphysema and the like, it will probably be admitted that it is due to the more ready and perfect vibration of the thoracic walls which the relative increase of air beneath their inner surface permits. This explanation, however, will scarcely apply to the higher pitch which the percussion note usually then acquires, and is certainly not applicable to those cases in which high-pitched resonance occurs over partly consolidated lung, or lung in the neighbourhood of consolidated tissue. Reverting to the explanation we have already given of the ordinary resonant sound yielded by the thoracic walls (namely, that in its production all those parts of the thoracic walls which are in contact with the lung under examination vibrate, bell-like, producing a somewhat obscure assemblage of fundamental and harmonic tones, the general effect of which is deep in some sort of proportion to the extent of surface which vibrates); and knowing that (other things being equal) the smaller a vibrating area becomes the higher will be the fundamental tone it yields; and seeing that such a diminution of vibrating area necessarily takes place when there is extensive consolidation or fluid effusion, and not improbably occurs in the first stage of pneumonia over the affected portion of lung; it seems reasonable to assume that mainly in these considerations is to be sought the explanation of the acoustic phenomenon in question. It must not be forgotten, however, that the increase of tension, which in pneumothorax, and in a less degree in pleurisy with effusion, the thoracic walls experience, also tends to the production of a higher note.

The question 'how far can the percussion note be modified by conditions within the chest other than those which have been discussed'? still remains for consideration. Can it, for example, be affected by the neighbourhood of solid matter separated from the parietes by a layer of crepitant tissue? Can it be modified by the internal resonance of cavities which abut upon the surface? The former of these questions has been answered in the affirmative by most writers; who assert that,

by regulating the force of the percussion stroke, the resonance due to the intervening lung and the dulness due to the subjacent solid structure, can be distinguished, and that thus the extension of the heart beneath the thin edge of the lung, and the ascent of the liver behind the lower margin of the right lung, can be easily detected. We confess we are not satisfied of the general truth of this assertion. We may remark, however, as bearing upon it—that the quality and power of a musical tone differ according to the part of a vibrating cord or surface which is struck, and that hence the quality and power of percussion tones are doubtless modified as we pass from the centre of a resonant area to its margins; and again that it is quite possible in the yielding chests of young children, if undue pressure be made by the pleximeter, to compress or displace the thin edge of the lung lying between the heart or liver and the parietes, and so to obtain dulness, where normally resonance should be elicited. But this is the consequence of pressure and not of percussion. As to the latter of these questions, there is no doubt, that if auscultation be practised at the same time as percussion, the resonance due to a subjacent large cavity may occasionally be recognised in the form of a superadded musical twang. There is, however, one variety of percussion sound which certainly owes its peculiarity to the conjunction of a sound produced within the chest, with that due to the vibration of the thoracic walls, namely, the *bruit de pot fêlé*, or *cracked-pot sound*—a sound which may be almost exactly simulated by clasping the hands crosswise and then striking the back of one of them sharply against the knee-cap. The 'chink' which distinguishes it appears to be due to the sudden compression of a portion of lung-tissue and the sharp expulsion of the air which it contained with an audible hiss through the bronchial tubes. To produce the sound the percussion stroke must be forcible, and made while the patient is expiring with his mouth open. It is chiefly producible in the front of the chest, either at the apex or in the mammary region. It may indicate the presence of a cavity in the lung, but is more commonly produced in the healthy chests of young children owing to the great yieldingness of their thin thoracic parietes, and in patients suffering from pneumonia or pleurisy in association with the high-pitched resonance so often present.

c. Resistance.—One further indication of importance often furnished by percussion is the presence of unyieldingness or resistance. In the percussion of healthy chests the resilience of the parietes can always be in some degree appreciated; it is indeed so constant and essential a factor of the process that on that very account it may escape observation. But in cases of solid growths in the cavity of the thorax, and in cases even of pleurisy with much thickening of pleura and much distension, the rigidity of the thoracic walls over some limited area, and their total want of elasticity and of yieldingness are quite remarkable and unmistakable.

2. By *auscultation* is meant the process of listening, either by applying the ear directly to the surface, or by the aid of some conductor, to sounds evolved within the body. The direct application of the ear to the surface is in some respects preferable to any other mode of auscultation. Many sounds are thus heard much more distinctly than they otherwise would be heard; and some delicate but distinctive sounds are wholly lost in their passage along a conducting rod or tube. But, on the other hand, the naked ear cannot be applied with ease to all parts which it is desirable to auscultate, nor can we by its aid limit our examination with precision to minute areæ. The objections on the score of delicacy and cleanliness are sufficiently obvious.

The instrument which is employed to convey sounds produced within the body to the ear of the observer is termed the '*stethoscope*.' Of this innumerable forms and varieties have been invented and are in use. As to material, they have been made of bone, ivory, silver, gun-metal, gutta-percha, and different kinds of wood; as to form, they are always cylindrical in the whole or greater part of their extent —in the latter case being provided at one end with a circular disc to fit the ear, at the other end with a conical expansion, the circular base of which is to be applied to the part under examination. Further, they are made of different lengths, sometimes solid throughout, but generally with a cylindrical channel running through the stem from the ear-piece downwards to the conical enlargement, where it undergoes a corresponding dilatation. The material, the length and the general form of the instrument are matters of very little real importance. The great desiderata are that it should be light and portable, that the ear-piece should be one that readily adapts itself to the ear of its possessor, and that the conical enlargement at the opposite end should be of medium size, and that (if provided with an opening) its margin should be sufficiently broad and rounded to admit of its adjustment without causing pain.

There are certain peculiarities in the acoustic properties of stethoscopes which it is well to be aware of. Solid stethoscopes undoubtedly convey sharp impulsive sounds and musical notes with great intensity; but they do not transmit the respiratory rustles and other feeble and unmusical sounds with anything like the distinctness with which the hollow stethoscope conveys them. The difference is very much that existing between a speaking-tube which readily transmits the whispered voice, and a solid rod which, with the aid of a sounding-board, reproduces at a distance the full music of a piano with which its opposite extremity is in contact. The hollow stethoscope, however, combines in itself the properties of both, and is therefore the preferable instrument for common use. Again, it is indisputable that certain sounds are much more distinctly audible with some hollow stethoscopes than others; and that this fact is, in

some instances, due to differences in the length of the instruments. The explanation appears to be that the tubes of stethoscopes consonate, according to their length, with certain definite notes and certain of their harmonics.

Besides the simple varieties of stethoscope above considered, there are two others which are often of considerable service. These are the *binaural* and the *differential* stethoscopes. In the former, which was invented by Dr. Leared, the stem arising from the conical end divides into two branches, the points of which respectively fit into either ear. Both ears are thus equally engaged in listening to the sounds emanating from the area under examination, which are hence intensified and on the whole more easily appreciated. The other form of stethoscope is also binaural in the sense that there is an ear-piece for each ear, and both ears are engaged; but the tubes which are prolonged from the ear-pieces remain distinct from one another and terminate each in a conical expansion. The advantage of this arrangement is that by it the auscultator is enabled to hear and determine the synchronism or asynchronism of sounds which are developed at different spots.

In using the stethoscope, it is of essential importance that (if it be hollow) its lower end should rest evenly on the surface to which it is applied, that the ear-piece should be adjusted accurately to the ear, that nothing whatever should be in contact with the instrument save the ear of the auscultator and the surface under examination, and that there should be no rustling or friction or other noises in connection with the patient's skin or clothes. It is always best to listen to the naked skin; and, if covering be necessary, it should be as thin as possible and in one layer only. Further, it is sometimes well to close the opposite ear against extraneous sounds.

a. Normal auscultatory phenomena.—The sounds audible through the stethoscope applied over the healthy respiratory organs are those of respiration, articulation, and phonation. In morbid conditions of these parts, the acoustic phenomena attending the several acts referred to become variously and often largely modified, and others of a totally different kind are often superadded.

i. *Auscultation of the breath.*—If we apply the stethoscope to the larynx or trachea during ordinary respiration, a somewhat harsh blowing sound is heard to accompany both the act of inspiration, and that of expiration. The sound is like that of the loudly-whispered vowel represented by the syllable 'ur,' or like the whispered consonantal sound of the letter *w*. Each sound lasts as long as the act which produces it, is uniform in character throughout, begins and ends abruptly, and is separated by an obvious though very short interval from the sound which follows it. That which attends inspiration is somewhat sharper and louder than the other, and both may be increased or diminished in intensity by varying the force of the respiratory movements. The sounds

are almost certainly developed at the narrowest part of the tube, namely the rima glottidis, by the rustle which its interference causes in the current of air passing through it. The slight but obvious differences in quality and force which distinguish them from one another are hence explicable; the sound produced at the rima being carried inwards with the inspiratory current, outwards during expiration. Ordinarily, the sound attending expiration is more audible to oneself and to bystanders than that attending inspiration. The sounds here described, though somewhat modified in character, are in general still audible over the manubrium of the sternum, and between the scapulæ, at and above the level of the roots of the lungs.

Over the lungs themselves the sounds which attend the respiratory movements are of a very different character from the above. The inspiratory sound is difficult to describe: it has a kind of rustling character, and is feebler and of lower pitch than the corresponding tracheal murmur; the expiratory sound is often absent, and when present is still feebler and lower in tone than the inspiratory sound. Moreover, the two sounds, instead of presenting uniform intensity throughout, and being separated by a distinct interval from one another, commence and die away so gradually that they seem like mere pulses of a continuous murmur. The healthy pulmonary sounds vary a good deal in intensity, and, in some degree, in quality in different individuals; there are also slight differences between them as heard at different parts of the same chest; and not unfrequently, especially at the apex, the sound towards the end of a deep inspiration assumes an indistinctly crepitating character. What is the cause of these sounds? That they are not made in the larger air-passages, and conveyed through the spongy tissue of the lungs to the surface, seems clear from the fact that in those cases where, from contraction of the larynx, trachea, or bronchial tubes (as in laryngitis, pressure of an aneurysm, and asthma), a peculiarly intense noise is made in these canals during respiration, the pulmonary murmurs, instead of being correspondingly augmented, are diminished or actually suppressed. The ordinary explanation is doubtless the correct one, namely, that they are produced in the minuter air-passages and air-cells by the passage of air to and fro in them, and by the changes of form—the movements—which these parts undergo.

ii. *In auscultating the voice*, it is important to recollect the fact that phonation takes place—the music of the voice is manufactured—at the rima glottidis by the vocal cords; that articulate sounds are formed only in the cavity of the mouth, by means, chiefly, of the lips, tongue, and palate. If the larynx or trachea be examined stethoscopically during the act of speaking aloud or singing, the musical notes which are evolved are conveyed through the instrument to the ear with almost painful force; similar sounds, diminished somewhat in intensity, are also audible over the manubrium of the sternum and between the

upper parts of the scapulæ behind. They are still audible, but with much less force, over the whole of those portions of the chest which have lung-tissue subjacent to them. The sounds are usually somewhat more intense above than below, in front than behind, and at the lower part posteriorly sometimes present, even in health, a somewhat bleating character. The degree in which vocal resonance or *bronchophony* is audible varies in different individuals, chiefly in dependence on the pitch and quality of the voice. Thus it is, as a rule, more obvious in those who have a deep voice than in those whose voice is high, and in men therefore than in women or children. It may be added that it is often distinct when vocal fremitus is quite imperceptible, and that in some individuals it scarcely exists at all. The articulate voice is always best distinguished when the patient speaks in a whisper: words thus uttered are distinctly transmitted through the stethoscope applied to the windpipe in the neck, or along its course in the thorax, or over the situation of the bronchi; they may also occasionally be heard over the apices of the lungs of healthy persons, especially children. This phenomenon is termed '*pectoriloquy*.'

b. Abnormal auscultatory phenomena.—The respiratory sounds are often much modified in disease. We have adverted to the fact that they are frequently not only greatly diminished, but actually absent, in certain cases of obstructive disease of the larger air-passages; they are enfeebled also whenever the respiratory movements are themselves feeble, and are generally much weakened or even annulled where the lung is compressed, consolidated, displaced, or where fluid, air, or solid matter lies between it and the thoracic walls. On the other hand, the respiratory sounds are necessarily intensified whenever the acts which produce them are unusually vigorous. It is due, doubtless, to this cause alone that they may often be heard with preternatural loudness over the healthy lung of a patient whose other lung is pneumonic or compressed by pleural effusion.

i. *Tubular or bronchial breathing* is a modification of respiratory sound frequently heard in lungs consolidated by pneumonia, compressed by pleuritic effusion, or containing smallish cavities, of whatever origin, imbedded in airless tissue. It almost exactly resembles the breath-sounds audible over the trachea. The inspiratory and expiratory elements begin and end abruptly, are uniform throughout, and separated from one another by a distinct but short interval; moreover the expiratory sound is somewhat deeper and less distinct than that of inspiration. They vary in quality in different cases and under different circumstances, but are generally higher in pitch than the tracheal sounds are. It is necessary, in order to their full development, that the respiratory acts be moderately forcible, that the air-tubes of the portion of lung under examination be not completely obstructed, and that they do not contain mucus or other matters which are productive of crepitating and other such adventitious sounds. Hence, in pneumonia, tubular

breathing may be absent or incapable of recognition if the bronchia be blocked up with casts; in pleurisy, if the compression of the lung be so great as to involve the obliteration of the tubes; in pulmonary excavation, if the vomicæ have no free connection with the air-channels; and in all such affections when the cavities or tubes are loaded with mucus or other fluids. Various explanations have been offered of the production of tubular breathing. By some it has been held that the sounds heard over the affected portion of lung are simply those manufactured at the rima glottidis, conducted to the ear through the diseased tissues. Others consider that the tubular sounds are actually produced by the to-and-fro movement of air in the tubes of the diseased tract. While others again, with Skoda at their head, regard them as the laryngeal sounds increased and modified by consonance in the bronchial tubes. An insuperable objection, it seems to us, to the truth of Skoda's explanation is the fact that consonance either increases the intensity of obvious musical tones, or develops an obvious musical tone from unmusical sounds or from vibrations which are musical in rhythm, but of themselves too feeble for the ear distinctly to appreciate. But the tubular sounds heard over a pneumonic lung are no more musical than those heard over the trachea. An objection to the second explanation resides in the fact that, in the majority of cases in which tubular breathing is heard, the affected lung-tissue neither expands nor contracts during respiration, so that there can be no to-and-fro movement of air in its tubes to cause the sounds which may be heard over it. The first explanation appears to us to be substantially correct, for the following reasons:—the sounds of tubular breathing are like those produced at the rima glottidis during respiration; there is no doubt whatever that these, as well as all other sounds developed at this orifice, are readily conveyed, with little change of character, along the patent bronchial tubes, as along so many small stethoscopes, towards their peripheral distribution; the intensity of the tubular sound is proportionate, in great measure, to the intensity of the laryngeal sound, and indeed a distinctly tubular sound may, even in health, be developed and actually overpower the normal respiratory sounds when patients who are told to breathe deeply breathe noisily through the larynx. We are by no means prepared to deny that to-and-fro sounds, differing little from those originating in the larynx, may be produced by the to-and-fro movement of air in bronchial tubes connected with lung capable of respiration, and that such sounds may contribute in some cases to the collective result which we term tubular breathing. Whatever explanation be adopted, however, there is no doubt that the homogeneousness of texture which a consolidated or compressed lung presents allows, far more readily than normal spongy lung-tissue does, of the transmission of sounds which are developed within or conveyed into its substance; and further, that the total suppression of the healthy respiratory murmur, which characterises all those conditions of lung in which

tubular breathing is heard, contributes importantly to its ready recognition.

ii. *Amphoric, cavernous, or metallic breathing.*—These terms are employed to designate the peculiar quality of sound which may sometimes be heard over cavities containing air, and usually communicating with the external atmosphere by means of the bronchial tubes or other passages. It consists in a peculiar metallic ring, or musical twang, following upon the respiratory or other sound which calls it forth. A closely similar twang attends the footfall of a person walking between high walls, or over a vault, and may be recognised in perfection if a child's india-rubber ball be placed in contact with the ear and then sharply tapped or filliped. The addition to any other intrathoracic sound of the musical prolongation here referred to is always indicative of the presence of a cavity containing air; and it may sometimes be heard almost as distinctly in a cavity the size of a walnut as in one corresponding in capacity to the whole of the pleural sac. Its presence does not absolutely prove that there is communication between the cavity and the outer air, although in the great majority of cases such a communication does in fact exist; nor does it prove that the cavity to which it is due is an abnormal cavity, for it may, when detected at the lower part of the left side of the chest, be referrible to the stomach; nor, again, does it necessarily throw light on the form of the cavity or the structure of its walls, although, for the most part, we have reason to suspect when we hear it that the cavity or some part of it is of a rounded form, and that the walls are somewhat smooth and elastic, or, at all events, of such a character as to allow of reverberation. The cause of this amphoric resonance is obviously the reverberation, or succession of echoes, which occurs between the opposite sides of the cavity when any impulse or sound capable of originating it reaches the air in its interior. The chief conditions under which amphoric resonance manifests itself in connection with cavities are the following:—First, it attends the respiratory sounds, and more particularly that of inspiration. It is important, however, to observe that the respiratory sounds yielded from a cavity are, apart from the superadded resonance, tubular; and that if, from any circumstance, the musical twang be absent from them, there is nothing left by which they can be distinguished from ordinary tubular breathing. It is probably never produced in this case unless the cavity communicates with a bronchial tube or by a fistulous opening with the external air; and although it is probably not essential to its production that there shall be actual movement of air into and out of the cavity, there is no doubt that such movement tends largely to intensify it. Second, it attends both the sounds of vocalisation and those of coughing. Third, it may be evolved over large cavities by percussion of the thoracic walls which bound them, and especially if the percussion sound be sharp and short, as it may be made by employing two coins,—one as a plexímeter, the other as a hammer

(*bruit d'airain*). Fourth, it gives a metallic quality to the various râles or rattles which are produced in them or in their vicinity by the passage of air through fluid. It should be noted, however, that short sharp sounds like those of ordinary largish crepitation more readily induce an audible echo than do the duller less intense sounds of respiration and the like; and that hence crepitation often becomes metallic in small cavities, which give no such quality to respiratory, vocal, or tussive sounds, and sometimes even in the normal cavities of the bronchial tubes. Lastly, in large cavities we not unfrequently get that perfection of amphoric resonance which is termed '*metallic tinkling*'; a sound which is always most characteristically evolved in response to some sharp detonation, such as is produced by the bursting of a largish bubble or by the fall of a drop of fluid from above on to a surface of fluid below.

The cavernous echo, although in many cases remarkably distinct and unmistakable, is in some cases so feeble that it fails to be transmitted along the ordinary stethoscope, and can be detected only by aid of the binaural stethoscope or by the ear applied directly to the chest. Further, it may be, and often is, effectually concealed by the intervention between the cavity and the thoracic walls of a layer, however thin, of crepitant lung-tissue. And, again, it is important to know that cavities of considerable size, especially if there be no communication, or only imperfect communication between them and bronchial tubes, often yield no sound whatever due to themselves, and merely very feebly conduct tubular or even healthy respiratory sounds due to the lung-tissue in which they are imbedded.

There are yet one or two other sounds which may be developed within cavities, and may hence be included within the meaning of the term *cavernous respiration*. It is possible, for instance, that a cavity may be of such a size and shape as to be capable of resonating to some particular note; and that the production of that note by the patient in his larynx may be attended with special resonance within the cavity. And, again, it sometimes happens that when a cavity communicates, by a flap-like or valvular opening, with a bronchial tube, there is no sound audible over the cavity during ordinary respiration or during the early period of a forcible inspiration; but that during the course of the latter the air rushes into the cavity with an audible click, hiss, or gurgling sound—a phenomenon which is repeated whenever the patient inspires deeply.

iii. *Bronchophony, pectoriloquy, and ægophony.*—The terms pectoriloquy and bronchophony have been employed with great laxity, even by those who assume to be authorities upon the subject of auscultation. It has been frequently asserted that bronchophony as it becomes more marked passes into pectoriloquy, as though the two conditions were mere grades of the same phenomenon. This, however, is certainly not the fact; bronchophony never becomes converted into

pectoriloquy, although they are often associated; loud bronchophony, indeed, drowns the pectoriloquy with which it may be associated; and, in order to be certain of the existence of pectoriloquy, it is always best to eliminate the effects of bronchophony by making the patient speak in a whisper. As we have already pointed out, bronchophony is the offspring of laryngeal intonation, pectoriloquy of the articulate sounds developed within the cavity of the mouth.

Bronchophony, in its pathological sense, means preternatural distinctness, or loudness, with little alteration of quality, of the laryngeal musical tones as conveyed to the ear through the tissue of the lung. Its intensity, as well in disease as in health, presents considerable variations. Hence in determining the presence or absence of abnormal bronchophony we must not be content to note that the voice-resonance is louder in one part than another; but we must observe whether it is relatively loudest over those parts of a lung in which normally it is comparatively feeble; and especially we must be careful to compare the resonance of the voice in corresponding parts of the two sides of the chest. Bronchophony is generally developed over consolidated lung-tissue—pneumonic, tubercular, or other—and over the sites of vomicæ. And its development in abnormal situations is clearly due to the same combination of causes as that to which we have ascribed the phenomena of tubular breathing:—namely, first, the conduction of the musical vibrations along the patent bronchial tubes or tubes and cavities, into the very substance of the consolidated tissue; and, second, the ready transmission of these vibrations thence through this tissue to the surface of the chest. Skoda attributes bronchophony, as he does tubular sounds, to consonance of the laryngeal sounds within the bronchial tubes. We are far from denying that the tubes may consonate to musical sounds, or that they do so consonate in certain cases. But a tube of a certain definite length can only consonate to a certain definite note, and possibly to some of the higher harmonics of that note; and assuming (what seems scarcely possible) that the length of tube capable of consonating is to be measured from the rima glottidis to the terminal part of a bronchial tube at the base of the lung—a length of about twelve inches—the lowest note to which it (being a pipe closed at both ends) could consonate would be one produced by undulations a foot long, or one lying between B and C of the treble clef. There are good reasons for believing that the consonating note would be much higher. Now, if this explanation were true, the deeper tones of the voice, which are actually loudest in bronchophony, should be comparatively inaudible, and of acute tones one only, or one and some of its harmonics, should be conveyed to the ear. But this is certainly not the case.

Pectoriloquy implies the conveyance through the stethoscope placed on the chest of the articulate utterances of the person auscultated, as though he were applying his lips to the instrument and speaking

through it into the ear. We have pointed out that this phenomenon is always to be heard most distinctly when the patient whispers, because it is then uninterfered with by the noise of the laryngeal notes. There is another reason why it should then be most audible. Since articulate sounds are produced in the mouth, it is obvious that, in order to reach the bronchial tubes, they must pass the portals of the larynx. But in loud speaking these portals are closed, and must hence materially obstruct the transmission of such sounds; in whispering, on the other hand, they are to a greater or less degree patent, and the obstacle to their transmission is necessarily proportionately diminished. Pectoriloquy and bronchophony are not necessarily concurrent phenomena. Nevertheless, it is certain that pectoriloquy, like the other, is often detected both over consolidated lung-tissue and over cavities. We believe that it is most frequently and most distinctly audible over cavities which communicate freely with bronchial tubes.

Ægophony is a modification of bronchophony, and gradually passes into it. It is generally compared, as its name implies, with the bleating of a goat, or with the squeaking voice adopted by the exhibitors of 'Punch and Judy.' These comparisons are by no means inapt. The voice transmitted along the stethoscope differs materially in quality from the voice as it emanates from the patient's mouth; it is, even if musical and full-toned as uttered, tremulous, bleating, and high-pitched as it reaches the auscultator's ear. Some degree of this quality of sound may occasionally be recognised, even in health, over the lower part of the chest behind. But it is only heard in perfection in the neighbourhood of the lower angle of the scapula in cases of moderate pleuritic effusion; and indeed, when well marked, may be regarded as pathognomonic of this condition. It is obvious that the peculiar bleating high-pitched character is due, as Dr. Stone has pointed out, to imperfect transmission of the voice, to the fact that its graver tones are lost or greatly enfeebled in transmission, while the higher tones and the harmonics of the graver tones are comparatively unaffected. In support of this view may be mentioned the fact that the ægophonic sound, though apparently clearer, is often distinctly feebler than the normal voice-resonance to be heard over the healthy lung. Sound, as is well known, is readily transmitted through either gases, fluids, or solids, but it does not so readily pass from one of these media to the others; and it seems obvious therefore that the sounds produced within or carried into the bronchial tubes should experience some degree of filtration (so to speak) in passing from the tubes to the solid lung-tissue, from this to fluid, from this again to the thoracic parietes, and thence through the stethoscope to the ear. High notes are more penetrating than those of graver tone, and hence would be less likely to suffer in their passage.

In association with pectoriloquy, bronchophony, or ægophony there can generally be detected a distinct whiff of tubular quality, either

accompanying or following the articulate or vocal sounds. In bronchophony and ægophony this is perceptible almost exclusively at the end of syllables, and chiefly at the end of those terminating with the explosive consonants *b*, *p*, *d*, *t*, *k*, and hard *g*, and is obviously due to the non-vocal rush of air through the open glottis, which as a rule follows on the utterance of these sounds. In whispered pectoriloquy a similar whiff not only succeeds each syllable, but accompanies it during the whole period of its enunciation. These are merely tubular expiratory phenomena, due to the same cause as determines the ordinary tubular expiratory sound, and have no special significance. It should be added that, under similar circumstances, a like whiff or blowing sound follows each sonorous expiratory shock of cough.

iv. *Crepitation. Râles.*—When mucus, serum, blood, or other fluids are contained in the air-tubes, the passage of air through them is attended with a variety of sounds to which the above and other names have been given. These are for the most part due to the passage of air in the form of bubbles of various sizes, and to the rupture of these bubbles at the surface of the fluid through which they pass, or to the separation of sticky surfaces. The size of the bubbles necessarily has a relation to the size of the tubes or cavities in which they occur. Thus if they be formed in the air-cells or bronchial passages they must be excessively minute; if in the trachea or larger bronchi they are generally of considerable size. The sounds to which they give rise depend partly upon their size, partly upon their number, partly upon the dimensions of the channel or cavity within which they are contained, and partly on the presence or absence of consolidation in the lung-tissue around. *Fine crepitation* (*crepitant râle*) is produced only in the air-cells and bronchial passages, and may be regarded as almost characteristic of the first stage of pneumonia. It is apparently due to the rupture of innumerable small bubbles, which individually are almost inappreciable, but collectively constitute a sound which has been aptly likened to that produced by rubbing the hair between the finger and thumb. *Crepitation* (*mucous râle*).—In all forms of crepitation, except that just spoken of, the bubbles which burst at one time are comparatively few; moreover they are individually distinguishable, and differ to some extent from one another in sound. The collective sonorous result, therefore, is more or less coarse and irregular. In some cases two or three crackles or clicks only can be detected in the course of an inspiration or expiration. In other cases they are so numerous that the whole of inspiration and perhaps the whole of expiration are noisy with them. It would be impossible to describe all the minute varieties of crepitation which may be included under the name which we have here selected. It is sufficient to say that they are probably all due to the presence of fluid in medium-sized and large tubes, that the differences which are presented depend partly on the quantity of fluid present, partly on its quality, and partly on the force

with which air is driven through it, and that when the larger crepitation approaches that of pneumonia in quality, it is often termed '*sub-crepitation*' or '*sub-crepitant mucous râle.*' *Gurgling.*—This term fairly well explains itself, but is at the same time difficult to define. It implies partly large crepitation, such as may be heard in the trachea, partly the sounds which result from the mere agitation of fluid, falling, splashing, churning, and the like. It occurs in large tubes and cavities. *Metallic Crepitation.*—This term may be applied to large crepitation in which the bursting of the bubbles is attended with a distinct musical twang or metallic resonance. It is developed either in cavities or in large tubes. We have pointed out that cavernous or metallic respiration is never met with in undilated bronchial tubes; the sharp, short sound, however, of a bursting bubble develops an audible resonance under conditions which would fail to affect similarly the prolonged and comparatively feeble respiratory murmur.

v. *Rhonchus.*—This word is often used synonymously with râle, and both are often applied to all varieties of unnatural sounds caused by the presence of fluid in the bronchial tubes, or by diminution of their diameter. Râle, however, strictly means rattling or crepitation, which is essentially an unmusical sound; whereas rhonchus signifies snoring, a sound always to some extent musical, and may conveniently be made to embrace all abnormal musical sounds which are occasioned in the bronchial tubes. Such sounds have sometimes a deep tone, almost exactly like an ordinary snore, or the cooing of a dove; sometimes, on the other hand, they are high-pitched and of a whistling or hissing character. The deeper notes are usually termed '*sonorous,*' the acuter notes '*sibilant.*' The former, like the voice itself, may produce distinct fremitus in the thoracic parietics, and both may be distinctly audible, not only to the patient himself, but to the mere bystander. The cause of rhonchus is, not the bursting of bubbles or the passage of air through fluid, but the passage of air through a tube narrowed at some point either by thickening of its parietes or by the adhesion of a plug of tenacious mucus. The almost complete closure of the tube, like the corresponding closure of the glottis in intonation, compels the passage of the air in a series of successive puffs, which soon become rhythmical, and hence a musical note results. The pitch of the musical note depends on various complex conditions, the exact influence of each one of which it would be difficult to estimate, but is determined in a very considerable degree by the size of the bronchial tube within which it is developed. Thus, as a general rule, hissing and whistling sounds or sibilant rhonchi arise in the smaller tubes, and grave tones or sonorous rhonchi are the product of the larger ones.

vi. *Splashing.*—In large cavities containing air and limpid fluid, especially therefore in cases of effusion into the pleura, associated with pneumothorax, a distinct splashing sound may often be caused by the process termed '*succussion*;' in other words, by giving the patient a

smart shake. This sound is often audible to the patient himself as well as to other attentive listeners standing by. It may, of course, be more readily recognised by auscultation.

vii. *Amphoric bubble.*—In cases of hydro-pneumothorax may also be very rarely recognised a sound to which the name 'amphoric bubble' may perhaps be given. Our attention was first directed to it by Dr. T. A. Barker. On applying the stethoscope to the back in the interscapular region while the patient was sitting erect, and then making him gradually bend his trunk forwards, a sound, exactly like that which occurs during the decanting of wine, was distinctly audible. It was single only, but could be elicited as frequently as the patient was made to bend his body forwards to a certain angle. It was obviously due to the facts—that the partially-collapsed lung hung down from the apex of the pleural cavity so as to form an incomplete septum between its anterior and posterior parts; that the lower margin of the lung dipped into the pleural fluid, thus rendering the air-chamber behind the lung and that in front of it discontinuous; and that consequently, with change of posture, the level of the fluid tended to rise in one cavity and sink in the other, until the sudden passage of air from the one to the other was permitted under the septum.

viii. *Friction-sounds* are caused by the attrition of opposed pleural surfaces. They never occur in the healthy pleura, and it is essential to their production that the surfaces be roughened by inflammatory or other deposit. Further, as a rule, they have very little intensity, and are scarcely if at all audible beyond the spot at which they are developed. Friction-sounds present many varieties of character. In some cases there is a uniform to-and-fro murmur accompanying inspiration and expiration, and having a close resemblance to the sound produced by rubbing two surfaces of paper together. In some cases the sound differs little if at all from some forms of intra-pulmonary crepitation: there may be a continuous crackling attending one or both respiratory movements, or merely a few isolated clicks or crepitations. In a large number of cases the sounds, whether they be fine or coarse, occur in a series of irregular jerks. The jerks, indeed, may exist without the presence of actual friction sounds, in which circumstances the respirations become (over limited areæ) 'jerky,' or, as they are commonly called, 'wavy.' Friction-sounds have received various names, such as grazing, rubbing, creaking, and the like, which to some extent express their quality. They have also been described as 'superficial' in character. It need scarcely be remarked, however, that this epithet can have no other meaning, as applied to sounds, than that they are loud or distinct. Its use is altogether objectionable, as tending to cause confusion between the facts which we observe and the inferences we deduce from them. In cases of pleural friction, the rubbing of the opposed surfaces may produce a tremor in the thoracic walls, readily detectable by the hand. It may be observed

that loudness or roughness of friction sound by no means necessarily implies either roughness, hardness, or abundance of lymph. The loudest and coarsest sounds are occasionally produced by the thinnest, softest, and most recent films.

E. *Detection of Cavities, Consolidated Lung, and Pleural Effusion.*

Before leaving the subjects of auscultation and percussion it may be convenient to recapitulate the phenomena which attend and indicate the presence of cavities, of consolidated lung, and of pleural effusion.

1. *The detection of cavities* is often very important; and in a large number of cases, no doubt, by considering the patient's history, the results of periodical examinations of his chest, and the presence or absence of certain special acoustic phenomena, we may arrive at a fairly correct conclusion. But the acoustic phenomena which by their presence prove the existence of a cavity are, as Skoda asserts, very few indeed. Dulness, bruit de pot fêlé, normal resonance, tympanitic resonance, high-pitched resonance may each be present. Feebleness, with indeterminate character of the respiratory sounds, tubular sounds, gurgling, may also each be present in its turn. There is probably always more or less marked bronchophony and pectoriloquy. Pectoriloquy, indeed, is more distinct, as a rule, over cavities than over merely consolidated lung. The only sounds, however, which positively indicate the presence of a cavity, are: first, the musical or metallic ring or resonance which sometimes accompanies the respiratory sounds, the voice, the movements of fluid in the cavity, and the percussion stroke upon its walls; second, the splashing sound caused by succussion; and, third, the production of the amphoric bubble to which we have adverted. But these sounds may all be absent from cavities even of large size.

2. The *conditions which collectively indicate consolidation* are sense of resistance, impaired or annulled resonance, increase of vocal fremitus, tubular breathing, or correspondingly modified conditions of rhonchus or crepitation, bronchophony, and pectoriloquy. These conditions are, however, by no means necessarily all present in every case.

3. The *indications of pleural effusion* are dulness on percussion, with variation of the limits of dulness and resonance in accordance with variation of posture, tubular breathing, or more frequently extreme feebleness or absence of respiratory sound, impairment or suppression of vocal fremitus, and ægophony. To which may be added, dilatation of the affected side and intercostal spaces, with sometimes obvious fluctuation; and displacement of the diaphragm downwards and of the mediastinum to the opposite side. But, again, many of these phenomena are often absent from otherwise well-marked cases of effusion.

II. LARYNGITIS, AND TRACHEITIS.

Causation.—The chief cause of laryngeal and tracheal inflammation is exposure to cold or wet, or both. It is then sometimes the primary affection, but is often a mere extension of ordinary catarrh or of acute bronchitis. It may be due, however, to many other causes:—to the local operation of irritating gases, fluids, or solid particles, among which may be enumerated boiling water, vomited matters, and puriform secretions furnished by the lung itself; to the presence of certain morbid conditions or diseases, such as variola, measles, scarlet fever, diphtheria, erysipelas, and we may add syphilis and tuberculosis; to the extension of inflammation from subjacent tissues; and even, as regards the larynx, to sustained or violent exertion, as occurs in clergymen and other public speakers, and in those who strain themselves in coughing or shouting. It may be determined also by local violence. There are, further, many conditions which predispose to it; among the most important of which is the fact of having suffered from a previous attack, and the presence of Bright's disease.

Morbid anatomy.—The local changes which attend and indicate laryngitis are those of inflammation of mucous membrane generally, with modifications due to peculiarities of arrangement and structure which the laryngeal tissues present. The mucous membrane and subjacent parts are congested and œdematous; and the epithelial surface, at first (as in ordinary nasal catarrh) preternaturally dry, soon secretes, though not in large quantities, a glairy, transparent mucus, which subsequently becomes thick and muco-purulent. In ordinary mild cases the tumefaction and reddening are slight yet pretty uniformly diffused—the vocal cords being probably more or less injected and swollen, and studded with flakes of adherent mucus. In more severe cases the submucous tissue may be largely infiltrated and œdematous; and hence the affected regions often assume a translucent, almost jelly-like, aspect, though still presenting a congested surface. Such swelling, or œdema, may affect mainly the epiglottis, arytenoepiglottidean folds, false vocal cords, or some other limited tract, or may be general. It must be borne in mind, however that those parts the tissues of which are closest in texture suffer least in this respect, and that hence the free edge of the epiglottis and the true cords for the most part escape. In most cases the secretion from the mucous membrane presents simply the ordinary characters of mucus or mucopus. But in some (even in the absence of diphtheria) an adherent false membrane forms upon the surface. This sometimes follows the attempt to swallow boiling water. Ulceration is an unusual sequel of ordinary inflammation. It occurs most commonly, perhaps, in the course of phthisis and constitutional syphilis, even when no specific lesions are present.

Ulceration in phthisis may be the result of simple excoriation. It then begins with round or oval shallow, saucer-like depressions, of an ashy colour and with congested margins. Its most important, if not commonest, seat is the point of the processus vocalis. Ulcers in this situation incline to extend deeply, to expose more or less of the arytenoid cartilages, and to lead to their partial or total destruction by caries or necrosis. There is a great tendency, indeed, both in phthisis and in syphilis, for ulceration to involve the cartilages—arytenoid, cricoid, and thyroid—and to cause their erosion or necrosial destruction. But in some cases the cartilaginous affection takes its origin in inflammation of the perichondrium. For the most part the necrosed cartilages have undergone more or less complete ossification. The forms of laryngitis last referred to may be regarded as essentially of a chronic nature; but simple laryngitis also may become chronic. The anatomical characters of this variety differ but little from those of the acute affection. The chief distinctions are that, in the former case, the inflammatory redness is less intense, and the thickened tissues are more opaque and apparently more solid: they lose their peculiar œdematous character. When laryngitis becomes chronic the follicles of the affected surface often undergo hypertrophy. To such cases the name of '*glandular laryngitis*' has been given.

The changes which take place in tracheitis are essentially identical with those which characterise laryngitis. The surface, which is at first drier than natural, soon secretes an over-abundance of modified mucus, and occasionally, like that of the larynx, gets covered with an adherent pellicle. The mucous membrane itself, and the subjacent tissues, become congested and infiltrated; and not unfrequently, especially in syphilis and phthisis, ulceration takes places. The ulcers are mostly, in the first instance, mere excoriations, which tend gradually to increase in area and thus to coalesce, and in depth so as gradually to expose the cartilages. The latter may thus get eroded or necrosed, and even detached and expectorated. Abscesses may form in the walls of the trachea or external to them, and communications may be established between its tube and that of the œsophagus. The healing of ulcers whether in the larynx or trachea may produce serious cicatricial contraction.

Symptoms and progress.—1. *Acute laryngitis* is mainly dangerous from the fact that it is liable to cause serious obstruction to the passage of air through the rima glottidis, and hence death from suffocation. The inflammation is for the most part of little intensity, and gives rise to comparatively slight constitutional disturbance. There is usually during the earlier period of the affection more or less elevation of temperature, acceleration and hardness of pulse, flushing of the face, furring of the tongue, thirst, and loss of appetite. But in favourable cases these symptoms soon subside, and in unfavourable cases get replaced by those of asphyxia.

The special symptoms of laryngitis are often preceded by those of ordinary catarrh, and especially by those of catarrhal affection of the fauces, which, in many respects, they resemble. The patient complains of dryness or roughness, soreness, itching, pricking, or aching, or it may be of several or all of these sensations, which he refers to the back of the throat and to the region of the thyroid cartilage. There is generally also some tenderness to touch, and there may be absolute pain when the parts are roughly handled. The sense of soreness is aggravated by the act of swallowing, especially if solid matters be taken; and there is commonly also a good deal of aching thus caused besides soreness. The dryness and irritability of the throat compel the patient nevertheless to make constant efforts at deglutition, and at clearing the throat, and excite more or less frequent spasmodic attacks of cough. The voice gets altered in quality, and respiration somewhat impeded. Examination with the laryngoscope reveals congestion, with more or less thickening, of the mucous membrane; and if the parts above the vocal cords be much affected they may entirely conceal the rima glottidis and its surroundings from view.

Certain of the symptoms here enumerated require to be considered a little more in detail. Some degree of interference with the freedom of respiration is probably always experienced, and this, under the influence of excitement or sudden spasm, may readily amount to manifest dyspnœa; expiration is a little prolonged, and tends perhaps to be wheezy. But very often matters become much more serious; both inspiration and expiration (the former more especially), even when the patient is at rest, get harsh or whistling, noisy and prolonged, and he suffers from continuous difficulty of breathing. In cases of still greater severity all the symptoms of asphyxia become developed; the patient sits up in bed gasping for breath, which is still harsh, wheezy, or whistling; with his head thrown back, his mouth open, his nostrils dilated, his respiratory muscles acting with spasmodic force; anxious, restless, throwing his arms about, or clutching at any support which may be near; with eyes prominent and staring, face livid and ghastly, skin bathed in sweat, and pulse rapid, small, failing, and perhaps irregular. Under these circumstances death may occur suddenly from complete obstruction of the rima glottidis. But more commonly the patient begins to ramble, and presently passes into a condition of insensibility, upon which death gradually supervenes.

The voice is almost invariably altered in quality; it becomes hoarse, uncertain, or reduced to a whisper. In the beginning it is in general merely hoarse; it is somewhat rougher than natural, and at the same time deeper toned—phenomena which depend, either on the adhesion of mucus to the edges of the vocal cords, or on some modification in their thickness, elasticity or tension. This hoarseness is sometimes apparent only on rising in the morning, and disappears

during the day; it is apt, however, to be brought on again, and to be converted into actual aphonia, by unwonted use of the voice. At a later period of the disease, when the tissues above the vocal cords are highly œdematous, or the cords are much thickened and scarcely moveable, complete aphonia is usually present.

The cough varies in severity; sometimes it is incessant, or comes on in uncontrollable paroxysms. But it is generally attended with so much pain in the larynx that the patient endeavours (probably in vain) to suppress it. It is always at first, like the voice, hoarse and loud; and in many cases, especially in children, and where there is manifest dyspnœa, its inspiratory element is long, loud, and whistling, and the expiratory effort is attended with a remarkably harsh, sonorous, metallic clang. Later on, the cough, like the voice, becomes ineffective, wheezy, or aphonic.

Acute laryngitis is very apt to be attended or followed by bronchitis, or (especially in children) by collapse and lobular pneumonia—complications which aggravate the patient's symptoms and add very materially to his danger. It is sometimes as rapidly fatal as almost any disease with which we are acquainted; but in a large proportion of cases is so mild in its symptoms that but little attention is paid to it; yet it is always attended with risk, and should be carefully treated.

The frequency of the occurrence of laryngitis in a mild form is evidenced by the frequency with which persons, after exposure to cold, suffer from soreness referrible to the larynx, and hoarseness or loss of voice. This affection generally lasts for three or four days, subsides with increase of laryngeal secretion, and leaves no ill consequences behind. Dr. Cheyne asserts that hoarseness is an uncommon phenomena in the catarrhal affections of young children, and that its occurrence should make us dread the supervention of croup. Our own belief, on the other hand, is that hoarseness is not uncommon in children, and that it has no more serious import in them than in adults. The phenomena, however, of slight laryngitis in children under two or three years of age, and even in those who are a little older, are often so remarkable that they are confounded with those of *spasmodic croup* or *laryngismus stridulus*. The child, after having suffered from slight catarrhal symptoms, or sometimes in the midst of apparently good health, wakes suddenly during the night in an agony of dyspnœa. He starts up in bed with a look of extreme anxiety and terror, gasps for breath, inspires laboriously with a hissing or whistling sound, and coughs at intervals with a series of harsh, loud, metallic, expiratory shocks; his voice is hoarse or reduced to a whisper. After the symptoms have lasted half an hour or more, during which time the patient has been enduring all the horrors of impending suffocation, they subside, the skin gets moist, and he falls into a comfortable sleep. The next day he probably appears to be pretty well, although there may still be some hoarseness of voice and

the cough may still have a croupy character. It is not uncommon for such attacks to occur two or three nights or more in succession. There can be no doubt that they are mainly spasmodic; and there is some reason to suspect that they are often induced immediately by the entrance of saliva, or even of regurgitated food, during sleep, into the larynx. They are seldom fatal. Neither of the above forms of laryngitis, however, differs essentially from the rarer cases in which the symptoms early assume an aggravated character, and in which the patients die, suffocated, at periods varying from a few hours to two, three, or four days.

2. *Chronic laryngitis.*—Under the head of chronic laryngitis may be included: first, simple laryngitis, which has assumed a chronic form; second, aphonia clericorum; and third, ulcerative processes, connected especially with pulmonary tuberculosis and syphilis. In the first variety the symptoms differ but little from those of the acute affection, excepting in their comparative mildness. They are liable, however, to exacerbations, and rapid œdema of the submucous tissue may at any time ensue. Dr. Mackenzie states that in this form of chronic laryngitis the aryteno-epiglottidean folds are comparatively rarely congested and swollen, but that it is chiefly the false vocal cords, capitula Santorini, and epiglottis which suffer.

Aphonia clericorum may originate in catarrh, like other forms of laryngitis, or may be the result of simple over-exertion. It soon, however, and mainly in consequence of the persistent use of the voice, becomes a chronic affection. Its symptoms are like those of ordinary chronic laryngitis, but on the whole are more mild. The patient, indeed, often suffers from little except a sense of dryness in the throat, persistent hoarseness, and a tendency to hawk and clear the throat. Laryngoscopically, the appearances are those of chronic laryngitis. It is stated, however, that in this case there is a special tendency to hypertrophy of the laryngeal glands, and that their enlarged orifices may often be distinctly recognised.

The laryngeal affection which so commonly attends pulmonary phthisis creeps on insidiously, and is sometimes far advanced before the pulmonary disease has made very manifest progress. It differs from the varieties of chronic laryngitis above considered in its progressive aggravation and its incurability. At the beginning it presents no special symptoms; but as the disease goes on complete aphonia, dyspnœa which may be exceedingly severe, and pain and difficulty in swallowing, become established; indeed, in many cases swallowing becomes almost impossible, on account of the passage of food through the rima glottidis when the act is attempted. On laryngoscopic examination, the soft parts are seen to be more or less thickened, sometimes congested, sometimes pale, and for the most part opaque; and Dr. Mackenzie draws attention to the fact that the aryteno-epiglottidean folds usually look like 'two large, solid, pale, pyriform tumours,

the large ends being against each other in the middle line, and the small ones directed upwards and outwards.' The presence of ulcers may sometimes be recognised. Syphilitic affections of the larynx are not wholly specific. But, whether specific or not, the symptoms to which they give rise are those of progressive chronic laryngitis. In the later stages of constitutional syphilis extensive ulceration of the larynx is not uncommon, and in this case, as well as in so-called 'laryngeal phthisis,' there is a great tendency for caries or necrosis of the various cartilages to take place. Here, however, the epiglottis is most prone to suffer. Such complications, no matter what their cause, always largely diminish the ultimate prospect of even partial recovery, and bring in their train special symptoms in addition to those of simple laryngitis. Among these may be mentioned: infiltration and œdema of the tissues of the neck superficial to the laryngeal cartilages; fetid breath and purulent discharge; the occasional separation of portions of cartilage or bone, which may either be expectorated or cause sudden death by obstructing the laryngeal orifice; the formation of abscesses or sinuses which may open in various positions; the perforation of arteries, with profuse and fatal hemorrhage; and occasionally, as a sequela of the separation of sequestra and cicatrisation, permanent and serious contraction of the glottis or other parts of the laryngeal canal.

3. The symptoms due to tracheitis are scarcely distinguishable from those of inflammation of the larynx. It may be observed, however, that in inflammation limited to the trachea there is not necessarily any pain in the pharyngeal stage of deglutition, or any affection of the musical quality of the voice, and that, while the danger of suffocation is less, the benefit to be expected from tracheotomy is also less. Further, some tenderness in the course of the trachea may be expected, some pain in the same situation on coughing, and some tenderness or soreness in the passage of food along the œsophagus.

Treatment.—The treatment of laryngitis may be divided into the constitutional or general, and the local, of which the latter is by far the most important. The local treatment to the exterior of the larynx comprises leeches (which should be applied over the upper part of the sternum), blisters and other counter-irritants (which are also best applied in the same region); and poultices or hot fomentations over the larynx itself. For internal local treatment may be employed: the inhalation of steam, simple, or medicated with volatile aromatic or sedative substances such as turpentine, camphor, benzoin, creasote, or conium; the inhalation of atomised fluids such as solutions of sulphate of zinc or copper, acetate of lead, alum, nitrate of silver, perchloride of iron, or tannin; the application, by means of a sponge or brush, of strong solution of nitrate of silver (℥j, ad ℥j), tincture of perchloride of iron (℥j, ℥ij, ad ℥j), or any of the other articles just enumerated; the insufflation of finely-powdered astringents or sedatives; and scarifi-

cation of the congested or œdematous tissues. In order that the internal local treatment may be effectual, it is important that (excepting in the case of simple inhalation) the remedies should be applied by means of special apparatus under the guidance of the laryngoscope. The application of ice or cold compresses to the exterior of the larynx, and the sucking of ice, are measures which may often be adopted with advantage. As to general treatment, we must be governed mainly by the constitutional condition of the patient and by the character of his attack. In acute cases, ipecacuanha, tartar emetic, and other nauseating remedies have been largely advocated; opium, as in most inflammatory affections, especially such as are attended with pain or distress, is often of extreme value. Warm baths, and the retention of the patient in an equable, warm, moist atmosphere, are generally of use in the treatment of acute cases; in the treatment of chronic cases, iron and other tonics, cod-liver oil, change of air, and, if need be, iodide of potassium, or mercurial salts.

In the laryngitis which so often attends an ordinary catarrh it is advisable to keep the patient in a warm atmosphere, at any rate free from exposure to draughts, to apply hot fomentations or mustard plaisters externally, to order him to gargle his throat frequently with warm milk or with slightly astringent solutions, or to relieve his faucial discomfort by the use of gelatinous or oleaginous substances—among which may be included common calvesfoot jelly and black currant jelly—or to inhale steam. Diaphoresis may be encouraged, and expectorant medicines may be administered. Opium is of great value in relieving the patient's discomfort. When the case is severe from the beginning, or when it begins to assume a serious aspect, our local treatment must be more active: leeching externally, and scarification within, become then of essential importance. Sometimes in such cases swabbing the throat with strong solution of nitrate of silver, perchloride of iron, or alum, is followed by the best results. In the stridulous laryngitis of young children the danger is mainly momentary (so to speak), and due to spasm; and treatment, therefore, if it is to be efficacious, must be prompt. Generally it is advisable to place the patient in a hot bath, and to apply a sponge wrung out in hot water over the larynx. It is usually customary to administer an emetic dose of ipecacuanha or sulphate of zinc. It may, however, be questioned whether the inhalation of chloroform is not more likely to be beneficial than the use of an emetic. In the chronic form of laryngitis, local bleeding and scarification are rarely necessary except to relieve exacerbations; but blisters and other counter-irritants externally, and the systematic employment of medicated applications to the interior of the larynx, are then specially indicated. In the so-called 'aphonia clericorum' prolonged rest from the use of the voice should especially be enjoined. In all cases, whether they be acute or chronic, specific or non-specific, it must be borne in mind that we may

be called upon at any moment to save life by the performance of tracheotomy. The need for its performance must generally be determined at the moment. It is difficult to lay down precise rules for the guidance of the judgment of the medical attendant in such cases. It is probably sufficient to say that no one ought to be permitted to die of uncomplicated laryngeal obstruction without having that chance of recovery given him which tracheotomy affords; that it is unwise to delay the operation until the patient is moribund; that it is better to perform it too early or even needlessly than too late; and, lastly, that it should not necessarily be discarded even if the patient appears to be just dead.

III. BRONCHITIS.

Causation.—Inflammation of the bronchial tubes is dependent chiefly on exposure to cold. But it also arises, like laryngitis, from the inhalation of irritant matters; as a complication or sequela of various febrile disorders, such as influenza, hooping-cough, measles, and typhoid fever; and in connection with various idiopathic affections, more especially heart and kidney diseases. It may also be developed under the influence of pulmonary tuberculosis and carcinoma, and probably, too, in connection with syphilis and gout. Its prevalence depends largely upon temperature and season, and hence it is chiefly fatal in autumn and winter; it is favoured by such occupations as expose persons to the influence of irritant or other noxious matters, and such as necessitate frequent and sudden exposure to variations of temperature; it affects persons of all ages and of either sex, but it has a marked preference for such as have had previous attacks, and is especially fatal in early infancy and in old age.

Morbid anatomy.—Inflammation of the bronchial tubes, like inflammation affecting other mucous membranes, is attended with changes in their epithelial covering and glandular secretions, and in the subjacent tissues.

The discharge is, in the first instance, diminished in quantity, but soon becomes more abundant than in health, thin, transparent, and either watery or viscid, and subsequently acquires more or less opacity and thickness, and a yellowish or greenish tint. Sometimes it remains watery, sometimes assumes the characters of pus, and not unfrequently, if the inflammation be intense or the congestion great, presents streaks and spots of blood. Under the microscope the viscid transparent secretion presents abundance of shed ciliated epithelial and other cells; and the acquisition of opacity is connected with the more or less complete replacement of these by cells of embryonic character, fatty or granule cells, and pus corpuscles. In some rare cases groups

of bronchial tubes are found occupied by laminated fibrinous casts, which on separation present a branching or tree-like aspect.

The mucous membrane becomes congested, sometimes intensely congested, and the seat of minute extravasations of blood; at the same time it undergoes more or less infiltration and thickening, and may even acquire a granular or villous aspect, and a soft or pulpy consistence. It is important, however, to know that, in a large number of cases, especially chronic cases, the congestion disappears wholly after death, and the mucous membrane seems scarcely changed either in thickness or in texture.

The inflammatory process is limited to the surface of the mucous membrane; but it often extends to and involves the whole thickness of the bronchial walls, leading also to more or less serious infiltration and induration of the connective tissue which surrounds them. In the last case the muscular fibres may either, if merely irritated, be stimulated to unwonted action, or undergo atrophy or degeneration, and lose their contractile properties. In most cases of bronchitis the mucous membrane remains whole; but occasionally ulceration takes place. This is more common in phthisis than in the uncomplicated disease, and usually commences, as does tracheal or laryngeal ulceration, in simple excoriation. The excoriations, at first small and round or oval, gradually enlarge and coalesce, and at the same time tend to increase in depth. Thus the walls may undergo gradual removal (the cartilages disappearing either by caries or necrosis), the surrounding lung-tissue share to a greater or less extent in the destructive processes, and the tubes be converted into irregular channels bounded by diseased lung-tissue. In some cases gangrene occurs. Ulcerative destruction occasionally takes place from without, as when a pulmonary, glandular, or other abscess opens into an adjoining tube. It is thus that abscesses about the roots of the lungs discharge themselves into the bronchi, and that calcareous matter from diseased bronchial glands finds its way into these or smaller tubes.

Bronchitis is limited, in a large proportion of cases, to the tubes of large and medium size; but sometimes affects mainly or entirely the minuter tubes. In the latter case, not only is the affection marked by greater intensity of symptoms and aggravated danger to life, but the local pathological changes assume a more serious character; the thickening of the mucous membrane encroaches more seriously on the channels of the affected tubes, and their secretions tend to accumulate in them and to block them up completely. Hence post mortem we not unfrequently find the smaller tubes distended with pus or mucus, void of air, and quite impermeable.

The indirect influence of bronchitis over the structural condition of the bronchial tubes and of the proper tissues of the lungs is very remarkable. As regards the tubes, we have already pointed out that,

by extension of ulceration, they may be converted into irregular channels; this change, it need scarcely be said, may be seen in its greatest perfection in connection with the capillary or terminal tubules. But, independently of ulceration, the tubes, and especially the smaller ones, may undergo considerable dilatation from the combined effects of simple accumulation of contents and inflammatory weakening of their walls. In acute bronchitis, attended with much secretion, the lung-tissue often becomes preternaturally distended with air, and retains the accumulated air even after death; this condition is sometimes incorrectly termed emphysema; but it not unfrequently proceeds to actual emphysema, in which the vesicular structure is more or less seriously disorganised. Beside over-distension, the exactly opposite condition of pulmonary collapse is often met with, sometimes alone, sometimes associated with over-distension of other parts; collapse is intimately related to another frequent complication of bronchitis, and indeed passes by insensible gradations into it; we mean lobular pneumonia. All the secondary phenomena arising in the progress of bronchitis, which have here been enumerated—namely, dilatation and destruction of tubes, dilatation and destruction of air-cells or emphysema, lobular collapse and lobular pneumonia—form a more or less important part of chronic bronchitis, and tend both to aggravate its symptoms and to perpetuate them. It will, nevertheless, be more convenient to defer their complete discussion.

Symptoms and progress.—The symptoms of bronchitis comprise, in varying proportions, those of inflammatory fever, those of defective aëration of blood, and those directly referrible to the condition of the bronchial tubes and lungs; to which may be added those arising from mechanical impediment to the transmission of blood through these organs.

The symptoms of inflammatory fever are always most pronounced at the commencement of acute attacks and of exacerbations of the chronic affection, and often disappear wholly, to be replaced by other conditions, during the progress of the disease. The temperature, excepting in very severe cases, especially of capillary bronchitis, and in young children, rarely exceeds 100° or 101°. In exceptional cases it may mount to 102°, 103°, or 104°. With elevation of temperature there may at first be chills or rigors, and dryness of skin. But perspirations, more or less profuse, are very apt to alternate with dryness, or to replace it. The pulse becomes accelerated, the respirations somewhat hurried, the tongue furred; the patient has thirst, loss of appetite, constipation, and scanty turbid urine; he probably complains of headache and febrile pains in his limbs; and he is apt to be drowsy, though often wakeful at night.

Diminished aëration of the blood tends to the reduction of temperature, to interference with the processes of nutrition, and to enfeeblement of the heart's action and of the pulse. The temperature

of bronchitis may hence be subnormal even in acute attacks. The pulse, moreover, is sometimes full and incompressible, owing either to increase of arterial tension secondary to venous obstruction, or to poisoning of the nervous centres; and in chronic cases it is often abnormally slow. The face, and especially the lips and cheeks, assume a pale or livid hue; profuse perspirations break out; and there is a tendency to impairment of the mental faculties, to delirium, and coma.

The local symptoms are due to the processes going on in the bronchial tubes. They comprise cough, at first dry and irritable, later on freer and attended with expectoration; difficulty of breathing, with increase in the number of respirations and in the efforts required of the patient; comparative prolongation of the acts of expiration; and the various forms of rhonchus and crepitation, which are caused by thickening of the bronchial mucous membrane, or secretion into the tubes.

1. *Acute bronchitis.*—The symptoms of bronchitis vary considerably according to its severity and the conditions which cause or complicate it. In its mildest form it is a comparatively trivial affection. It then usually commences with ordinary catarrhal inflammation of the upper part of the respiratory tract, which gradually travels downwards, involving first the larynx, and then the bronchial tubes. It is attended with febrile disturbance, irritability of the bronchial mucous membrane, tickling or uneasy sensations in the throat, burning, soreness or rawness within the chest, and more or less frequent cough, the paroxysms of which cause considerable aggravation of the intra-thoracic discomfort. There is frequently, also, some tenderness over the manubrium, with tenderness and aching of the muscles of the upper part of the front of the chest. The cough is at first dry, but in a short time becomes loose and attended with the discharge of transparent glairy mucus. With the progress of the case the sputa get opaque and muco-purulent, then gradually cease, and health is restored at the end of a few days, or at most after the lapse of a week or two.

In more severe cases, the symptoms are the same in kind, but aggravated. The febrile phenomena which usher in the attack are more intense, the cough and pain in the chest are more distressing, and there is more or less obvious dyspnœa. There may indeed, while the mucous membrane is simply swollen, and the cough is yet dry, be great apnœal distress and lividity of surface, and the patient may even at this stage die asphyxiated. More commonly however, here as in the former case, the mucous surface ere long begins to discharge, and the cough to be attended with expectoration, which, except that it is probably much more profuse and apt to be streaked with blood, passes through the ordinary phases. During this period, also, death may take place from accumulation of fluid in the bronchial tubes and consequent slow asphyxia; or, without the actual supervention of

asphyxia, the patient may gradually pass into a typhoid state, with feeble, quick, irregular pulse, dry cough, copious sweats and delirium; or he may sink from a combination of these conditions. Occasionally death is sudden, owing to the sudden obstruction of some of the larger tubes.

The most dangerous form of acute bronchitis is that which commonly goes by the name of 'capillary bronchitis.' It is that form in which the inflammation affects mainly, if not exclusively, the minuter bronchial tubes. It is most common in children, yet not unfrequent in persons of more advanced age. The fever which ushers it in is generally pretty intense, the difficulty of breathing and lividity are considerable; the cough, however, may be much less troublesome than in other cases, and even during the stage of secretion may, owing to the difficulty of dislodging accumulations in the minuter tubes, remain inefficacious and dry. Further, there is generally comparatively little intrathoracic pain even in violent coughing. The tendency in capillary bronchitis is to speedy death from asphyxia and debility.

The auscultatory phenomena of bronchitis comprise mainly sonorous and sibilant rhonchi, and crepitation of various sizes. Musical rhonchi are chiefly heard during the dry stage, crepitation during the later stages, but even then musical sounds are apt to be present to a greater or less extent. In capillary bronchitis the rhonchus is mostly sibilant, and the crepitation small. The sounds elicited by percussion differ little from those of health. If the lung-tissue be much distended with air, as it often is, the percussion sound may be somewhat more resonant than normal; but obvious dulness is rarely produced, even if there be lobular collapse, unless the collapse be extensive, or unless pneumonia or other complications be present.

2. *Chronic bronchitis.*—Bronchitis often assumes a chronic form, especially among the labouring classes, and in middle or advanced life. It may become chronic, however, at all ages, and in persons of any grade of society. When a patient suffering from acute bronchitis continues to expose himself to the conditions which caused it, the inflammation is likely to be kept up; and, again, bronchitis is one of those affections which, when once they have been experienced and cured, tend to recur on the slightest provocation. The ordinary history of a case of chronic bronchitis is to the effect that the patient, after exposure to weather, probably during the winter, has an attack of the disease, from which he recovers during the ensuing spring, remaining fairly well until the approach of the following winter; that then a fresh attack is contracted, from which again recovery takes place; that these attacks of winter cough then recur annually, gradually increasing in severity and duration, and being separated from one another by shorter and shorter intervals of comparatively good health; and that each such successive interval becomes a period of increasing shortness of breath, until it merges in that of the bronchitic condition, which thus becomes continuous, although still probably

presenting winter exacerbations. Each bronchitic attack differs but little in its symptoms from an ordinary acute seizure, excepting perhaps that it is rarely attended with such manifest febrile disturbance, and that the expectoration is apt speedily to assume the muco-purulent condition and to continue of this character, and at the same time to become more or less abundant, until the approach of the long-delayed convalescence.

The successive long-continued attacks generally lead gradually but surely to those structural pulmonary changes which have been already enumerated, and to those various remote lesions referrible to long-continued congestion of the systemic venouss ystem which follow equally on this disease and on cardiac affections: the mucous membrane tends to secrete more abundantly than natural, even when the patient is otherwise apparently well; emphysema, or dilatation of the tubes, or both of these conditions, gradually supervene; the right side of the heart becomes dilated and hypertrophied; and systemic venous congestion ensues, in which the liver and kidneys especially share. The symptoms due to these lesions are consequently added one after another to those of simple bronchitis; the patient soon begins to suffer from persistent shortness of breath and bronchial accumulation, and sooner or later gets cyanotic or anasarcous and the subject, may be, of jaundice or albuminuria.

The thorax of a patient who has suffered long from chronic bronchitis gradually assumes, in consequence partly of his persistent powerful inspiratory efforts, partly of emphysema, a rounded form—the well-known barrel shape which is so common in this affection.

Cases of chronic bronchitis, within certain limits, differ widely from one another in their severity and in the symptoms with which they are attended. We may perhaps mention that in some the bronchial secretion is so scanty, other symptoms being well developed, that the affection has been termed '*dry bronchitis*;' that, in some the discharge is so profuse, that the name '*bronchorrhœa*' has been given to the malady; and that, in other cases, even where no gangrenous condition is present, the expectoration is disgustingly fetid—a condition which is said to be chiefly met with when there is dilatation of the bronchial tubes. The expectoration and the auscultatory, percussive, and tactile phenomena yielded by persons suffering from chronic bronchitis present no material differences from those presented by patients suffering from the acute disorder, and call, therefore, for no special description. Death, in which, sooner or later, the chronic disease so often terminates, is usually due either to asphyxia, to asthenia, or to a combination of these conditions.

The expectoration of laminated casts of the bronchial tubes is an event which may naturally be looked for in cases of diphtheria in which the diphtheritic process has travelled from the larynx into the trachea and thence downwards. And, indeed, since the diphtheritic pellicle

may form upon any part of any mucous membrane, there is little doubt that it occasionally forms in the smaller bronchial tubes independently of any such affection of the larynx, trachea, or bronchi, and that equally under these circumstances, expectoration of casts may take place. But occasionally such casts are spat up from time to time by patients, whom there is no reason to suspect of diphtheria. The causes, pathology, and symptoms of this affection, which has been termed '*plastic bronchitis*,' are alike obscure. All that is positively known is: that persons, after a longer or shorter period of ill-health, and symptoms something like those of slight chronic bronchitis or lobular pneumonia, expectorate either without warning or after prolonged dyspnœa, and as the result of a more or less suffocative paroxysm of cough, a larger or smaller quantity of this material, often in connection with hæmoptysis, which may be profuse, or with muco-purulent discharge; that this plastic expectoration may then cease or may continue off and on for an indefinite period; and that, although some of these patients die ultimately of phthisis, and some of the accidents which attend the process of expectoration, the majority appear to make a good and permanent recovery. There is good reason to believe that the portions of lung-tissue to which the obstructed tubes lead are in a state of more or less complete collapse or lobular pneumonia; and indeed, although in most cases there appears to have been perfect pulmonary resonance with more or less rhonchus and crepitation, a few have been recorded in which, as might be expected, there was circumscribed dulness, with total absence of respiratory murmur over the dull area. The co-existence, however, of pulmonary and bronchial lesions does not explain the nature of the relation between them. There is no doubt that, in hæmoptysis, blood occasionally coagulates in the bronchial tubes, and that in pneumonia bronchial casts of the same material as fills up the air-cells are now and then produced, but these seem to be quite distinct from the casts of plastic bronchitis, which probably originate *in situ*.

Treatment.—Bronchitis is one of the commonest diseases of temperate climates, one of the most frequent sources of incapacity for useful work and the enjoyment of life, and one of the most fruitful causes of death. Its treatment is therefore a matter of grave importance. It will be convenient to discuss it under different heads. *Hygienic treatment.*—This comprises the keeping of the patient in an equable and moderate temperature, not below 65° or 66°, and if possible not very largely exceeding this, and preferably, therefore, confining him to the house or even to one room; the maintenance of some degree of moisture of atmosphere; the use of hot baths, the Turkish bath, or the hot pediluvium; and the regulation of the diet according to the patient's capabilities and needs. *Local treatment.*—Under this head may be included: first, treatment applied to the skin, inclusive of counter-irritation by mustard plaisters, blisters, and the like, dry-

cupping, and the abstraction of blood either by leeches or cupping-glasses; second, treatment applied to the mucous membrane, such as the inhalation of steam, either simple or medicated with some of those substances which have been enumerated in the treatment of laryngitis.

Medicinal treatment.—The drugs which have been employed are various. Among expectorant or nauseating medicines, ipecacuanha, squills and tartar emetic hold a high place; stimulant drugs, such as the gum resins and balsams, more particularly benzoin, tolu, guaiacum, and ammoniacum, are often valuable; and as closely related in action to these may be enumerated ammonia, senega, and the stimulant vegetable tonics. Sedatives and narcotics, such as opium, conium, belladonna and hyoscyamus are of great importance; and in certain stages and in certain cases so also are sulphuric ether and lobelia. Lastly, tonics and alcoholic stimulants are often, and especially in the later stages of the acute affection and in chronic cases, of extreme value.

In ordinary mild bronchitis, little or nothing is needed beyond keeping the patient in a warm room, the inhalation of steam, the application to the chest of a mustard plaister, the use of the hot bath or pediluvium, the exhibition of small quantities of opium and ipecacuanha, and the relief of thirst and dryness of mouth by warm diluent drinks.

In acute cases of greater severity, it may be necessary to abstract blood from the surface of the chest. This can only be needed when there is extreme difficulty and pain in breathing, especially if at the same time there is reason to believe that the bronchial membrane is congested and swollen, and yielding but little secretion. The quantity of blood to be removed must be determined by the age and state of the patient, and by the effect of its removal. It is much better, however, to withdraw an adequate quantity at first than to repeat the operation over and over again. In such cases, too, counter-irritants and inhalation are of great value. As regards medicines, antimony or ipecacuanha in nauseating doses, combined, it may be, with squills, and above all with small doses of opium, and frequently administered, is generally useful. When the bronchial secretion becomes abundant and muco-purulent, these may still be continued, or may be replaced by the more stimulating forms of expectorant medicines. In this stage the combination of drugs recommended by Dr. Stokes, namely, ammonia, opium, and senega, is often of much service, as also are the balsams or gum-resins. When the patient suffers much from bronchial accumulation, an occasional emetic dose of ipecacuanha may be resorted to with benefit. Under similar circumstances, the persistent use of tartar emetic, in pretty large doses, associated with alcoholic stimulants, is frequently of great value. In protracted cases, and during convalescence, tonics are called for, and good nutritious diet. Few drugs are more valuable than opium in the treatment of bronchitis; it relieves pain and distress,

diminishes the irritability of the mucous membrane and the need for coughing, and probably also tends to reduce inflammation. At the same time its administration is often fraught with danger. It is generally best to give it in frequent small doses; and it is well to give it very cautiously or to withhold it entirely when the patient shows signs of imperfect aëration of blood, when his bronchial tubes are overloaded with mucus, or when he tends to ramble.

In chronic bronchitis, especially when exacerbations are present, the treatment must in the main be the same as that of the acute affection. On the whole, however, the abstraction of blood, and the use of medicines calculated to depress the patient's strength are not desirable. Counter-irritants, inhalation, stimulant medicines, tonics, and good diet are chiefly indicated. It is in these cases, too, that hygienic treatment is especially likely to be serviceable. The patient who is subject to a winter cough, increasing year by year in severity, and in whom emphysema and other such lesions are in progress, should dress warmly even in summer, should be careful not to expose himself to draughts or to the evening or early morning air, should give up those pursuits which expose him to the causes of bronchitis, and should pass his winters on the South Coast, or on the shores of the Mediterranean, or in some other warm equable climate, or else confine himself to a room or suite of rooms, well-ventilated, but kept at a uniform and comfortably warm temperature.

IV. PNEUMONIA.

Causation.—Inflammation of the substance of the lungs, like bronchitis, is due in the large majority of cases to the influence of cold and wet; and it would seem that it may, under special circumstances, be caused either by brief exposure of portions of the heated surface of the body to a severe chill, or by prolonged exposure of the whole normally warm surface to comparatively slight degrees of cold. It is especially common in temperate climates, and at those seasons (spring more especially) when the temperature is liable to great variations. It may also be caused by the spread of inflammation (whether originally due to cold or not) from other parts: as from the bronchial tubes, in cases of bronchitis, hooping-cough, measles, influenza, diphtheria, and the like; from the pleura in cases of pleuritis; or, if the pleural cavity be obliterated by adhesions, from the chest-walls or surrounding viscera. And again, it may be developed by the direct action of mechanical and other irritation, such as follows the inhalation of irritant gases, particles of dust or other such substances, solid bodies of larger size, vomited matters, or even water; or it may spring from the presence

of emboli in the branches of the pulmonary artery, or of tubercles or clots in the tissue of the lungs.

There are also many pathological conditions—especially the presence of pulmonary congestion or œdema, or of specific poisons or effete matters in the blood—which favour the occurrence of pneumonia. And it is probably due to one or other or all of them that pneumonia is so common in the course of heart disease, kidney disease, various infectious fevers, erysipelas, rheumatism, and many other inflammatory disorders. It is also very apt to occur in persons advanced in syphilis, or worn out whether by disease or over-work.

It must not be forgotten, however, that acute idiopathic pneumonia occurs with considerable frequency amongst those who seem to be in the best of health. This variety of the disease is met with at all ages and in both sexes; but it is more common in men than in women, and far more common among the working classes than others—facts which are explicable by the relatively greater exposure to the causes of pneumonia of those who have to earn their livelihood by the sweat of the brow. A previous attack seems to predispose to subsequent attacks.

Morbid anatomy.—It will be convenient, in describing the morbid anatomy of pneumonia, to distinguish, as has generally been done, two forms—namely *lobar* and *lobular* pneumonia, or, as they are termed by German writers, *croupous* and *catarrhal*. These names are none of them unobjectionable, and it might be better to replace them by the words *diffused* and *patchy*; the type of the former variety being furnished by the idiopathic affection, that of the latter by the condition which is secondary to diseases of the air-passages. The two varieties, however, pass into one another.

A. *Lobar pneumonia* begins with hyperæmia of the small vessels which are distributed in the walls of the air-cells and bronchial passages; swelling and tendency to proliferation of the epithelial cells of these parts; and exudation of inflammatory lymph (serum, albumen, fibrine), and of the corpuscular elements of the blood. The air-vesicles and passages communicating with them gradually become filled and finally distended with exuded matter, the air which they contained by degrees gets expelled, and the affected lung-tissue grows solid and heavy. If the parts be now examined microscopically, the dilated blood-vessels will be found to be crowded with their corpuscular contents, and the alveoli full of cells—some merely modified epithelial cells, with one, two or more nuclei, some cells undergoing fatty change (in other words, granule-cells), and others having the characters of leucocytes or pus-corpuscles—all blended together into a common mass either by an amorphous glutinous cement, or by a delicate fibrillated network. The ordinary process of inflammatory cell-proliferation has taken place, by means of which the epithelial cells have acquired a more or less distinct embryonic character; and to these, escaped leucocytes have been added. With the progress of the disease the con-

tents of the air-vesicles liquefy, and acquire more and more both the naked eye and the microscopic characters of pus. The fatty degeneration which has been referred to may, either before or after the liquefaction of the contents of the air-vesicles, become general throughout the accumulated cells, which may then, if not expectorated, undergo gradual absorption. The conversion of the inflammatory exudation into pus is occasionally followed by the breaking down of the lung-tissue here and there into abscesses; and occasionally by the occurrence of gangrene. It may be added that inflammation of the lung, like inflammation of other parts, rarely if ever takes place without there being more or less abundant serous exudation into the surrounding uninflamed tissues; and, further, that pneumonic inflammation tends, like most inflammations, to spread.

The progress of pneumonia through its various phases is quite gradual; nevertheless, there are at least three stages which severally present more or less obvious characteristic features. The first of these is the stage of *engorgement*, the second that of *red hepatisation*, and the third that of *grey hepatisation*. In the first stage the lung still contains air, though in diminished quantity; it is deeply congested, exudes more moisture than natural, is increased in weight, and is more easily lacerable than healthy lung-tissue. This is the period of congestion and commencing proliferation; and at this time the condition of the lung is scarcely, if at all, distinguishable from that of simple hypostatic congestion. In the second stage the lung is consolidated; it has lost its air, and its cavities are filled with coherent masses of cells; it is distended to its full size, and its constituent lobules are distinctly mapped out upon the surface; on section it appears to be pretty dry and slightly granular (a condition still more noticeable on the surface produced by laceration); and it presents a peculiar marbled aspect, which is due to the intermixture of nearly colourless inflammatory deposit, patches of congestion, and the irregular slate-coloured or black tracts which commonly stud the lung-tissue of persons who have reached adult age. The general hue of the lung is for the most part somewhat pale; there is probably, however, more decided congestion during life, and even after death the tissue is in some cases almost as deep in hue as we find it in pulmonary apoplexy. Sometimes, indeed, there is actual extravasation of blood. The lung-tissue is easily torn, and readily sinks in water. The third stage differs from the second, mainly in the assumption by the affected lung-tissue of a pretty uniform opaque greyish, yellowish or greenish tinge, in its largely increased friability, and in the ready exudation from the cut surface of thick, turbid, purulent fluid. In some cases the fluid is comparatively scanty; in some it is so abundant that the lung is like a sponge saturated with pus.

We have already mentioned the fact that there is generally, if not always, considerable œdema of the lung-tissue beyond the part actually inflamed. We may add that there is almost invariably a deposit of

inflammatory lymph on the surface of the inflamed portion of lung, as well as upon the parietal pleura in contact with it, and that this tends to diffuse itself over the serous membrane, more especially towards its base, but is not generally attended with any large amount of serous effusion.

Since pneumonia tends to spread, it naturally follows that different portions of affected lung often present well-marked differences of condition, and that we occasionally find all the recognised stages of pneumonia present at the same time in the same case. Inflammation may involve the lung to very various extents: thus it may be limited to a patch no larger than a walnut, or may include an entire lobe or even a whole lung; and, further, it may affect both lungs. It is curious how often it is strictly limited by the fissures or fibrous septa which separate lobes, and how often it is accurately mapped out by the margins of lobules. As regards position, it seems to be a well-established fact that the right lung is more frequently affected than the left, and the lower lobe than the upper. In reference to the latter point, however, it may be observed, that if we divide the lung horizontally midway between apex and base, there will be at least some two or three times as much lung-tissue below as there is above the plane of division, and that hence, if all parts of the lung be equally liable to inflame, inflammation of the upper part should be several times less frequent than inflammation of the lower part. The forms of pneumonia which supervene on hypostatic congestion, or come on in the course of renal and cardiac disease, or complicate pulmonary apoplexy and tubercle, differ little anatomically from that which has been here described.

B. *Lobular pneumonia* is especially the pneumonia of young children; it is not unfrequent, however, in older persons. In its best marked form the lung is studded with pneumonic patches, varying in size from about that of a pea to that of a filbert, and involving each one or more pulmonary lobules, circumscribed by the interlobular septa, and separated from one another by a network of still crepitant, and it may be perfectly healthy, lung-tissue. The pneumonic patches may be in the condition of engorgement simply, in which case their character may possibly fail to be recognised; or they may present the ordinary features of red or grey hepatisation. Further, by extension of disease, neighbouring patches may coalesce, and thus extensive tracts of lung-tissue become involved. Lobular and lobar pneumonia here pass into one another. True lobular pneumonia is always secondary to the blocking up of air-passages, and especially those of capillary size; and it may be excited immediately either by the gradual extension of the inflammatory process from the tubes to the air-vesicles, or by the entrance into the vesicles during inspiration of inflammatory products of the tubes which then act as irritants. But, whatever the cause, we find in the inflamed parts not merely overgrown and modified epithelial cells, but also, according to the stage of the disease, granular and embryonic cells

in greater or less proportion. The connection of lobular pneumonia with obstruction of tubes is further shown by the facts, that lobular collapse is often associated with it, and that then the collapsed and pneumonic conditions may often be seen to pass into one another by gentle gradations.

Closely related to lobular pneumonia is the disseminated pneumonia due to obstruction of small branches of the pulmonary artery, either by embolism or thrombosis, or in the course of pyæmia. In these cases, as in the other, the affected patches are usually of small size, and limited by the margins of lobules. But there is greater variety of result, especially in pyæmia; in which, while the patches sometimes present simple engorgement, or red or grey hepatisation, they not unfrequently are the seat of hemorrhage, or undergo rapid suppuration or gangrene. Lobular pneumonia is generally best marked towards the basal portions of the lungs, and the superficial patches are often the centres of areæ of pleural exudation.

In all forms of pneumonia, even in such as are not of bronchitic origin, there is a tendency to the development, sooner or later, of more or less bronchitis. But, apart from this, there is a marked disposition early in the course of pneumonia, to the effusion into the tubes from the inflamed air-cells of a transparent, very viscid fluid, uniformly stained with blood, and containing corpuscular elements; and, in some rare cases, this effusion, like that in the air-cells, whence it is derived, undergoes coagulation in the bronchial tubes, which thus become filled to a greater or less extent with casts consisting of coagulated fibrine and corpuscles.

Notwithstanding the frequency with which pneumonia proves fatal, it does not very often go beyond the third of the stages which we have described; sometimes, however, abscesses form, sometimes gangrene takes place, and sometimes the pneumonia lapses into a chronic condition. Pneumonic abscesses are usually of small size and irregular form; and in some cases, especially when they are developed in connection with lobular pneumonia, the terminal bronchial tubules are primarily affected, their parietes become destroyed, and the abscesses taking their course assume a dendritic character. Gangrene very seldom occurs in simple idiopathic pneumonia; it is chiefly met with in those cases in which the pneumonia is secondary to or complicated with some other affection. It is characterised by the breaking down of the lung-tissue into a fetid dirty greenish-yellow pulp, and by more or less greenish discoloration of the consolidated tissues around. Not unfrequently the latter are œdematous and present a slightly translucent aspect. The gangrenous condition may involve either an extensive tract of lung-tissue or several scattered patches, or even a single small patch. If it be recent at the time of post-mortem examination its margins will be found ill-defined; if it have existed for some length of

time the gangrenous cavity will probably be bounded by a well-defined edge. Of chronic pneumonia we shall speak at length hereafter.

Of the associated morbid phenomena of pneumonia there are several that call for mention, if not for detailed description. We have adverted to the co-existence with it of pleurisy and bronchitis; but besides these, we frequently observe, an herpetic eruption on, or in the neighbourhood of, the lips; more or less jaundice without obvious hepatic disease; intestinal congestion, with sometimes membranous patches on the mucous surface of the large intestine; and inflammation of the bronchial glands. Further, the conditions which give rise to pneumonia occasionally give rise at the same time to inflammation of other organs. Thus accompanying pneumonia we sometimes find inflammation of the brain, kidneys, bowels, or pericardium. It is common, after death, for the right side of the heart to be full of fibrinous coagulum which is prolonged into the pulmonary artery, while the left side of the heart is contracted and almost empty.

Symptoms and progress.—Idiopathic pneumonia is frequently ushered in with a day or two of feverish or undefinable feeling of illness. The invasion of the disease is generally marked by a sudden and severe rigor, or a succession of rigors, or in children by an attack of convulsions—phenomena which are attended with a rapid and considerable elevation of temperature, and the usual symptoms of inflammatory fever. The specific signs of the pulmonary affection usually declare themselves immediately or in the course of the next four-and-twenty hours; very rarely they are delayed for a still longer period. They consist in rapidity and shallowness of breathing, with more or less dyspnœa; dorsal decubitus; cough, soon attended with blood-stained glutinous sputum; pain probably in the affected side on drawing a deep breath; and, according to the stage which the pulmonary affection has reached, fine crepitation, or dulness with tubular breathing, and augmented bronchophony and vocal fremitus. While these local conditions are in progress, the patient's febrile state continues; his skin is hot and dry or perspiring, his tongue furred, his pulse accelerated; jaundice is apt to come on, and diarrhœa; his urine is scanty and perhaps albuminous; at the same time, probably, he suffers from more or less hebetude, with delirium, which comes on especially at night. The further progress of the case varies according to its severity. In very mild cases, after two or three days of illness, the patient's temperature falls, his other symptoms subside, and convalescence is established. In other favourable cases convalescence may be delayed for a week, ten days, or a fortnight; and the amendment may then be either sudden or gradual. In cases which end fatally, death may occur at any period of the disease, even during apparent convalescence, and is due, as a rule, either to asthenia or to gradual asphyxia, or to a combination of these conditions.

We will discuss *seriatim* some of the more important of the phenomena which attend pneumonia.

The respirations are usually hurried and shallow, and may vary in rate from the normal up to 50 or 60 and upwards in the minute; when very rapid they are usually attended with a sucking sound in the mouth, and expansile movements of the alæ nasi; there is often, but by no means necessarily, more or less severe dyspnœa, and generally there are signs of breathlessness when the patient attempts to speak.

Cough, which is sometimes very troublesome and even paroxysmal, is almost always present. It is at first dry, but is soon attended with the expectoration of transparent and very viscid mucus, tinged with the colouring matter of the blood. This is usually said to have a rusty tint, and indeed often has; but it varies in colour between a pale saffron and a bright vermilion, and in the latter case may be mistaken, on hasty inspection, for pure blood. After retaining this character for a few days, the expectoration loses its sanguineous tint and becomes opaque and greenish—acquires in fact a muco-purulent character—and then gradually diminishes in quantity. In some cases, instead of undergoing this, which may be regarded as the normal change, it acquires a deep purplish or reddish-brown tint and at the same time a more watery consistence. This form of sputum has been likened to prune-juice, and is generally a sign not only of increased congestion and escape of blood, but of the access of the third stage, and of an unfavourable issue. In some cases, again, the expectoration becomes distinctly purulent, or is attended with the horrible fetor which usually indicates pulmonary gangrene. The quantity and quality of the expectoration vary remarkably in different cases. In some there is absolutely none from first to last; in some the patient never coughs up more than one or two rusty-coloured sputa; in some the expectoration, even if abundant, never presents the characteristic tint. Pneumonic expectoration is characterised by the presence of a superabundance of common salt, and contains a considerable quantity of mucus and albumen.

There is much variety as to the presence and degree of thoracic pain. In some cases there is no pain whatever; in some there is a mere sense of heat; in some the patient has severe stitch whenever he coughs or draws a deep breath. This pain is pleuritic in character, and doubtless due to the coexistence of pleurisy.

In the first stage of the disease the most characteristic auscultatory phenomenon is minute crepitation, which may be audible during the whole of inspiration, sometimes during expiration as well, and not unfrequently only at the end of a deep inspiration, such as that which precedes a cough. In association with this there may be no change on percussion, or there may be high-pitched resonance or bruit de pot fêlé. The second stage is marked by the supervention of dulness over the consolidated portion of lung, with increase of vocal fremitus;

cessation of fine crepitation, and the development in its place of well-marked tubular breathing, and the corresponding whiffing character of cough and voice; bronchophony; and in some cases pectoriloquy. There may also be sharp metallic crepitation or rhonchus. In some cases (probably when the bronchial tubes leading to the consolidated portion of lung are completely obstructed) there is almost total absence of respiratory sounds and bronchophony over the affected region. It need scarcely be pointed out that, in consequence of the co-existence of pleurisy, it is common to get friction sounds mixed up with those due to pneumonia, and possible even for the pneumonic sounds to be suppressed or replaced by the phenomena indicative of pleurisy. At a later stage, when lung-tissue is breaking down, or resolution is taking place, tubular breathing gives way to a kind of coarse crepitation, to which the name of *crepitatio redux* has been given. This gradually passes into the ordinary bronchitic râles. It may be added that, when the pneumonic lung is consolidated, the movements of the thoracic walls in relation with it become impaired, and the resistance on percussion manifestly increased; and, further, that pneumonia may be present, deep-seated in the lung, or limited to its diaphragmatic or inner surface, and thus altogether escape detection by auscultation or percussion. Some degree of dulness on percussion usually persists long after the disappearance of the other local signs of pneumonia.

The cardiac pulsations are always increased in frequency during the febrile stage of the disease, but rarely increased proportionately to the respirations. Often indeed their ratio, instead of being about 4 to 1, sinks to 2 or $1\frac{1}{2}$ to 1. In adults the pulse may range from 80 or 90 up to 120; in children it is generally more rapid, and may rise to 200 and upwards. Extreme rapidity is generally associated with feebleness, and not unfrequently with irregularity, and is hence to be regarded as an unfavourable sign. In the beginning the pulse is often somewhat full and strong, but sometimes full, soft and dicrotous; later on it always becomes more or less feeble and dicrotous. During convalescence it may fall below the normal frequency. While pneumonia is in progress the systemic veins are apt to get overloaded, and the surface may assume a dusky hue. The blood always presents a large excess of fibrinogen.

The tongue is coated, and in some cases becomes dry and brown, and sordes accumulate upon the teeth. Thirst is pretty constant; there is always loss of appetite, and occasionally sickness. The bowels vary; sometimes they are not particularly affected throughout the disease; sometimes they are constipated; sometimes, on the other hand, there is more or less profuse diarrhœa, and this may be dysenteric in character. The occurrence of jaundice during the progress of pneumonia is neither uncommon, nor very important. It is said to occur most frequently in those cases in which the right lower lobe is

affected. But there is no more necessary connection between right pneumonia and jaundice than between left pneumonia and it.

The urine is scanty, dark-coloured, and of high specific gravity, presenting a diminished quantity of chloride of sodium and a great excess of urea and uric acid, with a tendency to the deposition of urates. Sometimes it contains also a little albumen, with hyaline, granular, or epithelial casts. During convalescence it gets much more abundant, pale, and of low specific gravity; and the urea undergoes diminution, while salt increases.

The face is more or less flushed in the early period of pneumonia, and may even be somewhat livid; the skin is generally hot and dry; but profuse sweats are not uncommon during the progress of the disease, and generally attend its decline. An herpetic eruption about the lips and alæ nasi is almost pathognomonic.

The patient at first complains of headache and general febrile pains. He is often drowsy, yet, at the same time, restless, especially at night time. Delirium is apt to come on early, at first being limited to the night, but subsequently becoming more or less constant. In some instances, and mainly in persons who have been given to drink, the nervous symptoms soon assume all the characters of delirium tremens. And again, patients, not otherwise obviously affected in mind, occasionally get suddenly and violently maniacal, the paroxysm possibly abating as suddenly as it arose. In fatal cases delirium is apt to pass into coma. Muscular tremors and subsultus, with loss of control over the bladder and rectum, are frequently observed in severe cases.

The temperature rapidly rises from the time of invasion, so that within a few hours, at most perhaps twelve, it has almost attained its maximum; this varies from 100° to 106°, or even more. Thenceforward the temperature remains high, probably increasing somewhat, with morning remissions and evening exacerbations, until the time of commencing convalescence, when it suddenly or gradually falls. In the former case it may sink to the normal or below it in the course of twenty-four hours. Occasionally in fatal cases the temperature rises rapidly before death.

The symptoms of uncomplicated idiopathic pneumonia are collectively so characteristic of the disease that it is almost impossible to mistake their significance. The affections, other than those of the respiratory organs, with which it is most liable to be confounded are typhus and enteric fevers. No real difficulty, however, can arise unless the specific characteristics of these fevers be in abeyance, and they be at the same time (as they often are) complicated with secondary pneumonia. It is altogether different, however, in respect of the various forms of intercurrent or secondary pneumonia, and of the lobular variety of the disease. These creep on, for the most part, insidiously in the course of other grave affections, which have already probably

produced serious pulmonary symptoms, such as dyspnœa, cough, expectoration of serous, mucous or bloody sputa, lividity of surface, and other indications of embarrassed circulation and carbonic acid poisoning; their onset is not usually marked by rigors or anything equivalent to rigors, nor is their progress usually attended with the high febrile disturbance which characterises the idiopathic variety; and, again, they are not often accompanied by labial herpes, or jaundice, and very often there is, excepting towards the close of the disease, an entire absence of delirium. The supervention of these forms of pneumonia may be suspected, in patients suffering from the various diseases which are apt to be complicated by them, when their symptoms, and especially those referrible to the respiratory organs, become aggravated; but they can only be positively determined by careful physical investigation of the condition of the thoracic organs. It must not be forgotten, however, that lobular pneumonia may be present to a considerable extent without producing either the characteristic dulness, the tubular breathing, or the other specific signs of the more uniformly diffused variety of the disease. The auscultatory and percussive phenomena indeed may differ little if at all from those which attend capillary bronchitis.

The breaking down of portions of lung-structure which occasionally attends the later stages of pneumonia does not reveal itself by any special sign, unless the cavities be such as, from their size or position, to give rise to characteristic auscultatory phenomena. In rare cases such abscesses burst into the pleura, or (the lung being adherent) perforate the thoracic walls, or form sinuses running down behind the peritoneum, and opening ultimately into the colon or some of the hollow viscera of the pelvis. The occurrence of gangrene is usually revealed by the disgusting fetor of the breath, especially during the processes of coughing and expectoration, and in a less degree by the look and smell of the sputa. Here also the cavities due to the destruction of lung-tissue may perhaps admit of detection. The presence of gangrene is generally attended with marked depression of the vital powers, or, in other words, collapse.

Pneumonia is always a disease of considerable gravity. Still, in its idiopathic form, it comparatively rarely kills, unless the portion of lung involved be extensive, or both lungs be attacked, or except in the case of persons advanced in years, or of those whose constitutions have been injured by long-continued bad habits, over-work, or disease. The secondary form of the disease, and especially the lobular variety, on the other hand, are exceedingly fatal, and may be included among the chief immediate causes of death in the various maladies which they complicate.

Treatment.—There are few diseases for which so many opposite plans of treatment have been employed with reputed success as for pneumonia. It is a disease, too, which, more perhaps than any other,

has on this very account been appealed to in proof of the change of type of disease. From the time of Laennec to about the middle of the present century almost implicit reliance was placed in the combined use of blood-letting, antimony, and mercury. Since then, especially dating from the time of Dr. Todd, these remedial agents have been to a very large extent discarded, and have got replaced by the free exhibition of alcoholic stimulants. Many, indeed, now regard all medicinal treatment as of little or no importance; and it is quite certain that a large number of even severe cases recover perfectly if left to nature and the nurse.

In the majority of cases of the idiopathic disease it is probably quite sufficient to keep the patient in bed in a comfortable, well-ventilated room, of medium temperature; to relieve thoracic pains with mustard plaisters and the like; to assuage febrile thirst by the exhibition of soda-water, orangeade, or lemonade; to support strength by the frequent administration of milk or gruel, or some equivalent nutritious fluid; and to relieve, from time to time, by simple measures, diarrhœa or constipation, and other remediable derangements of the various organs; and then, as convalescence comes on, to give vegetable tonics, and gradually to improve the diet in respect of both quantity and quality. It is doubtless true, however, that, in many cases, the above plan of treatment may be judiciously supplemented by other measures. Bleeding from the arm, or the local abstraction of blood from the chest by cupping or leeches, is certainly followed by relief to symptoms when employed early in cases in which there is high fever and much dyspnœa. We believe that bleeding from the arm is more efficacious than the other methods of bleeding, but in any case it is better to remove sufficient blood at a single operation than to be called upon to repeat it. Counter-irritants and detergents are often serviceable at a later period of the disease, or at the beginning of slight cases in which bleeding is not deemed necessary. They relieve pain, and sometimes diminish difficulty of breathing. Dry cupping is of especial value. Some physicians think it well to keep the affected side invested in a large poultice or a layer of cotton-wool; others prefer the application of ice-bags or cold compresses. We do not think that any benefit accrues from the former plan, but the latter probably has some advantage. Expectorants, such as ipecacuanha in small doses, may possibly aid those cases in which there is frequent and troublesome cough, with difficulty of expectoration; and, under the same circumstances, the addition of a small quantity of opium may be serviceable.

When the pulse gets very quick and weak, and delirium is established, especially if the patient present the general symptoms of delirium tremens, diffusible stimulants, such as ammonia, and alcoholic drinks, in quantities to be determined by their effects, are indispensable. It may be added that, with the object of reducing temperature, various

agents have been recommended; and possibly one or other of them may sometimes be used with advantage; among these may be enumerated cold baths, quinine in large doses, salicylic acid, veratria, digitalis, and aconite. The occurrence of suppuration or gangrene is a special reason for the maintenance of the patient's strength by nutritious food, stimulants, and tonic medicines. Opium is often of great service, but should not be given, or should be given very cautiously, when the patient is suffering from dyspnœa and insufficient aëration of the blood. The treatment of secondary pneumonia merges in the treatment of the disease it complicates. Its supervention, however, is on the whole a plea, not for depletion, but for the opposite plan, namely, the use of stimulants and of nourishment.

V. PLEURISY. (*Pleuritis.*)

Causation.—Pleurisy is either idiopathic, or the result of local irritation. The former class of cases includes pleurisy arising directly from exposure to cold—the form of pleurisy which corresponds to idiopathic pneumonia and bronchitis—and that which takes place in the course of acute rheumatism. Among the latter class may be enumerated, pleurisy due to extension of inflammation from inflammatory and other affections of the lungs, or thoracic parietes; that due to mechanical injuries, more especially to the rupture into the pleura of pulmonary cavities, or of abscesses of the liver or other neighbouring organs; as also probably the pleurisy which is so commonly associated with the progress of pulmonary phthisis and thoracic carcinoma. In addition to the varieties of pleurisy here enumerated must be mentioned those which are developed in the course of small-pox, scarlatina, enteric fever, pyæmia, albuminuria, and heart disease, and in respect of which these several affections act variously, sometimes as exciting, sometimes, and perhaps more frequently, as predisposing causes.

Morbid anatomy.—Inflammation of the pleura, like that of all other serous membranes, commences with hyperæmia of the blood-vessels, proliferation of the protoplasmic elements of the tissues, more especially the epithelium, and effusion of inflammatory lymph, comprising various but pretty considerable quantities of albumen, fibrine, and corpuscles. The last two for the most part remain adherent to the surface, forming the so-called 'false membrane;' the fluid, containing albumen, fibrinogen, and a variable proportion of corpuscles, accumulates within the serous cavity. The inflammatory process combines, therefore, three elements, which may conveniently be considered independently of one another, namely—first, hyperæmia and infiltration

of the serous and sub-serous tissues; second, the formation of a false membrane; and, third, the effusion of serum.

The first of these elements is the first in order of development; but it seldom attains a high degree or forms a prominent item in the collective inflammatory changes. There is always, however, more or less obvious infiltration—its amount having some relation to the intensity of the inflammatory attack; and this not unfrequently extends into the connective tissue round about, as, for example, along the interlobular septa of the lungs, and into the tissues of the mediastina, diaphragm, and external thoracic parietes. And thus it occasionally happens that the superficial stratum of lung-tissue becomes involved, that the diaphragm and intercostal muscles suffer, and that inflammation, commencing in one serous cavity, extends to those which are in its immediate vicinity.

The effusion of inflammatory lymph always begins early. At first it constitutes an exceedingly thin, granular, but more or less coherent pellicle, the presence of which renders the serous surface obviously rough to the finger and deprives it of its polish. This gradually extends in area and increases in thickness, usually becoming at the same time more and more yellow and translucent. The thickness which it may attain varies roughly from that of a mere film up to half an inch or even an inch. The character of its surface presents numerous varieties, which depend partly on the tendency of the lymph itself to be deposited in the form of a network, partly on the attrition to which it is exposed during the movements of the opposed surfaces upon one another, and partly on its stickiness. It thus acquires a more or less irregular ribbed, villous, or retiform character. When the opposed surfaces of an inflamed pleura are separated by fluid effusion it often happens that trabeculæ and bands or septa of inflammatory lymph pass irregularly between them. The attached surface of the false membrane is always closer in texture and tougher than its free surface, and becomes with age more and more firmly united to the proper serous membrane, with which indeed it ultimately gets incorporated. It is here, too, that organisation, with the formation of new blood-vessels, commences—a process which, if the case go on favourably, ultimately pervades the entire thickness of the false membrane, and leads to the blending of the opposed layers, to their conversion into connective tissue, and to the obliteration of more or less of the pleural cavity.

The fluid effusion varies greatly in quantity relatively to the other two products of the inflammatory process. It is difficult in many cases to account for this fact, but occasionally it is explicable in some degree on mechanical grounds. In the first instance the fluid is transparent yellowish or greenish, and probably presents flakes of lymph floating in it. In many cases it retains this character throughout; but in some it becomes turbid or opaline, and deposits a little milky sediment on

standing. Occasionally it acquires the characters of ordinary pus. It may be added that blood is sometimes extravasated, either from rupture of the new-formed vessels of the false membrane or from ulcerative destruction of the subjacent lung-tissue; that gas is occasionally present—an occurrence due either to an external wound or to some communication between the lung or intestine and the pleura; and lastly, that the purulent contents occasionally get fetid. The quantity of fluid effused may vary from almost zero up to two or three quarts.

In a large number of cases, especially those in which the pleurisy is due to the extension of inflammation from subjacent parts—as for example, when it arises in the course of peritonitis, pericarditis, lobar pneumonia, or the various forms of disseminated pneumonia—the discharge of serum is very scanty, and the lymph forms a thin film, which may be limited to the area primarily involved, and that opposed to it, or may gradually creep over the whole pleural surface. Moreover, in such cases it is far less liable to spread from below upwards than from above downwards; but generally even in slight cases with little effusion it is common for the inflammatory products (solid as well as fluid) to subside to the most dependent part of the pleural cavity and to accumulate there.

In other cases, and more particularly, perhaps, in the idiopathic form of the disease, in those varieties of it which attend small-pox and other eruptive fevers, or tuberculosis, and in that due to perforation of the pleura by abscess, effusion takes place rapidly and copiously. The effects of the accumulating fluid are the distension of the pleural cavity, the compression of the lung, and the displacement in different degrees of the surrounding organs. As the fluid rises in the thorax, more and more of the lung, commencing with its lower part, has its air squeezed out of it. Subsequently, perhaps, the whole organ suffers, and consequently becomes remarkably reduced in size, and compressed into the neighbourhood of its root and the upper part of the angle between the vertebræ and ribs. There, in fact, it may lie concealed from view by a layer of lymph continuous with that lining the thoracic parieties; and the unskilled pathologist might at first sight readily assume that the lung had undergone total destruction. If the lung have been the seat of consolidation, or if it have been previously bound to the parietes here and there by old adhesions, or if the distension of the pleura with fluid be incomplete, the compressed lung will probably hang or protrude more or less irregularly into the pleural accumulation. The abundant presence of fluid causes, in addition to compression of the lung, displacement of the heart and mediastinum towards the opposite side (especially observable when the left pleura is affected), depression of the diaphragm, and expansion of the outer parietes of the thorax, with widening and probably bulging of the intercostal spaces.

When suppuration (*empyema*) takes place (an occurrence of special frequency in the pleurisy of small-pox, scarlet fever, measles, and pyæmia, and in that of women who have just undergone childbirth, or from perforation), all the phenomena just described naturally ensue; but others due to the presence of pus will probably be superadded. The abscess sooner or later tends to point. Not unfrequently it opens into the lung; and often it makes its way through the thoracic parietes, forming, in the first instance, a sinus between the ribs, which probably become to a greater or less extent exposed and carious; then an accumulation between the ribs and the integuments, which, gradually enlarging, may develop, ere an external opening takes place, into a large superficial abscess. The route which such a sinus may take, and the point at which it may present, are liable to great variety. Thus sometimes the abscess appears at the upper part of the thorax and even above the clavicle; sometimes it opens in the loin below the level of the twelfth rib. Much more frequently, however, it occupies some intermediate position. In rare cases the empyema perforates the diaphragm, and it may then take the course of a renal or psoas abscess, and finally open in any of the situations in which such abscesses are liable to open.

The ultimate consequences of pleurisy are various. In the great majority of cases the fluid accumulation undergoes absorption, the partially-compressed lung recovers itself, and the effused lymph is slowly converted into a kind of cicatricial connective tissue, which remains permanently. This may ultimately constitute a mere white opacity upon some portion or portions of the pleural membrane; or, what is far more common, result in the formation of adhesions between the opposed surfaces. The latter may consist in a mere intervening film of connective tissue, or in groups of filaments and bands of various lengths, or in tissue as close, dense, and tough as cartilage or tendon, and which in process of time may become the seat of calcareous deposit. Adhesions may be limited to one or two points only, or may be generally but irregularly distributed, or may involve the whole extent of the pleura, the cavity of which then ceases to exist.

When the lung has long been compressed by fluid (whether serum or pus), and rendered entirely airless, especially if at the same time it has been covered with a thick dense layer of false membrane, the absorption or removal of the fluid is probably attended with little or no restoration of the lung; and the space which that organ occupied becomes filled up by the falling in of the surrounding parts. The mediastina and the heart are drawn over towards the affected side; the corresponding half of the diaphragm rises, carrying with it the stomach or the liver, as the case may be; the ribs get retracted and approximated; the shoulder falls; the spine bends in the same direction; and the patient's carriage undergoes a corresponding change. At the same time the adhesions probably remain abundant

and thick, and sometimes œdematous. In many cases, on the other hand, when compression has been less complete, or the adhesions are less strong, convalescence, even after an extreme amount of pleural effusion, is attended with more or less restoration of the affected lung —an event which often requires considerable time for its completion. In some such cases, when death has ensued before the entire removal of the fluid, the lung is found to be invested in a fenestrated layer of pretty dense false membrane, which by the general pressure it exerts renders the organ irregularly rounded, while the fenestræ permit of irregularly distributed lobulated protrusions of crepitant lung-tissue.

An empyema may, after the discharge of its contents, be followed by any of the consequences above enumerated; but, like other deep-seated abscesses, its cavity often fails to get wholly obliterated, and a sinus results, through which it continues to discharge for an indefinite period. This tendency for an empyema to remain open is occasionally traceable to a carious condition of the ribs. On the other hand, circumscribed collections of pus here as elsewhere sometimes dry up into caseous masses.

Lastly, inflammation may attack a pleura already partially or wholly obliterated by adhesions. In the latter case the consequences will probably be congestion, infiltration, and thickening of the pre-existing false membrane. In the former case the effused serum or pus will occupy either a more or less definitely circumscribed space or the whole pleural cavity, divided by bands and septa into a series of inter-communicating loculi. Such limited accumulations of fluid are occasionally met with in the interlobular fissures, or between the diaphragm and base of the lung, or between the inner aspect of the lung and the mediastina—situations in which they often escape recognition during life.

Symptoms and progress.—The symptoms of pleurisy present great variety, both in intensity and in kind—the differences being due mainly to differences in the extent, position, and intensity of the inflammation, in the circumstances under which it is developed, in the diseases with which it is associated, and in the stage at which it has arrived. The specific symptoms nevertheless are simple enough, and, in addition to the signs furnished by percussion and auscultation, principally comprise thoracic pain during respiration, dyspnœa, and inflammatory fever.

The invasion of idiopathic pleurisy is far from uniform in its symptoms. In some cases the patient complains only of a little feverishness, loss of appetite, and general malaise, together with a stitch or pain in one side when he breathes deeply, or coughs, or twists his body or moves the corresponding arm; and he may continue to follow his ordinary avocation until, in the course of a week or two, or more, he is restored to health, or until, at the end perhaps of an equally long time, increasing illness and difficulty of breathing make him consult a

medical man, who may possibly then find the implicated side distended with fluid. In other cases the patient is suddenly seized with rigors, or (and this may occur even in adults) an epileptiform attack, followed by high febrile symptoms and the characteristic stitch. In other cases, again, after he has complained for a day or two of some degree of feverishness and pain in the side, the symptoms, both local and febrile, assume sudden intensity.

But, however the disease comes on, whether with rigors or with none, whether slowly and insidiously or by sudden onset, it rarely happens, unless it be suppurative from the beginning or dependent on the presence of some blood-poison, that the temperature rises above 102°; often, indeed, it does not exceed 100°; and it may be scarcely above the normal. The condition of the pulse and the other general symptoms have some relation with the temperature. The pulse is generally full and vibratile or dicrotous, and somewhat increased in frequency, the skin is hot, the tongue more or less furred, the appetite impaired, the thirst increased, the urine scanty and high-coloured, and the bowels confined. There are probably also headache and general febrile pains. From the beginning the patient has a stitch, which is usually referred to the mammary region, and the presence of which renders deep inspiration and all thoracic movements painful, so that the breath becomes hurried, shallow, perhaps irregular, and frequently attended with an expiratory groan, and the patient avoids all unnecessary movement. There is usually also some tenderness on pressure and percussion of the affected side.

While these symptoms are present and the pleurisy remains in the so-called 'dry stage,' percussion may perhaps reveal some little dulness at the base of the pleural cavity, and auscultation may detect, here or elsewhere, some variety of friction-sound. Cough is often absent, and, when present, dry, or attended only with a little frothy expectoration. It is rarely severe, but is sometimes paroxysmal and troublesome, and always painful.

As effusion increases, the pleuritic stitch for the most part diminishes, and may at length wholly disappear. Meanwhile the febrile temperature and general symptoms of illness may remain at the same level, or undergo some diminution. Dyspnœa may or may not increase; and it is an important fact that the effusion of sufficient fluid to distend the pleural cavity is in some cases attended with little or no obvious dyspnœa so long as the patient remains at rest. On the whole, however, dyspnœa increases with increase of fluid accumulation; and the patient not only breathes rapidly, but suffers from much distress and anxiety, gets pale or livid, even to cyanosis, and presents all the phenomena of slow asphyxia. The presence of fluid in the pleura is indicated: by dulness on percussion up to the level at which the fluid stands—the level, in many cases, distinctly varying, in relation to the different points of the thoracic walls, with the patient's

movements; by suppression of vocal fremitus over the dull part; and generally by absence of respiratory sounds over the same region. Faint tubular sounds, however, or even an indistinct vesicular murmur is occasionally audible. Ægophony is usually to be heard about the angle of the scapula; sometimes, also, friction-sounds above the level of dulness, especially in front; and high-pitched resonance and the bruit de pot fêlé over the uncompressed portion of lung.

When the effusion fills the pleural cavity, and the lung is wholly compressed, dulness of the side, with absence of vocal fremitus, becomes general, and both ægophony and respiratory sounds cease. The last, however, may generally still be heard about the apex, in front and behind, and thence downwards behind, between the scapula and spine. But, in addition to these phenomena, the heart becomes displaced, the diaphragm thrust down, the side distended and almost immovable, with dilated intercostal spaces, over which, by careful manipulation, fluctuation may sometimes be detected.

Convalescence may commence at any stage. In a large proportion of cases, the patient begins to recover before there has been any obvious effusion of fluid; pain in the side gradually ceases, febrile symptoms (if there be any) subside, and friction slowly vanishes. In other cases convalescence does not commence until after fluid has accumulated, and more or less of the lung has been compressed. Here, again, convalescence is indicated by subsidence of fever and general improvement in the condition of the patient's bodily functions; his breathing becomes more natural, and his appetite returns. At the same time the effused fluid is gradually absorbed, the pleural surfaces come again into contact and consequently pain may return temporarily and friction be re-established. Indeed, friction is often a more marked phenomenon of convalescence than of the early stage of the disease. It may happen that, with the disappearance of the fluid, the lung enlarges, and healthy respiratory sounds are speedily restored; but, even in favourable cases, it is usually a long time (it may be months) before friction wholly disappears, and even longer before resonance and respiratory sounds return to the basal portion of the affected side of the chest. In less favourable cases, the lung is restored in part only, or remains permanently collapsed. Then all those changes in the form of the side and arrangement of internal organs, which have been already described, ensue. But even here some improvement may be hoped for in the course of years. The patient, however, usually remains weakly and short of breath.

The common cause of death in simple pleurisy is asphyxia due to the pressure of the accumulated fluid; the patient may die, however, from syncope or asthenia, and in either case death is apt to take place suddenly.

The supervention of suppuration—the development of empyema—is often insidious and unattended with either the aggravation of old

symptoms or the occurrence of new ones. The simple long persistence of copious effusion affords presumptive evidence of suppuration. Suppuration is generally indicated also when there has been, from the beginning of the attack, much fever, and rapid filling of the side with fluid; and especially when, in the course of a case hitherto of only moderate severity, rigors occur, and fever, becoming greatly augmented, continues augmented. The local indications of empyema are not necessarily more pronounced than the general symptoms. In addition to those of distension from mere accumulation, we sometimes observe general or partial œdema of the integuments on the affected side, sometimes distinct bulging of the intercostal spaces, sometimes unnatural distinctness of the superficial veins, and sometimes a circumscribed redness and induration, or a fluctuating swelling superficial to the ribs, due to the escape of matter from the pleural cavity through an intercostal space into the soft tissues beneath the integuments. We have pointed out that an empyema may burrow in almost any direction and discharge itself at almost any surface; the most important practical terminations of this kind, however, are by perforation of the lung and by perforation of the thoracic parietes. In the former case the patient suddenly expectorates a large quantity of pus, and may continue henceforth to discharge pus, either continuously in comparatively small quantities, or at irregular intervals profusely. In the case of discharge through the thoracic parietes, the abscess first points, and then opens either spontaneously or by operation, and as in the former case pus, in more or less abundance, escapes, and probably continues to escape. The sudden expectoration of pus, or the appearance of an abscess in the thoracic walls, is sometimes the first clear indication that there has been a circumscribed empyema. But it must not be forgotten that a superficial abscess often communicates, by a comparatively long and tortuous passage, with the internal abscess which gave it origin; and that hence (in the case of circumscribed empyema) it may be impossible to trace it back to its source, and make sure of its empyematic origin. Thus an abscess of the lower part of the pleura may be readily and pardonably mistaken for a perinephritic or lumbar abscess. The progress of a discharging empyema is, as has already been pointed out, apt to be very chronic, especially if the original cavity were large—the discharge then often becoming fetid; and in dependence mainly on the copiousness of the discharge, the patient becomes emaciated, and presents the ordinary symptoms of hectic fever. In many such cases, fortunately, more or less complete recovery takes place after a time; this event is, on the whole, more frequent when the empyema opens through the lung than when it discharges externally—a circumstance which seems to depend in some degree on the much greater tendency there is in the latter case than in the former to the decomposition of the purulent contents. In many cases, on the other hand, the patient sinks slowly

and at length dies, worn out and exhausted, or he is carried off by sudden intrapleural hemorrhage, or asphyxia.

It often happens that the communication of an empyema with the bronchial tubes, or directly with the external atmosphere, permits of the entrance of air into the pleural sac, and that hence pneumothorax is established. The supervention of this condition may ordinarily be recognised by the presence of augmented resonance over the air-containing portion of the cavity, of the splashing sound caused by succussion, of cavernous resonance, and probably of distinct metallic tinkling.

In the foregoing account we have discussed the symptoms mainly of simple unilateral idiopathic pleurisy; it may be added that the symptoms of the complicated disease are essentially the same, but that they are interwoven with those of the complicating disorder, and are sometimes masked by them; and further that both pleuræ are occasionally implicated, with corresponding aggravation of symptoms. We may also add that pleuritic patients, during the period of effusion, usually lie on or towards the affected side; and also that they much more frequently suffer from cough than might perhaps be gathered from the remarks we have made. The cough, however, is no necessary part of the disease, and is often due to the presence of associated pneumonia or bronchitis.

Treatment.—The treatment of ordinary cases of pleurisy is not usually a matter of anxiety. In mild cases of so-called dry pleurisy the application of a mustard plaister or other counter-irritant, the binding of the chest with a broad flannel roller or the affected side with strapping to restrain its movements, and the use of opiates in small doses will probably be sufficient.

In severer cases, in which there is manifest fever and increasing effusion, it is often beneficial to apply (according to circumstances) from half a dozen to a dozen leeches to the surface of the chest, to follow up their application by poultices or flannels wrung out in hot water, and then perhaps after a time by counter-irritants. In these cases, even more than in the former, opiates are of value, if only to alleviate pain and distress. Soda-water, or some other febrifuge medicine, may also be employed.

If the effusion still increase, and especially if the patient begin to suffer from shortness of breath, the arrest of the effusion and the removal of the fluid which has already accumulated become the chief indications for treatment. For these purposes diuretics, diaphoretics, and purgatives have each been strongly advocated, and among drugs mercury, antimony, digitalis, and iodide of potassium. We believe that all such agents are practically useless for the purposes here indicated, and that, if we are to trust in drugs at all, they should be those which, by tending to improve the general health of the system, tend indirectly to promote healthy action at the seat of disease: we mean

tonics, especially iron and quinine. Counter-irritants, and especially repeated small blisters, sometimes seem to aid absorption. The only other means at our disposal for the removal of fluid, and this is in many respects by far the best, is paracentesis. This operation was formerly greatly dreaded and seldom performed except in cases of empyema already pointing. It is in great measure due to Trousseau that, during the last thirty years, paracentesis has come to be recognised as a safe and efficacious procedure in cases of excessive accumulation of simple serum. More recently, especially since the introduction of suction instruments, and through the able advocacy of Dr. Bowditch, the use of the operation has been still more widely extended. The objects to be attained by paracentesis are: first, the removal of pressure from the lung so as to permit of its redistension; second, the prevention of death from suffocation; and, third, the removal of purulent fluid. It is also generally believed, and perhaps correctly, that the discharge of a certain proportion of fluid from a distended cavity promotes the absorption of the rest.

With the first of the above objects the fluid should be let out early, inasmuch as the longer the lung has been compressed and the more firmly it is bound down by adhesions the less likely is restoration to take place. With the second of these objects the pleura should be punctured either when the patient suffers from obvious difficulty of breathing, or when, even if dyspnœa seems absent, the cavity is greatly distended. The suspected presence of pus is always a legitimate ground for operation. In all these cases a fine trocar and cannula should be employed; the instrument should be plunged into the chest at a suitable point, generally, as recommended by Dr. Bowditch, an intercostal space directly below the angle of the scapula and above the lower limit of the opposite healthy lung; and the fluid should be removed either by the aspirator, or by a tube guarded by a valvular fold of goldbeater's skin, so as to prevent the admission of air. The entrance of air, however, though an accident to be avoided as involving additional risk, often has no ill effect. It is not generally advisable to attempt the removal of the whole of the fluid at one time. If pus be present it may be taken away by periodical aspirations, or its free discharge may be maintained through a permanent opening; we prefer the former, especially in the case of children. For the different methods by which at the same time a free escape of pus may be allowed and the entrance of air prevented, we must refer to surgical works. It is sufficient to say here that, so long as the discharge remains sweet, the entrance of air must be carefully guarded against; but that when it gets fetid, little is to be gained by further exclusion of air. It then becomes important to wash out the cavity daily either with pure water or with water medicated with quinine, nitric acid, chlorinated soda, or carbolic acid.

The operation of paracentesis with a very fine trocar and cannula, if

air be excluded, is perfectly harmless. And for this reason, as well as on account of the great importance of preventing permanent collapse of the lung, we strongly uphold the practice of the early, and if necessary, repeated removal of pleuritic fluid. Again, it is of little practical importance if in attempting paracentesis we wound the lung, kidney, or other neighbouring organs, and hence, although we recommend caution, we advocate early exploratory puncture when there is reason to suspect the presence of circumscribed accumulations of pus.

In the treatment of chronic pleurisy, or empyema, and during the whole period of convalescence, the importance of tonics, good diet, and change of air cannot be over-estimated.

VI. CIRRHOSIS. (*Chronic pneumonia. Fibroid phthisis.*)

Definition.—A distinction is not unfrequently made between cirrhosis and chronic inflammation of the lungs. It is difficult, however, to appreciate in what the difference consists; and we prefer, therefore, to regard the two conditions as identical. We mean by these expressions induration of the lung, by the development of nucleated fibroid tissue, either around the bronchial tubes, or in the interlobular septa, or in the walls of the air-cells, or in all these situations at once, and the consequent gradual effacement of the air-cells.

Causation.—There is reason to believe that cirrhosis is an occasional result of ordinary acute pneumonia; it is far more frequently, however, a sequel of catarrhal or lobular pneumonia, and of chronic pleurisy with effusion. A not uncommon cause is the habitual inhalation of solid particles, such as those of coal-dust, mill-stone grit, copper ore, flax-dust, and the like, by those whose occupations expose them to the danger of such inhalations. It is certain that it occasionally ensues on simple chronic bronchitis and on the retrogression of both grey and caseous tubercular deposits. The question how far, in some cases, it is to be regarded as the result of a constitutional taint, has been often raised. There is no doubt that we occasionally meet with a similar condition simultaneously involving several organs,—more especially the lungs, liver, and kidneys,—a fact which is certainly entitled to some weight on the affirmative side of the question. But, on the other hand, it must be remarked that hepatic cirrhosis is traceable, in the great majority of cases, to the influence of alcoholic irritation of the matrix of the liver, and that pulmonary cirrhosis (independent of tuberculosis) is usually limited exclusively to one or other lung—facts which are at least as weighty on the opposite side.

Morbid anatomy.—Cirrhosis of the lung consists essentially in the gradual invasion of the solid tissues of the organ by a nucleated fibroid

growth. This, on the one hand, surrounds and involves the bronchial tubes (especially the smaller ones) and the vessels which accompany them; on the other hand, invests the lung itself (which is then usually strongly adherent to the parietes) and separates its lobes from one another; and from both sides is prolonged into the inter-lobular septa, so as to divide the lung-tissue by bands of fibroid tissue of different degrees of density, thickness, and visibility, into a series of polygonal islets. With the further progress of the disease, the same kind of thickening takes place irregularly in the walls of the air-cells, so that before long the cut surface presents a coarse retiform arrangement of dense fibroid tissue; and this, gradually increasing, finally renders the whole organ, or portions of it, uniformly dense, hard, and airless. It must be observed that, although in cirrhosis there are usually both induration of the tissues around the bronchial tubes and dense adhesions between the opposed pleural surfaces, it often happens that the most obvious, if not the primary, change is that which pervades the ultimate tissue of the lungs. It need scarcely be said that this is necessarily the most important.

Accompanying the interstitial growth of fibroid tissue, there is usually a more or less abundant deposit of black pigment in irregular patches. This is natural in the lungs of persons advanced in age; but in cirrhosis it is often, if not always, excessive. The pigment is seated in the thickened walls of the air-cells and especially in the connective tissue which surrounds the bronchial tubes and vessels, and separates lobules from one another. It is always abundant also in the bronchial glands. It may often be found distinctly contained in the connective-tissue corpuscles, and taking the course of the lymphatic vessels. There is good reason to believe that it is to a large extent carbonaceous matter of extraneous origin, which has been inhaled into the lungs, has been absorbed by the mucous surface of the respiratory tract, and has then got deposited in the tissues and taken up by the lymphatics. The presence of pigment usually gives a peculiar mottled aspect to the sectional surface of the cirrhosed lung; but if in great abundance, it renders the tissues uniformly and intensely black.

The ultimate effect of cirrhosis of the lung, like that of the same condition in the liver, although it may perhaps under some circumstances cause temporary enlargement, is to produce gradual contraction and diminution of the organ. The progress of the disease is further always complicated with dilatation and other changes in the bronchial tubes, and not unfrequently with equivalent affections of the air-cells. The larger tubes are generally more or less considerably dilated, the fibroid and muscular bands which mark their mucous surface with longitudinal and transverse ridges are hypertrophied and produce a coarsely reticulated appearance, and the mucous membrane itself is probably congested and thickened. The chief changes, however, occur in connection with the smaller tubes, which in some cases

are dilated into bulb-ended channels; sometimes terminate in round or sub-globular cystiform expansions, from the size of a cherry to that of a small pea; sometimes open (several of them in common) into cysts or cavities of large size and irregular form; sometimes are continued into recently-formed and progressing cavities, which, when small, may easily be recognised as originating in the ulcerative destruction of the walls of the smaller tubes and air-passages. The mode of origin of dilated tubes in this and other pathological conditions will be considered hereafter. It will be sufficient to say here, that, in many cases, so-called 'dilated tubes' are merely tubes in communication with cavities whose walls have undergone cicatrisation; that there is (as might be supposed) a strong tendency for the adventitious fibroid growth of cirrhosis to undergo liquefaction under the influence of inflammatory processes commencing at the bronchial surface; and that the formation not only of vomicæ, but probably also of many dilated tubes, are referrible to such liquefaction. Ordinarily in cirrhosis the air-cells undergo gradual obliteration, their diminishing cavities being sometimes filled with disintegrating epithelial and other cells; but not unfrequently more or less emphysema is developed at the same time. When cirrhosis is limited to some comparatively small tract of lung, emphysema is common in the tissue which immediately bounds the indurated patch. Occasionally, also, the formation of a dense fibrous reticulum throughout the lung is associated to a greater or less extent with the breaking down of the thickened walls of dilated air-cells, so that the cut surface of the lung becomes not altogether unlike that of a coarse sponge. We have an impression that the condition last described may ensue on the retrogression of a crop of miliary tubercles.

Cirrhotic lungs present very great variety of appearance and character; at the same time it is easy to see that, however much they may differ from one another in the stage of the disease which they have reached, in the amount of pigment which is present in them, in the condition of their bronchial tubes, and in the tendency to the formation of vomicæ, they are all linked together by the community of their origin in simple fibroid overgrowth. The following are some of the varieties of cirrhosis which have been described and named:—*Red induration*, the name given to an early or slight condition of the disease, in which the lung is of large size, red, and fleshy, and, although denser than natural, and infiltrated to some extent with adventitious growth, is still generally crepitant; *brown induration*, the name employed to designate a condition of lung in which the capillaries are dilated and thickened, and in which the colour of the organ has a yellowish-brown tint, and the fluid exuding on pressure is similarly coloured, in consequence of the presence in the tissues of the lung of the colouring matter of the blood in the form of pigment-granules; brown induration is especially an accompaniment of heart disease; *grey induration*, the name which is sometimes applied to the condition of

the lung in advanced cirrhosis, when the organ is extensively infiltrated with fibroid matter and presents in consequence a general greyish tint and a more or less translucent aspect; *black induration*, which is sometimes used as the designation of that form of cirrhosis in which the cirrhotic tissue is largely infiltrated with black pigment, and of which the most striking examples are furnished by the lungs of persons working in mines or otherwise exposed to the inhalation of soot or other carbonaceous matters. It may be added that the pulmonary affections which are so frequently the causes of death amongst those who are engaged in certain avocations, as, for example, amongst miners, colliers, flax-dressers, millstone grinders, and the like, and which are commonly known as the phthisis of those who are thus respectively engaged, are mostly, as has already been indicated, of the nature of cirrhosis. They originate in the bronchitis which is caused and maintained by the constant inhalation of solid particles; of which many get deposited in the solid tissue of the lungs, and remain there permanently. The fibroid infiltration slowly supervenes. It appears from Dr. Greenhow's investigations that the nature of the dust inhaled does not exert any specific influence over the morbid changes which ensue. The nature of the imbedded particles can generally, however, be pretty readily recognised with the aid either of the microscope or of chemical reagents.

Symptoms.—The symptoms of cirrhosis of the lungs, apart from those of the numerous conditions which complicate it, and from those of the morbid conditions out of which it may have arisen, scarcely admit of description or recognition. The disease is one the progress of which is exceedingly chronic, and may be prolonged for five, ten, or even fifteen years.

It is easy to see that, if any large extent of lung-tissue be involved, the patient must suffer from progressive breathlessness; that from the obstruction which the indurated and contracted lung-tissue opposes to the pulmonic circulation, hypertrophy and dilatation of the right side of the heart must ensue, to be followed sooner or later by general anasarca; that there must gradually supervene impairment of nutrition, failure of the general powers of the body, weakness and emaciation; that the pulmonary changes must result in impairment of thoracic movement with retraction of the thoracic parietes, more or less obvious dulness on percussion, and either suppression of the respiratory sounds, tubular breathing, or (if there be secretion into the tubes) the various unnatural sounds which bronchial accumulation is competent to induce. Generally, moreover, there are present (at all events at some stage or other of the affection) more or less bronchitis with secretion, more or less dilatation of the tubes or air-cells, more or less breaking down of tissue with the formation of vomicæ, and more or less distinct inflammatory action; and the symptoms of these conditions must be added in order to have a true picture of the symptomatic phenomena of cirrhosis of the lungs.

Briefly, then, it may be stated that a patient with cirrhosis presents the following symptoms variously combined:—He has more or less obvious dyspnœa, especially on exertion, which gradually increases upon him, and is generally aggravated during the winter months, or by the occurrence of catarrh or pulmonary inflammation. Pallor and lividity of surface, with congestion of the nose, fingers, and toes, often supervene sooner or later. Cough is almost always present in a greater or less degree, and in some cases is very severe; it may, however, be wholly absent, especially during warm weather. It may or may not be attended with expectoration; but expectoration is often profuse, especially when the cirrhosis is complicated with dilated tubes or vomicæ, and generally muco-purulent or purulent. Under the same circumstances it is liable to be extremely fetid, and, in the case of colliers and others, almost black from the presence of pigment-particles. Hæmoptysis is not unfrequent. In many cases the sputa are merely streaked with blood as in ordinary chronic bronchitis; in some cases, however, more or less profuse hemorrhage occurs from time to time. This is due sometimes to perforation of blood-vessels in the course of destructive changes, sometimes to intense hyperæmia (probably of inflammatory origin) of the lining membrane of the dilated tubes. The auscultatory and percussive phenomena will be considerably modified according as dilated tubes or cavities are absent or present, and according as these are full or empty of fluid. The pulse may at first present little departure from the normal, but as the disease progresses it tends to become rapid and weak, and sometimes irregular; and at the same time, as has been pointed out, general anasarca may ensue. Elevation of temperature and other febrile symptoms are very variable in their occurrence. Not unfrequently, at certain periods of the affection, there is a total absence of them. But much more commonly the patient presents more or less of the usual symptoms of hectic fever: some degree of elevation of temperature, which, however, is liable to fluctuations; perspirations; loss of appetite; sometimes vomiting and diarrhœa; and gradually increasing emaciation and debility. The local and general symptoms and history of cirrhosis not unfrequently closely resemble those of retraction of the lung after simple pleurisy, or those of chronic bronchitis with emphysema, or those of phthisis.

Treatment.—Our principal aims in the treatment of cirrhosis should be, by attention to hygiene and diet, to arrest the progress of the morbid process, to prevent the supervention of complications, and to maintain the bodily strength. For these purposes change of scene, removal to a mild but equable and bracing air in the summer, and to a warm southern climate in the winter, the avoidance of night air, exposure to sudden chills, over-fatigue and the like, the use of good, wholesome, and abundant diet, with a moderate amount of stimulants, and the exhibition of quinine, iron, cod-liver oil, or other tonics, are of vital importance. When the cirrhosis is due to occupation, the

patient should give it up and follow some more healthy pursuit. But, in addition, symptoms, as they arise, will necessarily call for treatment: cough and expectoration may demand opiates and expectorants, hæmoptysis astringents, shortness of breath diffusible stimulants, diarrhœa medicines which check the alvine flux. It is needless, however, to pursue the list of possible complications and to indicate the various methods by which they may severally be relieved.

VII. TUBERCLE.

(Laryngeal and pulmonary phthisis. Tubercular pleurisy.)

Causation.—The ætiology of tuberculosis is a subject of the highest interest, and at the same time one of extreme difficulty. There are few affections in which the influence of hereditary taint is so strongly shown. It is a well-established fact that children of tubercular parents are pre-eminently liable to tubercular affections, and not only so, but that, if one parent be tubercular, the children who most resemble that parent in conformation are usually most prone to be affected; and, further, that parents, themselves seemingly healthy, or at all events free from tubercle, not unfrequently beget a family of children who die one after the other of pulmonary phthisis. In the case last referred to the tubercular tendency of the children may be due either to the transmission of a taint which is latent as regards the parent, or to the fact that one or other parent is scrofulous or syphilitic, or in some other way impaired in health. But tuberculosis does not occur only among those who inherit a tendency to it. Climate has certainly some influence in its production; for it is much more frequent in temperate climates than it is in those which are either very cold or very hot; and Dr. Buchanan's and Dr Bowditch's researches seem to prove that in temperate climates it prevails far more extensively in low, damp situations than it does in such as are elevated and dry. There is no doubt that conditions which produce deterioration of the general health tend ultimately to induce tuberculosis: among which may be enumerated, inadequate nourishment, excessive work with insufficient rest, and want of fresh air. Hygienic defects of this kind are specially injurious to the young. Other causes of tuberculosis are, occupations which necessitate the inhalation of solid irritating particles (for there is no doubt that tuberculosis, as well as cirrhosis, and not unfrequently both in combination, are thus produced), and the cachexiæ which follow or attend upon enteric fever, measles, hooping-cough, syphilis, diabetes mellitus, and various other diseases. No age is free from liability to tuberculosis; it is extremely common in young children, but, putting

these on one side, the age of greatest liability is from twenty to thirty or thirty-five. The influence of sex is uncertain.

Morbid anatomy.—1. *Laryngeal tubercle* always manifests itself in the form of minute grey granulations, which may easily be overlooked, but which, nevertheless, present all the microscopical and other characteristics of grey tubercles. They are situated in the substance of the mucous membrane, and tend after a time to form small round shallow ulcers, which by their coalescence constitute sinuous but rarely extensive tracts of ulceration. It is very common in the course of pulmonary phthisis for the larynx to get implicated; but to what extent this implication, in many cases, is due to actual tuberculosis of the larynx is a matter of considerable doubt. The mucous membrane becomes congested, œdematous, and thickened, and excoriations appear, which sooner or later extend deeply, exposing the cartilages, and causing their erosion. These deep ulcers are most commonly situated towards the posterior extremities of the vocal cords, and involve the anterior processes of the arytenoid cartilages. All the cartilages, however, are liable to be thus affected.

The trachea and bronchi are subject to the same pathological changes as the larynx: their mucous membrane gets congested and thickened, excoriations manifest themselves with or without the pre-existence of miliary tubercles, and occasionally the cartilaginous rings become exposed and eroded, and even detached and expectorated.

2. *Pulmonary tubercle.*—Those who deny the identity between grey and yellow tubercles will, equally with those who maintain the opposite thesis, admit that the two varieties often co-exist in the same individual; and, on the other hand, those who believe in their identity will, equally with their opponents, acknowledge that cases of tuberculosis are not unfrequently met with which are apparently characterised by the exclusive presence of one or other form. It will be convenient therefore, while acknowledging their tendency to pass the one into the other, to describe them independently, as we not unfrequently meet with them in typical cases.

Grey tubercles vary in bulk from mere points up to the size of a small pea, but do not usually exceed that of a pin's head; they are grey, somewhat hard, and slightly translucent; they are sometimes sparsely scattered, sometimes closely set, in some cases distributed with tolerable uniformity, in others forming scattered groups or clusters of various sizes. In the last case, those in the central part of a group coalesce, to a greater or less extent, and form tracts individually as large as a marble or walnut. The development of grey tubercles is occasionally limited to one lung; more frequently it comprises both, and may then involve them equally or unequally. They are in some cases distributed throughout the whole organ, in some limited to certain regions, generally the apex; and for the most part, even when universally distributed, they are most numerous and advanced in the upper

part of the lung. The growth of miliary tubercles is always, in a greater or less degree, associated with other morbid conditions of the lung: these are especially congestion and œdema of the pulmonary tissue, consolidation of the intermediate tracts of lung, and bronchial catarrh mainly implicating the minuter tubules. As regards the consolidation, it must be observed that this may be of the nature of ordinary pneumonia, with impaction of the air-cells with corpuscular elements; or of the nature of cirrhosis, with fibroid thickening of the walls of the air-cells and of the other connective tissues of the lung. A later change is the breaking-down of the consolidated bits of lung and the formation of vomicæ. Such cavities usually commence at the apex, and may be limited to that part. They may vary from the size of a pea up to that of an orange or beyond, and may present every variety of form. They are usually surrounded with a greater or less thickness of indurated tissue, and often present abrupt well-defined margins.

For the most part, miliary tubercles are developed with great rapidity, and tend to a rapidly fatal issue. Occasionally, however, their progress is arrested, and the patient recovers, but with more or less permanent damage to the tissue of the lung. When this happens in respect of discrete tubercles, the organ gets seamed throughout with minute patches of cicatricial tissue, the fibres of which have something of a stellate arrangement, and within the limits of which the lung-tissue presents, from the presence of concurrent emphysema, a coarsely spongy character; and occasionally in the centre of the scars minute fibroid knots or concretions may be recognised. When the affection becomes arrested after groups of tubercles have got consolidated by the intervention of inflammatory overgrowth, more or less extensive tracts of tissue, probably studded with cretaceous or caseous masses and black pigment, assume a cirrhotic character, and contract, while usually more or less emphysema arises in their immediate neighbourhood. Further, when cavities have formed, they either shrink and become lined with a definite smooth membrane, continuous with that of the bronchial tubes, or possibly, in rare cases, get obliterated.

Yellow tubercles in process of development present an opaque, yellowish-white, slightly granular character. They are peculiarly dry and friable, furnishing no juice, but readily yielding, on being scraped or squeezed, a pulpy detritus. They are usually of larger size than grey tubercles, and present for the most part a well-defined outline and more or less irregular form. They evidently comprise groups of air-cells or lobules, and are hence polygonal when cut across; but when divided in the direction of the bronchial tubes, are found to involve the minuter branches of these and to be arranged upon them in a lobulated or foliaceous manner. In their early stage a cross-section will probably have the size of a split tare or pea; they soon, however, partly by individual growth, partly by coalescence, assume

larger dimensions. Occasionally, as the result of such coalescence, large tracts of lung-tissue, possibly the whole of a lobe, become uniformly infiltrated—a condition to which, in the nomenclature of the College of Physicians, the name of 'chronic pneumonic phthisis' has been given.

Yellow, like grey tubercles, usually commence at the apex of a lung, sometimes at the apex of the lower lobe, and gradually spread thence downwards. They are usually, too, more advanced at the apex than elsewhere. It must not be forgotten, however, that they may originate and attain their most advanced stage in any part of the lung. The tendency of yellow tubercles to undergo liquefaction is far more marked than that of grey tubercles; so that, although a lung may become very largely involved without breaking down, in the great majority of cases softening takes place both early and extensively. In one case of rapid phthisis which came under our notice, destructive softening must have been almost coetaneous with the development of the tubercles, for though both lungs were thickly studded with cheesy masses, there was scarcely one of them which was not almost wholly converted into a flocculent-walled cavity. The lungs, indeed, were lighter than natural, and appeared at the first glance to have large air-containing bullæ thickly disseminated throughout their substance. The vomicæ of this form of phthisis usually originate in the upper parts of the lungs, and there attain their chief development. They commence with the liquefaction of those portions of the masses which immediately bound the bronchial passages and smaller tubules; so that, in the first instance, though roundish when cut transversely, they present a dendritic form when the incision takes the course of these channels. A cavity once commenced increases more or less rapidly in size, and ere long, by coalescence with neighbouring cavities, may assume gigantic proportions. It may even occupy the whole of a lobe. Large cavities are usually more or less anfractuous in form, and often crossed by bands of condensed tissue, comprising vessels (mostly impervious) of considerable size. Cavities in process of formation present more or less ragged parietes; but when they have ceased (as they often do cease) to enlarge, their surfaces get smooth, and even polished, and the tissues round them more or less indurated. Yellow tubercle not unfrequently undergoes retrogressive changes. These consist in its gradual conversion, first, into a mortary, and lastly, into a calcareous inert mass, encapsuled by a dense fibroid envelope. The contraction of cavities, the calcareous conversion of tubercular masses, and the induration of the tissues around, are always attended with diminution in the bulk of the affected portions of lung, and compensatory expansion or displacement of the neighbouring healthier tissues.

In both forms of tuberculosis, it sometimes happens that gangrene

takes place; or that profuse hemorrhage occurs either from intensely congested surfaces or from perforation occasionally preceded by aneurysmal dilatation of an artery; or that the tubercular vomica, like any other abscess within the chest, opens into the pleura, or through the outer thoracic walls, or perforates the diaphragm.

3. *Pleural tubercle* differs in no important respect from tubercle of other serous membranes. It appears almost invariably in the form of minute greyish spots variously arranged, sometimes occupying the serous membrane itself, sometimes apparently imbedded in the substance of recently-formed false membranes. These bodies may be scattered over the whole surface, or limited to certain spots; and are generally, even when widely spread, most thickly congregated in certain regions where it may be presumed they originated. They are often specially numerous between the lobes and upon the diaphragm. When very abundant they touch one another, or coalesce so as to form extensive tracts. When this takes place the opposed pleural surfaces are usually adherent, and the tubercular laminæ appear to occupy the substance of the intervening false membrane. As the tubercles increase in size and run together they assume an opaque buff colour and become friable, resembling in look and consistence cheesy masses in the lungs. Pleural tubercle is, in the great majority of cases, associated with tubercle of other organs; occasionally, however, it is primary in the pleuræ, and may even be limited to one. It is very commonly associated with tubercle of other serous membranes; and, as might be supposed, is usually coincident with some amount of similar disease in the lungs. It is nevertheless a fact that it is by no means a frequent complication of pulmonary phthisis, notwithstanding that pleuritic inflammation is an invariable attendant on that affection. Tubercle of the pleura is not necessarily accompanied with inflammation of that membrane; in most cases, however, sooner or later, and sometimes from the very commencement, inflammation takes place, and the usual phenomena of pleurisy then combine with those of tuberculosis,—false membrane is formed, effusion takes place, perhaps suppuration ensues, and indeed any one or all of the various events which have been already fully considered under the head of pleurisy are apt to supervene.

It would be out of place here to enter at any length upon the associated morbid anatomy of tubercular affections of the respiratory organs, which, however, plays so important a part in the progress and symptoms of ordinary cases of pulmonary phthisis. It will be sufficient to draw attention to the fact, that tubercles are rarely limited to these organs, and that their simultaneous development in other organs may induce consequences of much more urgent gravity than those referrible to the laryngeal, pulmonary, or pleural affection. Among the more important complications of pulmonary phthisis are inflammation of the lungs and pleuræ, tubercular meningitis, tubercular peritonitis,

and tubercular ulceration of the intestine, to which may be added fatty and lardaceous degenerations of various organs.

Symptoms and progress.—So much attention has been devoted to the symptomatology of pulmonary phthisis, so much has been written on this subject, and so elaborate are the details with which we have been furnished, that it seems at first sight an almost hopeless task to endeavour to compress our description of the symptoms of the disease within reasonable limits. When, however, we bear in mind that, in most of the elaborate accounts to which we refer, the symptoms of pulmonary phthisis are made to include the symptoms due to tuberculosis of all other organs, those referrible to the many complications which are apt to supervene in the course of phthisis, and besides these the symptoms of the various forms of ill-health which so often precede phthisis, it will be seen that the symptomatology of the pulmonary affection has been overlaid with an abundance of matter which, however important, does not immediately concern us now. The following description will be limited almost exclusively to the symptoms which are referrible to the affections of the respiratory organs themselves.

In a large number of cases the invasion of pulmonary phthisis is remarkably insidious. A patient who has previously, it may be, enjoyed robust health, slowly and without obvious cause becomes weak and thin, probably suffering at the same time from slight remittent febrile symptoms; or, possibly after exposure to the causes of catarrh, he becomes the subject of dry irritating cough which he cannot shake off, and ere long experiences loss of flesh and strength; or he suffers in the first instance from slight symptoms of laryngeal inflammation, which slowly increase in severity; or, without previous warning, he has a sudden and profuse attack of hæmoptysis, on the subsidence of which some of the various symptoms above considered supervene; or a patient, subsequent to an attack of fever, or pneumonia, or in the course of some wasting disease, is attacked with cough, and the symptoms of phthisis gradually replace those of the primary malady. The frequent occurrence of gradual deterioration of health, without the presence of any specific symptoms of disease, prior to the obvious development of pulmonary phthisis, has induced many physicians to believe in the existence of a stage of phthisis antecedent to that of tubercular deposition—a belief, however, based on utterly insufficient data.

But in whatever way phthisis first manifests itself, the symptoms of the fully-developed disease ere long become established. These consist mainly in cough, attended with more or less abundant muco-purulent expectoration, and occasional or frequent hæmoptysis; hectic fever, marked by more or less regularly periodical febrile exacerbations, profuse perspirations, especially at night time, rapid emaciation and loss of strength; and the local evidences, on percussion and auscultation, of progressive involvement and destruction of lung-tissue.

We proceed to discuss the various symptomatic phenomena of phthisis *seriatim*. In a certain number of cases the symptoms of which the patient first complains are referred to the larynx; and it may be that throughout the whole course of the affection the laryngeal symptoms continue chiefly distressing to him. These differ scarcely at all from those of ordinary chronic laryngitis except in their obstinacy and progressive character, and in the gradual supervention of emaciation and loss of strength, and of indications of advancing pulmonary disease. In a still larger number of cases, and indeed in a very large proportion of the entire number of cases of phthisis, laryngeal symptoms of a more or less severe character come on sooner or later in the course of the pulmonary disease. These are sometimes simply irritative or catarrhal, and subside; but more frequently they resemble in all respects, inclusive of their causation and progress, those of the earlier laryngeal affection. It is a question which can scarcely be said to be even now clearly decided, whether laryngeal phthisis (as it is termed) ever actually precedes the pulmonary disease. The general belief is that it is always secondary, and there is no doubt that at post-mortem examinations laryngeal phthisis is never found unassociated with tubercles in the lungs. The laryngoscopic characters of laryngeal phthisis have been described under the head of chronic laryngitis.

The presence of cough is one of the most constant and striking phenomena of phthisis. It generally begins early, and increases in frequency and severity with the progress of the disease. In the beginning it is usually short and hacking, and either dry or attended with scanty glairy expectoration. It is probably then due to slight bronchial irritation only, and the discharge consists of bronchial mucus. With the advance of the disease and the breaking down of the pulmonary tissue, the sputa usually become increased in quantity—often very profuse—and at the same time opaque, yellowish or greenish, and purulent, often nummulated, sometimes fetid. The expectoration is not necessarily distinguishable from that of bronchitis. It is furnished partly by the inflamed bronchial tubes, partly by the tubercular vomicæ; and sometimes, by careful microscopic examination, pulmonary tissue may be detected in it. The cough has no special characteristics by which it may be distinguished from that of bronchitis, or (if the larynx be affected) from that of laryngitis. It presents, however, considerable differences in different cases; in some it is scarcely a matter of complaint from first to last; in some (especially chronic cases) it presents periodical variations, increasing, for example, in the winter or cold weather, subsiding in the summer time; but in the majority of cases it is a serious and increasing cause of distress.

Hæmoptysis is one of the commonest accidents of pulmonary phthisis. It occurs at some period or other in the course of the great majority of cases. Sometimes it is the first indication of the disease;

more frequently it comes on at a later period. It may be only sufficient to tinge or streak the expectoration, or it may be limited to an occasional succession of sanguinolent sputa; or, again, it may be sudden and profuse—the patient bringing up in a very short time half a pint, a pint, or even a larger quantity of blood. It may be so profuse, indeed, at any stage of the disease that the patient is suddenly carried off either by choking or syncope. But these sudden and profuse hemorrhages are usually among the earliest symptoms of phthisis.

Difficulty of breathing is a common but not necessary phenomenon. It may be severe if the larynx be largely affected, or if there be much accumulation in the bronchial tubes, or effusion into the pleuræ. In most cases, however, the patient makes little or no complaint on this score; he no doubt readily loses wind on even slight exertion, and habitually, perhaps, the respirations are more or less augmented in frequency; but when he is at rest his breathing does not usually trouble him.

The patient often suffers from stitch or burning or other kind of pain in the chest. This may occur on one or both sides, often at one apex, but is not limited to any one part. Pain is by no means always present; some patients never experience it; others suffer from it occasionally only; in some cases it is pretty constant and severe. It is usually augmented by movement of the chest, and especially by deep breathing or coughing. It is mostly due to pleuritic complication.

The physical signs of pulmonary phthisis are such as would naturally arise from progressive consolidation and contraction of the lung, the formation of cavities, the accumulation of secretion in them and in the bronchial tubes, and pleuritic inflammation and exudation. In considering the significance of the physical signs we must never forget that, as a rule, tuberculosis commences at one or both apices of the lungs, that excavation usually first takes place in the same situation, and that the morbid processes tend to travel downwards.

The presence of small discrete tubercles in the lungs, even if they be very numerous and close-set, does not necessarily affect the character of the percussion note or the sounds which may be heard on auscultation. We can, therefore, readily understand that pulmonary tuberculosis may have made considerable progress before giving distinct local indications of its presence; and we must not too readily assume, because we hear nothing amiss, that therefore the patient is free from tubercle, or that he is in the so-called 'pre-tubercular stage.' Generally, however, even if there be no dulness, there are bronchitic signs—rhonchus, crepitation, and the like—and these are probably most marked over the upper parts of one or both lungs; or pleuritic friction, or jerky respiratory sounds, which have sometimes been attributed to the presence of circumscribed patches of pleuritic inflammation, may be audible in the same situation. When, however, tubercles have coalesced into masses,

say from the size of a walnut upwards, and abut upon the surface, their presence materially affects the quality of the percussion note over the area to which they correspond. There is then more or less marked dulness on percussion, the extent and completeness of which are determined by the extent and bulk of the consolidated tract. Dulness from tubercular disease is generally indicated by the facts: that it occurs mainly at the apex in front or behind; that it is rarely equal in these situations, and still more rarely equal in the corresponding points of both apices; and that it tends gradually to extend from above downwards so as to involve more and more of the tissue of the lung. In association with dulness there is usually increased sense of resistance on percussion, increase of vocal fremitus, diminished movement during respiration, and more or less obvious flattening. The latter condition is especially noticeable when it occurs beneath the clavicle. The auscultatory phenomena at this stage are mainly those which attend the second stage of pneumonia—tubular breathing, together with (if the tubes contain secretion) rhonchus, gurgling, crepitation, or occasional clicking or creaking sounds, bronchophony, and probably also pectoriloquy. It need scarcely be added that, if the consolidated patch be imbedded in the substance of crepitant lung, little or no indication of its presence may reach the ear. The phenomena which attend the presence of vomicæ are very various and by no means always characteristic. The existence of one or several small cavities in the midst of consolidated tissue does not obviously modify the percussion note due to the consolidation. Large cavities, indeed, surrounded by a thick layer of condensed lung-tissue, generally yield almost absolute dulness. In other cases, however, the formation of a cavity in consolidated tissue is attended with the redevelopment of resonance, which may become almost normal, or may be high-pitched, or present the characters of the bruit de pot fêlé. On auscultation over cavities we may detect (if they contain fluid) large crepitation and gurgling—sounds which may also be heard over the larger bronchial tubes when imbedded in condensed lung-tissue; or (if they be empty) some modification of tubular breathing. Occasionally (and this may be the case in respect of cavities no larger than a walnut) we may hear distinct cavernous sounds. Metallic tinkling is seldom audible over tubercular cavities. In some cases no sounds whatever are produced within a cavity, and all that one hears are normal or abnormal respiratory sounds transmitted from the parts beyond. Both bronchophony and pectoriloquy may usually be recognised; pectoriloquy, however, is on the whole more marked here than over solid lung, bronchophony perhaps less marked.

In delicate patients in whom no obvious consolidation can be recognised, the persistent presence, at one or other apex, of harsh and prolonged expiratory murmur, of a few clicking sounds, of rhonchus, of crepitation, or of jerky respiration, is ground for the gravest suspicion. By some physicians, moreover, a systolic murmur over the pulmonary

artery and its main branches, or in the course of the subclavian artery within the chest, is equally regarded as an indication of the presence of tubercular consolidation—the belief being that the murmurs are produced by the pressure of consolidated tissue upon the vessels in question. They are probably anæmic.

The state and action of the circulatory organs are for the most part such as we meet with in all chronic diseases attended with progressive debility and emaciation. In the earlier stages of phthisis the pulse is usually increased in frequency and hardness; with the advance of the disease its frequency becomes augmented, but there is diminution of fulness and force. With increasing enfeeblement of the circulation it is not uncommon for some degree of anasarca to supervene, especially if the enfeeblement of the left side of the heart be associated, as it occasionally is, with hypertrophy and dilatation of the right side. As a rule, however, the heart undergoes general atrophy. In many cases the anasarca is limited to the lower extremities, and is then often due immediately to thrombosis of the iliac veins. It is doubtless owing to the same enfeeblement of the circulation that various parts, and more especially the nose, ears, fingers, and toes, frequently get congested, livid, and tumid. A clubbed condition of the fingers and toes (although by no means confined to phthisis) is, as is well known, of common occurrence in the chronic form of the disease. Each ungual phalanx becomes swollen and bulbous, and at the same time more or less congested; and in consequence of the grape-like form which it assumes the nail, which occupies the upper half only, becomes bent over the summit, forming a kind of sloping roof.

The symptoms referrible to the stomach and bowels are generally of considerable importance. The tongue may be clean throughout the greater part of the patient's illness; it is often morbidly red, however, and often more or less furred, and towards the fatal termination is apt to get dry, glazed, and fissured or aphthous. There is usually more or less thirst. The condition of the appetite presents great variety. In some cases the patient has a good, and possibly voracious, appetite; in other cases it is capricious; while in others again there is complete anorexia, and probably great irritability of stomach, with gastrodynia, nausea, and sickness. The latter conditions depend in some cases on catarrhal inflammation of the mucous membrane of the stomach, and are often associated with thinning and dilatation of that organ. Phthisical patients are exceedingly liable to suffer from diarrhœa, which is often very obstinate and profuse, and often assumes a dysenteric character. Persistent diarrhœa, indeed, may be the most serious of all the morbid conditions from which the patient suffers. It is due, in the great majority of cases, to coincident ulceration of the bowels—a lesion which complicates fully one-half of the cases of pulmonary phthisis, and which may outrun, if it do not precede, the pulmonary disease. Diarrhœa may result, however, like the dyspeptic symptoms, from mere

catarrh, or some other form of irritation of the mucous membrane. It is a well-recognised fact that tubercular patients are peculiarly apt to suffer from fistula in ano. The presence of a fatty liver is seldom indicated by symptoms, but may occasionally be recognised by the increased bulk which the organ attains.

The nervous system does not usually present any very characteristic morbid phenomena. The patient may be more or less irritable, or, on the other hand, apathetic; he is sometimes desponding, but much more frequently hopeful, buoying himself up even to the last with the prospect of eventual recovery.

Hectic fever and gradual emaciation are by far the most important and striking of the general phenomena of phthisis. They commence in most cases long before the actual proofs of the growth of tubercles exist, and they continue, as a rule, throughout the whole duration of the disease. It is important, however, to observe that phthisical patients often undergo temporary improvement, that under judicious management they often gain flesh and strength, sometimes never lose flesh, and that they not unfrequently remain free from fever for weeks together—sometimes, indeed, have scarcely any febrile symptoms during the whole course of their illness.

The hectic of phthisis is almost typical in the distinctness of the daily remissions and exacerbations which attend it. There is usually some elevation of temperature after food, especially after hearty meals, but the maximum occurs mostly in the afternoon or evening. The minimum temperature in the day may be normal, or even below the normal; the maximum may reach anything from 101° to 104° or even 105°. In most cases, however, the lowest temperature is still considerably higher than natural, and the range less wide than the above figures might seem to indicate. Not uncommonly the temperature falls considerably as the fatal end approaches. The patient usually suffers during the period of exacerbation from heat in the palms and soles, and flushing of the cheeks, which is for the most part vivid and circumscribed, and has received the name of the 'hectic flush.' Perspiration is a common and distressing symptom. The patient complains little of this while he is awake, but when he is asleep sweats are apt to break out profusely all over him, rendering his surface damp and sodden, and his linen and coverings in some cases so wet that the moisture may be wrung out of them. These perspirations are sometimes absent, or they may intermit, or be so slight as to be of little significance.

The emaciation of phthisis is intimately related to the presence of hectic fever, both being the consequence mainly of the rapid disintegration of the corporeal tissues. All parts of the body, doubtless, waste; some, however (especially the brain and nervous system), less than others. The heart dwindles, the bones and muscles become atrophic; but the most obvious change is in the fat, which gradually

and for the most part almost entirely disappears. The limbs and trunk consequently shrink, and their surface falls into wrinkles; the skin of the face and forehead becomes closely applied to the subjacent bones and muscles, so that the cheek-bones, and especially the zygomatic arches, acquire remarkable prominence, and the movements of the muscles get painfully visible. The skin itself in many cases grows thin and brittle, the nails dry and inclined to split, and the hair thin and scanty. In some cases, however, no such changes in the cutaneous organs take place, and indeed the hair occasionally presents extraordinary luxuriance. The extreme emaciation favours the development of bed-sores on the buttocks and elsewhere.

It need scarcely be added that, in a large number of cases of phthisis, the presence of complications already adverted to—more especially cerebral, peritoneal, and renal tuberculosis, and degenerative affections of the liver and kidneys—adds other symptoms to those which have been enumerated, and materially modifies the progress of the disease.

Several varieties of phthisis, which do not necessarily correspond to the several varieties recognised by the pathologist, are described by those who look at the disease from the clinical point of view. They may be ranged under the heads of acute and chronic phthisis.

The great majority of cases of phthisis belong to the chronic category. They commence in one or other of the ways already indicated, and the general symptoms and local changes which have been above discussed become gradually established. In many cases the disease progresses uniformly, no amendment whatever takes place, and the patient sinks probably in from six to twelve months from the first manifestation of symptoms. In some cases the duration of the disease is greatly extended: the patient suffers from the ordinary symptoms of phthisis, or it may be from those of a simple bronchial attack, and then appears to recover more or less completely; but after a while the symptoms recur, and again amendment follows; and again and again, it may be, these alternations of illness and comparatively good health take place, until at length the symptoms of the disease become continuous, and the patient gradually sinks. In such cases the tubercular process probably takes place in successive crops; and it is in them that post mortem we find intermingled, extensive tracts of indurated and contracted lung-tissue, encapsuled mortary or cretaceous masses, cavities with cicatrised parietes, and emphysema. Tuberculosis dependent on the constant inhalation of irritating matters, for the most part takes a similar course. Indeed, when phthisis becomes thus chronic, it approaches, both in its morbid anatomy and in its symptoms, the lung-affection already discussed under the name of cirrhosis. In some cases the progress of the disease becomes permanently arrested; and then, in proportion to the extent to which the lung-tissue may have undergone disorganisation, is the restoration to health complete or incomplete.

In acute phthisis the patient may die in the course of a few weeks, and generally dies within three months. Two varieties of acute phthisis may be distinguished:—first, that in which the tubercles are from the first mainly if not entirely yellow, and in which there is very rapid breaking down of lung-tissue; and, second, that in which the tubercles are miliary. The first variety resembles the ordinary chronic form of phthisis in its symptoms, excepting only their intensity and the extreme rapidity of their development. It usually begins suddenly with high fever and rigors, and pulmonary symptoms which have a close resemblance to those of ordinary pneumonia. It is in fact with this disease that it is especially liable to be confounded. The second variety also comes on more or less suddenly with fever and rigors, and possibly, but not necessarily, some bronchial irritation and cough. The symptoms indeed, both at the onset and for some time—sometimes throughout the whole course of the disease—have a marked resemblance to those of enteric fever with pulmonary complication. The state of the pulse, tongue, and cerebral functions may be identical in the two affections; in both the bowels may be constipated or loose, in both there may be abdominal tenderness and tumefaction; in both the febrile temperature shows marked remissions; in both dyspnœa is apt to supervene, and the face to become ghastly or livid. The motions, however, are rarely peasoup-like in the tubercular disease; the pain in the abdomen is less constant, and, if present, is due to peritoneal tuberculosis, and therefore less likely to be localised in the cæcal region; the temperature does not present the uniform variations characteristic of enteric fever, and there is an absence of the typical typhoid rash. The disease may prove fatal without the development of dulness, and without the evidence of the formation of cavities. Usually, however, as the disease advances, the lungs undergo more or less consolidation, commencing at the apices and extending downwards.

Death in phthisis is due in most cases to asthenia, generally of slow development, but sometimes rapid, and immediately referrible either to extremely profuse alvine flux, or to sudden and copious hæmoptysis. In some cases it may be referred in part or wholly to asphyxia. Such may be the cause of death in phthisis associated with laryngeal disease, or much secretion into the bronchial tubes, or in which there is sudden effusion of blood into the air passages.

There are no symptoms by which tubercular pleurisy can be distinguished from the simple inflammatory affection, apart from its intractable character, and the concurrent or consecutive appearance of tubercles in other organs.

Treatment.—It is of the first importance in the treatment of the early stages of phthisis, and indeed in the treatment of all persons in whom a tendency to phthisis appears to exist, to adopt every available measure to promote the general health, to take every precaution against

the infraction of hygienic laws. It is obvious, therefore, that many of the details of treatment are such as, under the circumstances, common sense would dictate. We may however enumerate a few of the matters here referred to. The diet should be wholesome and nutritious, and fairly distributed among the recognised meals; the patient should keep good hours, refrain from all overwork, whether mental or bodily, clothe himself warmly, and live in temperate, well-ventilated, but not draughty rooms. At the same time he should not refrain from amusement; nor need he abstain from business or other occupations if they be not too absorbing, or of an unhealthy nature; and he should (if the weather be suitable) take an ample amount of gentle out-door exercise.

Good milk and eggs are probably especially valuable as articles of food for phthisical patients, but in their dietary frequent changes and considerable latitude are often necessary; and, further, alcoholic stimulants, though probably not essential, are often apparently very beneficial. For the purposes of bodily warmth flannel should be worn next the skin.

The question of change of air is at this time one of the most momentous that can be raised. Shall the patient leave his home, and if so, whither shall he go, and when? The great desideratum for phthisical patients appears to be a climate of moderate temperature, liable to slight variations only, and neither largely saturated with moisture nor of extreme dryness. It is exceedingly rare, however, for any climate to possess such uniformity of qualities during the whole year, and it is generally necessary therefore, in order to secure the full benefit of climate, to change the locality according to the season. And hence it will be understood that while most fairly healthy inland or seaside places in this country may suit phthisical patients reasonably well during the warmer months of the year, it will probably be necessary to select some sheltered spot upon the South Coast for winter residence; that, while the bracing atmosphere of Scotland or Sweden, or of the higher regions of Switzerland and Tyrol may be exceedingly suitable during the summer, the south of Europe, or the north of Africa, or the Azores or Canary Islands, may be especially beneficial during the winter. There is, however, large choice, and it may be added that a sea voyage is often of great service. But, notwithstanding the enormous benefits that not unfrequently accrue from judicious change of climate, or the permanent removal to a locality which experience may have shown to be specially suitable for the patient, it must not be forgotten that such changes often entirely fail to do good, and that they are altogether uncalled for and useless when the disease is acute in its progress, or far advanced.

In addition to the above hygienic measures, and to the same end, it is generally advisable to have recourse to medicines. No drug with which we are acquainted has any specific influence over the tubercular

process. But there are some drugs which, by improving the general health, tend indirectly to check its progress. Of these iron, quinine, and other vegetable bitters are amongst the most valuable. But there is one article—drug or food—namely cod-liver oil, which during the last thirty years has acquired a special reputation. There is little doubt on the part of practical physicians, none on the part of the public, of the great value of this in the treatment of phthisical and scrofulous patients. It may be given in doses of from a drachm to an ounce three times a day. It is generally advisable, however, to begin with a small dose in order to avoid the production of nausea, and prevent the patient from taking a dislike to it. It is now largely believed that the virtues of cod-liver oil depend simply upon the fatty matter of which it mainly consists; and hence it has been assumed that other fats might prove equally beneficial. The use of cream, neat's-foot oil, olive oil, and other vegetable and animal fats, and of glycerine, has consequently been recommended. A great and sometimes insuperable bar to the administration of food, and to the use of the remedies which have been enumerated, is the irritability of stomach which is so often associated with phthisis. Hence, in a large number of phthisical cases, the condition of the stomach claims our first attention. It is impossible to lay down special rules for their treatment; we must have recourse to some of the various measures which are serviceable in the more ordinary forms of dyspepsia, and above all, perhaps, we must adapt the tonic or combination of tonics we employ to the condition of the patient's stomach.

A great part of the treatment of phthisis usually consists in treating symptoms as they arise. None of these symptoms probably is special to phthisis, and all may be treated in accordance with the rules which guide us under other circumstances. Local pains must be obviated by counter-irritation; laryngeal affections by counter-irritation, by applications to the interior of the larynx, and by inhalation; cough and expectoration, according to circumstances, by expectorants—ipecacuanha, and the like—astringents, or sedatives; diarrhœa by lead or tannic acid, or other of the numerous remedies which check intestinal secretion or assuage peristalsis. It must be added that, for the above and many other purposes, no one remedy is so generally useful as opium in its various preparations; it relieves pain and discomfort, diminishes cough and expectoration, and restrains the action of the bowels. The nocturnal perspirations often defy treatment; to check them it is desirable that the patient should not be heavily laden with bed-clothes, and that his room should be cool. The surface of the body too may be sponged before he goes to sleep. The mineral acids, oxide of zinc, and various other astringent remedies have been largely employed for the same purpose. Also food or wine given in the night shortly before the hour at which perspiration usually occurs seems occasionally to prevent it. With the object of arresting hæmoptysis, the use of ice

and ice-cold drinks and foods, and the administration of astringent drugs, especially digitalis, lead, ergot, and gallic acid, are usually advocated.

In conclusion, it may be pointed out that, while circumstances occasionally arise to render the local abstraction of blood or the use of emetics or purgatives necessary, all depressing treatment is, as a rule, to be eschewed; that during the later period of the disease stimulants are often of extreme value; and, further, that in cases of acute phthisis tonics, cod-liver oil, and change of air are usually equally valueless.

VIII. SYPHILIS.[1]

Morbid anatomy.—1. *Larynx, trachea, and bronchial tubes.* The mucous membrane of the larynx may become the seat of erythematous inflammation during that early period of syphilis in which the skin is similarly involved; and there may even be some excoriation of surface. At a later period, usually, mucous tubercles arise on the laryngeal surface, in common with that of the neighbouring pharynx. These commence as small gland-like elevations, which gradually extend and coalesce. They may appear at any part—on the epiglottis and vocal cords, but especially (according to Dr. Mackenzie) on the inter-arytenoidean fold. They furnish a pretty abundant secretion, and tend to ulcerate—the ulceration extending both in surface and in depth, and leading, according to its situation, to the more or less complete destruction of the epiglottis or vocal cords, and not unfrequently to caries or necrosis of the thyroid, cricoid, or arytenoid cartilages. During the so-called 'tertiary period' of syphilis gummata appear beneath the mucous membrane, involving not merely the connective tissue but the muscles and other parts. In this way tumours of considerable bulk may be developed. These, like other gummata, tend to undergo rapid degenerative changes, and to end in the formation of deep unhealthy-looking ulcers. All syphilitic ulcers when they extend deeply, are apt to involve the destruction of the cartilages, to lead to communication between the larynx and œsophagus, and to lay open arteries sufficiently large to allow of fatal hemorrhage; and all, when they heal, leave dense reticulated cicatrices which not unfrequently produce serious contraction of the channel of the larynx. Syphilitic disease of the perichondrium may arise independently of ulceration or gummata, and lead to necrosis of the cartilages.

Similar affections to those just described may involve not only the trachea, but probably also the bronchial tubes. There is reason to

[1] Syphilitic affections of the larynx and trachea have already been briefly considered in connection with chronic laryngitis.

believe that a form of syphilitic erythema may attack the bronchia as well as the trachea; and there is no doubt that syphilitic thickening of the mucous membrane and gummata of their deeper tissues are not altogether unfrequent in both situations. These affections also in their further progress lead to ulceration, destruction of cartilages, and cicatrisation with contraction.

2. *Lungs.*—Syphilitic affections of the lungs are imperfectly understood. It is well known that syphilitic patients frequently become the victims of pulmonary phthisis, and the question has naturally presented itself whether the apparently tubercular formations in such cases may not be really syphilitic. The question is not altogether easy of solution; for while, on the one hand, we have no reason to believe that the presence of syphilis excludes that of tuberculosis; on the other, we know that there is a close anatomical resemblance between syphilitic gummata and tubercles. There is no reasonable doubt, however, that the lungs occasionally present specific syphilitic lesions; and that these commence with proliferation of the connective tissue of the organ, therefore beneath the pleura, around the bronchial tubes and vessels, and in the interlobular septa; and terminate in the formation of ill-defined patches, in which the tissue is indurated, greyish, and contracted, and converted mainly into fibroid or cicatricial tissue, studded in some instances with caseous or calcareous nodules. The caseous nodules in the lung as elsewhere, though having a close resemblance to tubercles, are usually much less friable than these latter are; the fibroid growth differs little, if at all, from that observed in ordinary cases of chronic or fibroid pneumonia; but Drs. Greenfield and Goodhart[1] point out that in its early stage it is characterised by great vascularity. It may be added, that the above growths are usually associated with syphilitic affection of the bronchial tubes, that they commonly abut on the surface of the lung, and that the pleura covering them is apt to be thickened, seamed and puckered. Virchow and others have described a kind of *white hepatisation* of the lungs of stillborn syphilitic children, the origin of which they refer to the syphilitic poison. The lung, or lungs, or large portions of them, are dense, yellowish-white, opaque, tough, but retaining the impress of the finger. They have been observed in association with syphilitic pemphigus.

Symptoms and progress.—The symptoms of syphilitic disease of the larynx are essentially the same as those which have been ascribed to chronic laryngitis. The affection, however, especially if it be connected with the formation of mucous tubercles or of gummata, is extremely intractable, rarely terminating in complete restoration to health, and frequently leading to one or other of the graver lesions which have been enumerated. Syphilitic affections of the bronchial tubes equally simulate in their symptoms ordinary chronic bronchitis.

[1] 'Transactions of the Pathological Society of London,' vol. xxviii.

With the few clinical facts which we possess in reference to the subject it would be a mere exercise of ingenuity to describe at length the symptoms which may be produced by pulmonary syphilis. It will be sufficient to say that the diseases with which it is most likely to be confounded are chronic bronchitis, chronic phthisis, cirrhosis, and the consequences of these affections. Profuse hæmoptysis would seem to be not uncommon. The presence of a history of syphilis, and of the superficial indications of syphilis, would, of course, furnish an important element of diagnosis.

Treatment.—It need scarcely be said that, in treating syphilitic affections of the larynx, trachea, bronchial tubes and lungs, our main trust must be placed in ordinary anti-venereal remedies.

IX. TUMOURS.

A. *Tumours of Larynx.*

Morbid anatomy.—The larynx is a not unfrequent seat of adventitious growths. 1. Of *non-malignant* kinds the most common are fibromata. These may be simple tumours of a rounded or lobulated form, more or less distinctly pedunculated, and varying, it may be, from the size of a filbert downwards; or they may be similar bodies associated with more or less obvious overgrowth of the mucous glands and the formation of cysts; or they may have a papillomatous or warty character. The last are far more common than the others. They rarely exceed the size of a horse-bean, and are not generally larger than a split-pea. They are, however, often multiple, and tend to spread over a considerable surface. Fibromata usually originate on or near the vocal cords, but are not limited to these localities. They are not unfrequent in children, but may appear at any age. 2. *Malignant* tumours are mostly epitheliomatous, but are sometimes sarcomatous or cancerous. The first of these commences either in the pharynx, involving the laryngeal tissues secondarily, or at some part or other of the surface of the larynx. The others originate among the deeper textures.

Symptoms and progress.—The symptoms to which simple polypi give rise creep on gradually, and probably consist at first in a little hoarseness or loss of voice, with a tendency to clear the throat or cough, and an occasional feeling as if there were some foreign body in the larynx. The phenomena, indeed, are undistinguishable at this time, except by means of the laryngoscope, from those of ordinary subacute laryngitis. At a later period their presence gives rise to more or less complete aphonia, to more or less serious impediment to respiration,

and finally to death from asphyxia. If the laryngeal tumour be pedunculated, the symptoms are apt to vary with its change of position, and especially the patient is sometimes liable to sudden attacks of choking (which may at any time prove fatal) owing to the impaction of the tumour in the rima glottidis. If it be large as well as pedunculated, it may sometimes be seen at the back of the throat without instrumental aid.

Malignant growths commence, like other growths, insidiously, making little show and producing few symptoms; and for awhile, indeed, there may be nothing to cause grave suspicion. Gradually, however, they enlarge, form tumours which from their mere bulk interfere with respiration and other necessary acts, undergo destructive changes which give rise to copious discharges, involve the epiglottis, aryteno-epiglottidean folds, vocal cords and other parts, and perhaps cause sinuses to form in the neck or openings between the larynx and œsophagus. Sometimes they lead to the erosion of arteries and sudden arterial hemorrhage. It must be added that carcinoma, commencing in the glandular or other tissues external to the larynx, occasionally involves that organ in the course of its extension; and sometimes, indeed, when the external tissues are largely infiltrated, the larynx gets imbedded in them and fixed. At first it is difficult, if not impossible, to recognise the presence of carcinoma, even on laryngoscopic examination. The rapidity of the growth of the tumour however, the progressive character and extent of the ulceration which attends it, the fetor of its discharge, and the gradual involvement of the concatenate glands, all tend finally to render the diagnosis clear.

Treatment.—Previous to the use of the laryngoscope, even simple tumours of the larynx were to a large extent fatal. They were then rarely recognised; and being allowed to grow without let or hindrance, ended by asphyxiating the patient. With the laryngoscope, however, their recognition is, even if they be small, comparatively easy, and their cure for the most part a matter of little difficulty. If they be small or pedunculated, they may be removed by means of curved forceps of special construction—for those growing at the back or front forceps opening laterally; for such as grow at the side forceps opening anteriorly and posteriorly. In some cases they may be removed by knife-edged forceps or scissors. It is inexpedient to employ much force, for the mere crushing which follows the attempt at removal (especially if it be repeated) often leads to atrophy of the growth. Occasionally, when the irritability of the larynx is extreme, or the patient is suffering from great dyspnœa, or under other special circumstances, it becomes essential to perform tracheotomy previously to operating on the larynx itself. Astringent and other applications to the mucous membrane are of little or no use. The treatment of malignant affections of the larynx can never of course be curative; occasionally, however, some relief may be afforded by the various local measures

which are serviceable in chronic laryngitis. In any case, tracheotomy is sometimes necessary to prevent death from suffocation.

B. *Tumours of Lungs and Pleuræ.*

Morbid anatomy.—Non-malignant growths of the lungs and pleuræ are of little pathological and still less clinical interest; *malignant growths*, on the other hand, are highly interesting and important. There is probably no form of malignant disease which has not been discovered at one time or other in these organs; and probably each form has (apart from its microscopical characters) special peculiarities as to distribution, progress, and symptomatology. In the present state of our knowledge, however, it would be an excessive and needless refinement to discuss them separately.

Malignant disease either originates within the lungs, or extends to them by continuity from the mediastinum or other adjoining parts, or is secondary to similar disease of some distant organ. In the first case it usually constitutes a solitary mass; in the second it often runs along the bronchial tubes; in the third it is for the most part multiple. These several features are, however, by no means absolutely distinctive. The morbid process begins in the connective tissue, and therefore either in the walls of the air-cells, in the interlobular tissue, in the course of the bronchial tubes and their attendant vessels, or in the thickness of the pleural membrane and subpleural tissue.

When the growth extends along the bronchial tubes the connective tissue which surrounds them becomes infiltrated and thickened, and the tubes and vessels set, as it were, in the solidified tissue. Moreover the walls of the bronchial tubes themselves ere long get involved: in some instances those parts only which are external to the cartilages, in some those only which are internal to them, but for the most part their whole thickness. The affection of the mucous surface manifests itself by the appearance of small, disc-like elevations, which are at first scattered, but soon run together, forming a uniformly elevated slightly translucent tract, from which all the normal longitudinal and other markings are more or less completely effaced. Although the disease commences at the root of the lung, and is for the most part further advanced there than it is elsewhere, its distribution is not always continuous; but tracts of healthy and diseased tissues and healthy and diseased tubes frequently alternate one with another.

Malignant disease, attacking the pleura and subpleural tissue, frequently appears in the form of small greyish lenticular thickenings, which have been likened to drops of tallow or wax. They are sometimes so little prominent and thin as to be scarcely visible; sometimes they form very distinct papular or tubercular elevations. In its further progress, the former variety tends gradually to form tolerably uniform tracts of considerable extent; the latter variety tends to the

production of pedunculated outgrowths, which may hang singly or in clusters into the pleural cavity, and may vary from the size (say) of a pin's head up to that of a bunch of currants, an orange, or a cocoa-nut.

Whenever the morbid growth commences in the tissues which surround the bronchial tubes, or in the subserous tissue, there is a disposition for it to extend into the substance of the lung along the interlobular septa, and consequently for the affected lung to assume some of the characters which are so common in cirrhosis, and to some extent characteristic of it.

In most cases malignant disease of the lungs shows itself in the form of one or more distinct tumours. These, while yet of moderate size, have a more or less rounded form, and if they abut on the surface often assume there the central depression and the tumid margin which are so common in hepatic cancer. When such tumours increase in size, as they usually do, with some rapidity, they become more or less irregular in form; and, from the fact that they are then apt to get blended with the results of inflammation and of pulmonary hemorrhage, their limits are often difficult to define. It must be added that in the progress of extension of malignant disease from the bronchial tubes or pleura, it is not uncommon for distinct tumours to form here and there in the lungs. Malignant growths of the lungs, like those of all other organs, tend rapidly to undergo degenerative changes, and hence soon break down and form vomicæ. Not unfrequently sloughing takes place in them, and masses of the morbid tissue may become detached.

Symptoms and progress.—The symptoms to which malignant growths of the intrathoracic respiratory organs give rise are, for many reasons, exceedingly variable. If the disease affect principally the serous membrane, the symptoms have a more or less close resemblance to those of ordinary pleurisy; if the bronchial tubes be its main seat, the symptoms naturally approximate to those of bronchitis; if tumours form, they may, if sufficiently large, afford the physical indications of consolidation; and if they undergo softening, of the presence of vomicæ, and may be attended with more or less abundant purulent or bloody, and probably fetid expectoration. Still it must not be forgotten that malignant growths, in the early period of their formation, not unfrequently give no sign whatever of their presence, and that the patient may seem in fair, if not in his ordinary robust, health, up to the sudden supervention of some inflammatory complication or of hæmoptysis.

Malignant disease affecting the pleura generally ere long excites some inflammatory action, and the formation of false membrane, with temporary stitch and friction sound audible during respiration, and is probably always, sooner or later, attended with effusion of fluid. This effusion usually gives rise to no symptoms beyond those due to the compression which it exerts. It tends so to accumulate as to distend

the pleura, and is persistent, always returning and generally with rapidity after paracentesis. It is mostly mere ordinary dropsical serum. In some cases, however, it is distinctly inflammatory; and it may be purulent. But it not unfrequently happens, especially if the adjoining lung-tissue be implicated, that it is mixed more or less copiously with blood, or that it is stained with altered blood-pigment, being green, yellow, or brown, or that it is glairy like the fluid from an ovarian cyst.

When the bronchial tubes and the parts surrounding them are the chief seats of disease, the symptoms, as before indicated, are mainly bronchial. Assuming that the patient has never previously suffered from bronchitis, the symptoms would be characterised by their insidious approach; but when so far developed as to be a source of distinct discomfort they would differ little if at all from those of progressive subacute or chronic bronchitis in some of its phases. There would probably at first be some difficulty of breathing, increased by exertion, with little or no distinct local evidence of lung-disease, or at most a little localised sibilant or sonorous rhonchus. With the advance, however, of the malady, secretion would take place from the affected tubes, and cough, with more or less abundant expectoration, would be added to the other phenomena.

Small malignant growths within the lungs, even if numerous, yield no distinctive physical indications of their presence. They may, however, give rise to congestion, œdema, or inflammation in their vicinity, and may hence become revealed. Larger tumours are often solitary, and limited therefore to one locality; but they may involve the whole of one lobe, or even more, of a lung. These, like smaller growths, are often associated with other local morbid conditions, which increase their apparent bulk and add to or aggravate the patient's symptoms.

When malignant tumours are of sufficient size to be recognisable by physical signs, we find, in many cases, that there is dulness on percussion over the region which they occupy, and that there is also a total absence of healthy respiratory sound, of tubular breathing, of all forms of rhonchus and crepitation, and of bronchophony, pectoriloquy, and vocal fremitus. The local indications, indeed, are rather those which we are accustomed to regard as characteristic of pleural effusion than those which we usually associate with consolidation. The explanation of this peculiarity lies in the fact that malignant growths usually form solid masses, the bronchial tubes which permeate them being compressed or otherwise obliterated; and that hence they are as distinct acoustically from the lung-tissue as is an accumulation within the pleura or the heart itself. There are many exceptions, however, to this rule—some due to the presence of surrounding inflammatory changes, some to the continued patency of tubes and to the greater or less abundance of secretion in them, some to the form-

ation of vomicæ. The presence of malignant tumours in the lungs necessarily causes, sooner or later, more or less dyspnœa and cough, with expectoration which varies in its character—being sometimes at the beginning mere bronchial mucus, but later on becoming muco-purulent, and at length purulent, hemorrhagic, and fetid. Occasionally the sputa are glairy, and green, yellow, or brown—exactly like the fluid which is perhaps accumulating at the same time in the pleura. The general symptoms of malignant disease of the lungs are, for the most part, those of gradually increasing debility and emaciation, often associated with those of hectic fever, the patient at length dying exhausted, and sometimes before death passing into the typhoid state. Death may be due, however, less to the progress of the cancerous growths than to the secondary phenomena, such as pleural effusion and bronchitic obstruction, to which they give rise; and hence it may occur at a comparatively early period of the disease, or, at all events, before the opportunity for accurate diagnosis has presented itself.

It has been assumed, in the foregoing account, that the disease has primarily or mainly implicated the lungs or pleuræ. It need scarcely be added that, when the thoracic affection is secondary to more advanced disease elsewhere, it very often fails to reveal itself by symptoms. Further, it will be readily gathered that the diagnosis of pulmonary or pleural malignant disease is often a matter of extreme difficulty—indeed, in most cases, it can only be arrived at by a very careful collation of all the facts of the case, and by close and continuous observation of the patient's progress. The presence of malignant growths elsewhere of course furnishes a most important clue. This clue is not likely to fail in cases in which the pulmonary affection is secondary and comparatively unimportant. It may be absent, however, from those cases in which its aid is most needed. Nevertheless, careful examination should be made from day to day, for very often indeed in the progress of such cases enlargement of glands at the root of the neck, in the axilla or thoracic parietes, or development of growths in connection with the ribs or connective tissue of the thoracic walls becomes manifest, and throws a new and important light upon phenomena which were hitherto obscure.

Treatment.—The treatment of malignant disease of the lungs and pleuræ needs no special description. The treatment, already considered, of pleuritis, bronchitis, and pneumonia includes in some measure that of the several affections which have just been discussed—with the important reservation that all that can be done for malignant disease is palliative, and that therefore lines of treatment which may be pushed with advantage in the case of the inflammatory affections must be cautiously followed in respect of their malignant counterfeits. The relief of pain and discomfort, and the administration of nourishment, should be our chief aims.

X. PARASITES. HYDATIDS.[1]

Morbid anatomy.—The only parasitic disease of the lungs which has any practical interest is that due to hydatids, and this is of exceedingly rare occurrence in England. It appears to be common in Australia. There is usually only one tumour present, and this is said to be generally situated in the lower part of the right lung. The hydatid tumour may attain the size of a large cocoa-nut or exceed it; may be situated (as it usually is) wholly within the substance of the lung; or, originating beneath the visceral pleura, may form, as it were, an outgrowth from the lung into the pleural cavity. The walls of the cyst which contains the parasite vary in thickness and density. The parasite is usually solitary, containing echinococci but not secondary cysts. Hydatids of the lungs, like hydatids elsewhere, are liable to cause serious consequences, either by the mere pressure which they exert, or by undergoing suppuration, or discharging their contents into the pleural cavity or bronchial tubes; they are also liable to undergo degenerative changes with gradual contraction and calcification.

Symptoms and progress.—Hydatid tumours of the lungs, when small, occasion little or no uneasiness, and necessarily therefore fail to be discovered. But as they enlarge they are after a while apt indirectly to cause bronchitic symptoms and occasional attacks of more or less profuse hæmoptysis, and thus a series of symptoms which are exceedingly liable to be mistaken for those of phthisis. Their recognition may be rendered possible either by the special features of the tumours to which they give rise, or by the sudden expectoration of their contents. An hydatid cyst is always tense, and of a globular or ovoid form, and hence, when it attains any considerable size, tends, on the one hand, to cause a circumscribed bulging of the thoracic parietes with widening and more or less protrusion of the corresponding intercostal spaces, and, on the other hand, to displace the mediastinum and diaphragm. The localised bulging of the chest-wall, with the possible detection of fluctuation and hydatid thrill, and the circumscribed dulness on percussion which probably transgresses the median line of the thorax, without extending at the same time to its summit, are strongly indicative of the presence of a pulmonary hydatid, especially if these conditions have come on without febrile or acute symptoms and the general health of the patient has been, and remains good. There will also be over the area of dulness total absence of respiratory sound and vocal fremitus. It is stated that the voice sometimes presents at the margins of the tumour an ægophonic character. It need scarcely be

[1] For fuller details in reference to the pathology, diagnosis, and treatment of hydatids we must refer the reader to the account of these parasites given further on amongst the diseases of the liver.

insisted that the signs above enumerated are not absolutely pathognomonic of hydatids; they may be equally present (occasionally at least) in cases of circumscribed empyema or solid tumours. The rupture of an hydatid cyst and the escape of its contents into the bronchial tubes is attended with sudden suffocative cough and profuse expectoration, which may, according to circumstances, be limpid and watery, or purulent, and may contain echinococci, hydatids, or fragments of hydatid membrane. The detection of the parasites, either by the naked eye or by the microscope, necessarily removes all doubt as to the nature of the case.

Hydatid tumours not unfrequently undergo cure—sometimes by spontaneous retrogression, sometimes after the discharge of their contents by the bronchial tubes or some other route. On the other hand, the patient may die either suffocated by the sudden irruption into the air passages, or exhausted by long-continued suppuration, or from the rupture of the cyst into the cavity of the pleura and consequent empyema.

It is in many cases impossible to distinguish accurately between hydatid tumours of the base of the right lung and those developed in the upper part of the right lobe of the liver. For the latter often protrude far into the chest, displacing the diaphragm and base of the lung upwards; and cause marked bulging and dulness of the corresponding region of the thorax. Moreover they are very apt to perforate the diaphragm and to open either into the pleural cavity or into the lung itself, and in the latter case to discharge their contents by expectoration. When, however, hepatic hydatids are expectorated, biliary colouring matter is usually sooner or later mingled with them. On the other hand, hydatid abscesses of the base of the lung may perforate the diaphragm and so lead to the formation of abdominal abscesses.

Treatment.—There is little room for special treatment in the case of pulmonary hydatids. When, however, they are recognised as obvious tumours, they may be treated on the same principles as hydatids of the liver—a subject fully considered further on.

XI. BRONCHIECTASIS. (*Dilatation of Bronchial Tubes.*)

Causation and morbid anatomy.—It is theoretically an easy matter, but practically very difficult, to draw the line between simple dilatation of bronchial tubes and vomicæ which have become lined with a smooth membrane, and are in direct continuity with tubes. We make this distinction incidentally only, for our description of dilated tubes will embrace without distinction the several morbid conditions which are commonly confounded under the term.

Dilated tubes then (using the term in its widest sense) may be arranged in three categories; namely, 1st, that in which the dilatation involves the tubes in their whole length, and is consequently cylindrical or moniliform; 2nd, that in which it affects only the terminal portions of the tubes, and assumes a globular form; and 3rd, that in which the expansions are large, more or less irregular in shape, and communicate with one or more bronchia.

A. In the first of these varieties, the dilatation, which usually commences in tubes of the first or second order, is continued thence, with little or no interruption, through the succeeding tubes almost to their terminations in the air-cells. It is generally relatively greater in the smaller than the larger tubes; the larger tubes indeed often being scarcely implicated, while the smaller measure from $\frac{1}{4}$ to $\frac{1}{2}$ inch or more in diameter. Hence the affected canals form, in some cases, a series of hollow cylinders of nearly uniform calibre; in other cases a series of channels which actually increase in diameter towards their distal extremities, where probably they undergo in addition sudden and considerable enlargement. The dilatations are rarely, however, entirely uniform, but generally present a somewhat irregular or moniliform condition. The parietes of the dilated tubes vary in character; sometimes they are generally thickened, sometimes exceedingly thin and delicate, sometimes the mucous membrane is fairly normal, sometimes thick, congested, and pulpy. In the larger tubes it often happens that the fibrous and muscular bands stand out in strong relief, the interstices being correspondingly deep, and occasionally forming distinct pouches. This form of dilatation seldom involves both lungs at the same time, and still more seldom the whole of one lung. It is most frequently met with in the lower and middle lobes, and even then is usually restricted to certain of their tubes only. The dilated tubes are in some cases surrounded by fairly healthy, in some by emphysematous, in some by collapsed, lung-tissue. Cylindrical dilatation of the bronchial tubes is probably always secondary to chronic bronchitis, and more especially to capillary bronchitis with abundant accumulation of fluid; and is due primarily to the concurrence of inflammatory enfeeblement of the bronchial walls, and their distension by accumulated fluid contents.

B. In the second variety, the expansion is limited almost exclusively to the terminal portions of the smallest bronchial tubes and bronchial passages. The dilatations are globular or nearly so in form, and vary perhaps from the size of a small pea up to that of a filbert; they present a smooth internal surface; their parietes are comparatively thick, dense, and opaque; and for the most part they communicate severally with a single bronchial tube, the orifice of communication with which is sometimes small and difficult to find. In some cases such dilatations are scattered irregularly throughout emphysematous or otherwise diseased lung-tissue; in some cases they are grouped more

or less thickly in some corner of a lung; or they involve the whole of one lobe, or even of a lung. The lower lobe is most frequently affected. In the best-marked cases the whole of a lung or of its lower lobe is diminished in size, and riddled with globular cavities which are separated from one another by airless, collapsed, indurated lung-tissue. The history of these cases is usually very obscure; but there is good reason to believe that they originate, not in ordinary chronic bronchitis, but in either atelectasis, collapse, lobular pneumonia, or some other such condition. And it is probable that, in the first instance, the accumulation of muco-purulent fluid or of pus within the terminal bronchial tubes leads to the destructive ulceration of their walls and in a greater or less degree of the tissues which surround them; that thus small cavities or vomicæ, communicating with tubes, are formed within the solidified tracts of lung-substance; and that finally each cavity attains a certain size, assumes a definite form, and becomes lined with a membrane continuous with that of the bronchial tubes.

C. The third variety comprises those cases in which the lung contains one or more cavities, irregular in shape, and various but often considerable in size, each of which opens freely into one or two bronchial tubes. In some instances these cavities are solitary, situated in the apex, base, or elsewhere in the substance of the lung, and surrounded by or imbedded in dense fibrous or cicatricial tissue. In other instances the whole of one lobe, or the whole of a lung, contracted and cirrhosed, is studded with them. They are often lined with a perfectly smooth, polished membrane, continuous with that of the communicating bronchial tubes, but sometimes exhibit a more or less eroded or flocculent surface. In some cases they and the bronchial tubes connected with them present a thick, pulpy, deeply-congested lining, which may perhaps be undergoing excoriation. These cavities are not of course, strictly speaking, simple dilatations of tubes; but obviously originate in vomicæ—tubercular, pneumonic, gangrenous, or other. They are always surrounded with a greater or less abundance of indurated tissue, and in a considerable number of cases are associated with obvious or advanced cirrhosis.

The determining cause of the several forms of bronchiectasis which have been considered is no doubt disease involving the bronchial tubes themselves—disease attended with softening or destruction of their walls, and distension of their channels from undue accumulation of fluid within them. Yet it cannot be denied that other influences co-operate in some degree with these, the most efficacious of them being the constant tendency of a retracted side to recover its form, partly by its inherent elasticity, partly under the influence of the inspiratory efforts. It must be remarked, however, that this latter influence can scarcely be operative unless the lung-tissue generally be indurated and reduced in bulk, and that in spite of it many vomicæ contract, and some possibly cicatrise.

Symptoms and progress.—It will probably be gathered from the foregoing account of the morbid anatomy and causation of bronchiectasis that its special symptoms are in almost all cases necessarily mixed up with those of the maladies out of which it arises and with which it is associated. Moreover, its own symptoms are not very characteristic. Those which may more especially be looked for are shortness of breath and cough, with abundant muco-purulent expectoration, not unfrequently mingled with blood, and occasionally fetid with a fetor which is little, if at all, distinguishable from that of gangrene. This is believed to be due to decomposition of the retained sputa. The physical signs which probably present themselves are more or less retraction and immobility of one side or of a portion of one side of the chest, dulness on percussion, with large crepitation or gurgling, and other indications of the presence of vomicæ. It must not be forgotten, however, that difficulty of breathing may be scarcely noticeable if one lung continue perfectly healthy, and that distinct indications of the presence of cavities are not likely to be observed if the cavities be small or deeply-seated and surrounded with comparatively healthy tissue. Profuse and repeated attacks of hæmoptysis, due to intense inflammation and congestion of the lining membrane, or to its erosion, are not uncommon in the last of the three varieties of bronchiectasis, and occasionally cause death. The diagnosis of dilated tubes rests on a careful consideration of the history of the case, on the slow progress of the pulmonary lesion, or its limitation to one lung, and often to its lower half, and on the profuseness, long continuance, and often fetor of the expectoration.

Treatment.—No special treatment is required in bronchiectasis. For the most part the rules which have been laid down for the treatment of chronic bronchitis are applicable here.

XII. EMPHYSEMA.

Causation and morbid anatomy.—Emphysema is due to the dilatation and rupture of the air-cells of the lungs. Two forms of it have been described, namely, the *interlobular* and the *vesicular*: in the former of which air is extravasated into the interlobular spaces, whilst in the latter the dilated and ruptured vesicles communicate simply with one another.

A. *Interlobular emphysema* takes place only in those cases in which the pulmonary lobules are separated from one another by distinct intervals occupied by connective tissue. This anatomical peculiarity exists in the lungs of young children and of some of the lower animals, but is absent from the lungs of human adults. Interlobular emphy-

sema is, therefore, a phenomenon of childhood only, and is occasionally observed, post mortem, in cases of hooping-cough, lobular pneumonia, and capillary bronchitis, especially where there have been repeated violent paroxysmal attacks of coughing. The escape of air into the interlobular spaces is indicated anatomically by their marked dilatation and their occupation by what looks like strings of air-bubbles. The amount of emphysema present varies greatly. In some cases, on section of the lung, merely a line or two of emphysematous tissue is observed. In other cases the lobules are more or less extensively isolated from one another by a network of such tissue, and a similar mapping-out of them may be recognised on the surface of the lung. Occasionally it happens that the emphysematous condition extends along the connective tissue which surrounds the bronchial tubes to the root of the lung, and thence diffuses itself into the mediastina, neck, face, trunk, and elsewhere. The latter phenomenon is more frequently the result of gross mechanical injury than of infantile pulmonary disorders; and under such circumstances it is observed in adults as well as young children.

B. *Vesicular emphysema* is a much more common and, on the whole, a much more serious affection than the last. It occurs, with certain differences of character, under a variety of conditions. Thus it is not unfrequently met with in the lungs of persons dead of acute bronchitis; it is an all but universal attendant on the chronic form of that disease; it is one of the lesions which often accompany old age; it is a frequent associated phenomenon of the degeneration of tubercular masses, or of the contraction, from whatever cause, of circumscribed bits of lung; and it is also, as we believe, an occasional sequela of the obsolescence of scattered miliary tubercles. Under most of these conditions there has been persistent cough; under several of them, enfeeblement of the walls of the air-vesicles or their involvement in destructive processes.

1. The lungs of persons dead of acute bronchitis are often found much distended with air. They completely fill the portions of the thoracic cavity allotted to them, and do not collapse even after they are removed from the body. The air-vesicles are generally distended to the full, and the lung-tissue consequently is light, pale, and unusually spongy. This condition is always associated with obstruction of the bronchial tubes by secretion, and is primarily due to the comparative facility with which air is then drawn into the ultimate tissue of the lungs during inspiration, and the comparative difficulty with which it is then discharged during expiration. The appearance here referred to is very commonly regarded as an indication of emphysema. It is obvious, however, that there can be no disease of the air-cells, no vesicular emphysema, so long as the air-cells are simply distended and their walls remain sound. But when, as often happens at length, textural changes are superadded, when the walls of the air-cells become attenuated, bulging, perforated, and intercommunications

between neighbouring cells established, emphysema is really present. Nevertheless, it is not always easy, either with the naked eye or with the microscope, to distinguish between mere over-distension of the air-cells and the beginnings of actual emphysema.

2. The lungs of patients who have long suffered from chronic bronchitis are almost invariably emphysematous in a greater or less degree. When the emphysema is advanced and well-developed, the lung is usually of large size and exceedingly irregular in form—the increase of size and the irregularity being both due to the formation of clusters of emphysematous vesicles, which cause lobular, and in some cases pedunculated, protrusions from various parts of its surface. These are usually most abundant upon the anterior and outer surfaces of the lung, and especially upon its diaphragmatic aspect. If the lung be full of air, the prominence of these clusters is unmistakable, and the relatively enormous size of their vesicles obvious on even casual inspection. On section, the emphysematous tissue collapses much more readily than the healthier tissue around. If the distended lung be dried, before section, the distribution of the emphysema, not only at the surface but in the interior of the lung, may be easily recognised. The emphysema of chronic bronchitis has its origin, no doubt, in the emphysema of the acute disease, and comparatively slowly attains extreme development; and, like the last, it consists essentially in the attenuation and perforation of the walls of the air-cells, the distension of the multilocular cavities thus produced, and the atrophy and disappearance, to a large extent, of the capillary networks of the affected regions. The emphysematous blebs may, roughly speaking, vary in size from that of a tare or pea up to that of a filbert or walnut.

3. It is not uncommon to find a certain amount of emphysema in the lungs of old people who have never suffered materially from bronchitic symptoms. The emphysema here, however, is rarely extensive, and is often limited to the formation of fringes of emphysematous vesicles along the anterior and other sharp edges of the several lobes, and to the appearance of solitary vesicles scattered thinly over the general surface in connection with black pigmentary patches. When tubercular and other indurations and contractions take place in relation with any part of the pulmonary surface, so as to cause irregularity, puckering, and fissuring, well-marked emphysema (differing in no respect, except distribution, from that of chronic bronchitis) almost invariably manifests itself in the immediate vicinity. This variety of emphysema is most common at the apex. Again, emphysema arises in general but comparatively slight chronic disorganisation of the lung-tissue, general slight cirrhosis, or the obsolescence of widely distributed miliary tubercles. In these cases, as has been previously pointed out, the lung-texture is coarse, permeated by a fine network of what looks like cicatricial tissue, in the meshes of which the dilated and ruptured air-cells are contained.

The ætiology of emphysema is a subject of much interest, and has largely occupied the attention of medical men. The theories which have been proposed in reference to it may be divided into three groups: the first of which attribute it to the dilating force exercised during the act of inspiration, the second to the pressure exerted during the act of expiration, the third to nutritive impairment of the walls of the air-vesicles.

1. When we consider that the healthy lung is accurately adapted to the cavity which contains it, and that no enlargement of the chest voluntarily effected can possibly dilate injuriously the healthy lung within it, it is impossible to conceive that the emphysema of at least uncomplicated bronchitis can be referred to the force with which the lungs are inflated. Matters are, however, somewhat different when a lung, reduced in bulk by pleuritic adhesions or textural changes, is subjected to the rhythmical efforts of the corresponding thoracic wall to expand. It is conceivable then that while those parts of the lung, which are most firmly compressed or most dense from consolidation, would undergo little or no consequent change, those which are comparatively little compressed, or which are still fairly crepitant, might undergo disproportionate expansion, their air-cells might dilate, and their parietes become attenuated and finally ruptured. This indeed is the explanation commonly assigned to that form of emphysema which is developed in the vicinity of obsolescent tubercular masses and other patches of contracted indurated lung-tissue. It may be observed, however, on the other hand, that when, in lungs bound down and compressed by dense adhesions, portions of lung where the adhesions are thinnest expand and protrude beyond the general level, emphysema is certainly not commonly observed.

2. During ordinary expiration, when the lung-texture retains its normal elasticity, and the bronchial and other passages are freely permeable, the air-cells are subjected to very little pressure. In all those acts, however, in which with closed glottis the expiratory muscles are called into vigorous action, the pressure on the surface of the air-cells and air-passages becomes augmented, and in some cases very greatly augmented. The acts to which we here refer are, in ascending order, speaking and shouting, the blowing of wind instruments, the expiratory shocks of coughing, and the straining efforts which attend defæcation, parturition, and other forms of violent muscular exertion. But in these cases, again, if the passages are permeable the pressure is uniformly distributed, and injury to air-cells is little likely to result unless some of them have been previously impaired as to their power of resistance by morbid changes. The conditions, however, are altogether different when violent expiratory efforts are made, and made habitually, in the presence of obstructive disease of the bronchial tubes, whether the obstruction be due to accumulation of mucus or other matters within them, to thickening of their lining

membrane, or to their compression by morbid growths external to them. For it is easy to see that then while, during violent expiratory efforts, large portions of the lung whose tubes are little obstructed become comparatively empty, other portions (scattered probably among them) whose tubes are more completely obstructed remain distended, and become in consequence unduly exposed to the compressing force exerted by the thoracic parietes. For equal extents of surface a globe holds more than any other solid figure: it is obvious, therefore, that if a full globe be compressed it must either yield or burst. The same remark applies to a distended air-cell or a group of distended air-cells; and hence it is obvious that the effect of the expiratory compression of groups of full air-cells in connection with obstructed tubes must necessarily result in the expansion and attenuation of their walls, and sooner or later probably in their rupture. It is doubtless to this cause in large measure that the emphysema of bronchitis and the interlobular emphysema of childhood are due.

3. Many years ago Mr. Rainey demonstrated the occurrence of fatty degeneration in the walls of the dilated air-cells in some cases of emphysema. We have already alluded to the occurrence of emphysema in connection with cirrhosis and obsolescent miliary tubercles. But indeed it will be admitted without further formal proof that the walls of air-cells are liable to get weakened, and to lose their ready distensibility, under these and many other conditions, such as old age, and various forms of inflammatory and other processes. It is too obvious to need argument that enfeeblement of the walls of the air-cells must co-operate powerfully with the mechanical causes already discussed in the production of emphysema, and must, especially when of partial distribution, render even normal violence of expiration a source of danger.

In concluding this brief discussion of the causes of emphysema it must be added that, although we have considered them separately, they probably always act more or less in concert. Thus, before the expiratory acts can cause compression and rupture of groups of air-cells, these must have been distended with air by more or less powerful efforts of inspiration; and, even if the air-cells were previously healthy, their mechanical distension, attenuation, and laceration must result in more or less serious impairment of their powers of resistance for the future.

Symptoms and progress.—Emphysema in its slighter degrees is often present without causing distinct symptoms. Thus, in the limited emphysema which attends the obsolescence of tubercle, and in that which comes on in old age, there is often absolutely no sign to indicate its presence; and, even in the emphysema of bronchitis, there are often at first, and even for a considerable time, no phenomena apart from those of persistent bronchitis to justify its diagnosis. When, however, emphysema becomes considerable either in degree or in

extent it can scarcely escape recognition, notwithstanding the fact that even then its symptoms are mainly those of bronchitis.

The physical signs of advanced emphysema comprise alterations in the form and movements of the chest, together with percussive and auscultatory peculiarities. The chest tends to get dilated in all its dimensions, rounded from above downwards as well as horizontally, the ribs at the same time acquiring a less oblique direction than in health; the form which it assumes is that to which the term 'barrel-shaped' has been applied. The shoulders become elevated, and the muscles of expiration (more particularly those of the neck and shoulders) unduly prominent. At the same time the limits of the respiratory movements become narrowed, the chest during expiration retaining still the distended or barrel-like condition, and during inspiration undergoing little enlargement. On percussion there is usually increase of resonance over the chest, and more especially over those portions of it which correspond to emphysematous areæ; and the præcordial dulness is diminished in extent, or even abolished. The respiratory sounds are enfeebled.

The symptoms referrible to the lesion are shortness of breath, increased by exertion, and culminating at times in asthma-like attacks; duskiness and pallor of skin, and disposition on the slightest exposure to suffer from bronchitic attacks. Further, there is usually feebleness of pulse, and tendency to emaciation. The presence of any considerable amount of emphysema interferes seriously with the readiness of transmission of the blood through the lungs, and hence gradually the right side of the heart becomes hypertrophied and dilated, the cervical and other systemic veins get distended with blood, the circulation is impeded in the capillary vessels of the extremities and internal organs, and general anasarca, congestion of liver with jaundice, and congestion of kidneys with albuminuria, supervene. Indeed the consequences of emphysema are almost identical (excluding congestion of the lungs) with those of mitral valve disease.

Treatment.—The treatment of emphysema must necessarily be indirect. We cannot, either by drugs or otherwise, restore the affected air-cells to their former condition. But what we can do, in many cases, is by suitable medicinal and hygienic treatment to cure or relieve the bronchitis which is so often associated with it, to prevent the recurrence of such attacks for the future, to relieve cough and dyspnœa, and to promote the general health. In addition, we may forbid all violent exercise and over-exertion. Easy circumstances, early hours, wholesome but not excessive diet, gentle exercise, warm clothing, a genial climate, together with the careful avoidance of everything calculated to give cold, constitute the main elements in the successful management of those who suffer from emphysema. For the treatment of its complications we must refer to what is said under their respective heads; and especially we must refer to the articles on bronchitis and asthma.

XIII. CONGESTION.

Causation and morbid anatomy.—Congestion always coexists with inflammation. It frequently occurs, however, under other circumstances; and it is this variety that we propose now to consider.

1. *Congestion of the larynx, trachea, and bronchial tubes.*—Simple hyperæmia of these organs may result from obstructive disease of the right side of the heart or any other condition which causes accumulation of blood in the veins which lead from these parts, or it may be a sequela of repeated attacks of inflammation.

2. *Congestion of the lungs* is an expression in constant use, but is probably generally applied to cases of bronchial or pulmonary inflammation. Simple congestion, however, is of frequent occurrence, and is often a serious complication of other morbid conditions. It is an early and grave consequence of mitral valve disease, and other affections of the left side of the heart. It is a frequent complication of typhus and other infectious fevers, and even of the typhoid state. It doubtless also persists in some cases for a longer or shorter period as a sequela of inflammation. Congestion secondary to heart disease is for the most part general, and is often followed by rupture of the small vessels of the lungs and extravasation of blood. It leads consequently to the condition known as pulmonary apoplexy and to hæmoptysis. Further, it predisposes to pneumonia. The congestion which takes place in fevers is commonly termed 'hypostatic,' from the fact that it particularly affects the dependent, and generally therefore the posterior and lower, parts of the lungs. It is due, like the congestion which in the same affections occurs in other depending parts of the body, to feebleness of circulation and the disposition which the blood then has to yield to the influence of gravity. The affected portions of the lungs become dark, almost black, with congestion, œdematous, more or less devoid of air, heavy and lacerable: but they maintain their bulk, and generally crepitate in some degree. Not unfrequently this condition passes into pneumonia, and even gangrene. Congestion of the lungs, whether in connection with heart disease or febrile states, is always a matter of serious importance, partly because it indicates that there is something in the stage or nature of the malady which threatens danger, partly because it gives rise itself to serious symptoms, partly because it is apt to end, as the case may be, in pulmonary apoplexy, gangrene, or inflammatory consolidation.

Symptoms.—Of itself congestion of the air-passages is probably unimportant, leading at most to some increased secretion from the mucous surface and its glandular follicles, and to habitual tendency to cough and hawk, especially on rising in the morning. It is chiefly serious because its presence greatly increases the liability to inflam-

mation. The evidences of congestion of the lungs are mainly difficulty of breathing, with more or less lividity, and other asphyxial phenomena, cough with watery expectoration, and the presence of crepitating râles either generally distributed, or limited to or more pronounced in the lower and back parts of the lungs. The supervention of apoplexy, pneumonia, or gangrene, will be recognised by their special indications.

Treatment.—Very little can be done directly to relieve these mechanical congestions. It is sometimes, in cardiac cases, useful to remove blood by leeching, cupping, or venesection, or to act on the bowels or other emunctories. But, for the most part, all we can do is to treat the heart affection on the principles elsewhere laid down, and the febrile disorder according to its general indications.

XIV. DROPSY. HYDROTHORAX.

Causation and morbid anatomy.—Œdema constantly occurs as an incident of the inflammatory process, tuberculosis, and the growth of malignant tumours. Œdema of the larynx, which is so often a cause of death, is almost without exception the result of inflammation either of the larynx itself or of parts in its immediate neighbourhood; pneumonic consolidation of the lung is usually attended with more or less œdema of the lung-tissue surrounding the consolidated district; and pleurisy is rarely, if ever, unaccompanied by serous effusion into the pleural cavity. Independently of inflammatory dropsy, however, which does not now concern us, there are other forms of dropsy, for the most part of mechanical origin, which are sufficiently important in their effects to call for a few remarks. Dropsy of the respiratory organs, like dropsy of other parts, may be due, equally with congestion, to certain forms of cardiac disease and other affections involving obstruction of veins; it may arise, also, in the course of renal disease; and occasionally originates in mere anæmia.

1. *Œdema of the larynx* may occur in the course of renal and heart diseases, and in those cases in which, from the presence of mediastinal tumours, the vena cava descendens, or the innominate veins are obstructed; and possibly may, even in its uncomplicated state, cause dangerous symptoms and death. Generally, however, when serious symptoms arise, inflammation has become superadded to the dropsy; and indeed the supervention of inflammation doubtless constitutes the chief danger of this limited dropsy.

2. *Œdema of the lungs* is a frequent accompaniment of general anasarca, from whatever cause; but is especially common in scarlatinal affection of the kidneys and other forms of Bright's disease; and may

occur in a high degree, even when there is little or no dropsy observable in other parts of the body. An œdematous lung is usually voluminous, heavy and pale, and on section large quantities of serous fluid mingled with air-bubbles drain away or may be squeezed out. It contains much less air proportionately to its bulk than natural, but is rarely quite devoid of air in any part. If the lung be at the same time congested, it combines the characters of congestion and œdema. In hypostatic congestion, which has been already described, there is usually such a combination. Œdema, like congestion, sometimes results in a form of pneumonic consolidation, and not unfrequently in a condition of carnification or collapse.

3. *Pleural dropsy*, or *hydrothorax*, occurs under the same circumstances as œdema of the lungs, and is very often associated with it in the sense of being complementary to it. That is to say, hydrothorax which is a serious and frequent item of general dropsy (whether cardiac, renal, or pulmonary), and tends for the most part to arise whenever œdema of the lungs tends to arise, serves nevertheless mechanically to exclude the latter condition, and conversely. Thus, if there be much pulmonary œdema there is probably little pleural effusion; if much pleural effusion, little pulmonary œdema; and if there be coincidence of pleural and pulmonary dropsy on the same side, the latter is limited to that portion of lung which lies above the level of the pleural exudation, the rest of the lung probably being compressed, and void both of air and of excess of fluid. And further, it often happens that, if one lung be bound down by adhesions, the other being free, there is pleural dropsy exclusively upon the one side, pulmonary œdema exclusively upon the other. Occasionally hydrothorax becomes so considerable that it distends the pleural cavity and causes total collapse of the lung.

Symptoms.—The symptoms of œdema of the larynx, so far as they are important, have already been fully considered. Those of œdema of the lungs are nearly identical with those of congestion of the same organs. There is gradually increasing difficulty of breathing with lividity of surface and other symptoms of defective aëration of the blood—symptoms which need not be rediscussed, but which, if not relieved, increase in severity until death takes place. There is usually also, sooner or later, more or less cough, with expectoration of thin serous fluid. The presence of œdema does not necessarily entail local indications; the chest may be perfectly resonant, the breath sounds as nearly as possible healthy. Still, more or less crepitation may generally be recognised, especially behind and below; and with the occurrence of consolidation the indications of that condition are necessarily developed.

The general symptoms to which pleural dropsy gives rise are in the main those of pulmonary œdema. The local signs are those which have already been described as indicative of the presence of fluid in the

pleural cavity (page 390). It may be observed that whenever difficulty of breathing and other signs of slowly developing asphyxia arise, in the course of renal or cardiac disease, or of suspected general tuberculosis, or malignant growths, it is important to examine the thorax carefully in reference to the probability of pleural dropsy being present. And further, in relation to this point it is worth while to remark that there is often much more fluid in a pleural cavity than might be suspected from casual examination. For in determining its presence and amount, the patient is usually made to sit up in bed and bend his trunk forwards—a position which necessarily throws the fluid forwards and reduces the height of the level of dulness behind in relation to the landmarks which usually guide us.

Treatment.—The treatment of dropsy of the larynx, lungs and pleuræ, resolves itself into that of the diseases which give rise to it. We must refer, therefore, to observations made elsewhere upon the treatment of inflammations and tumours of these organs, and upon that of heart disease, bronchitis, and albuminuria. It may be pointed out, however, that when, even in advanced heart or kidney disease, or other affections, the patient with dropsical accumulation within the pleura is suffering much from dyspnœa, great (even though it be merely temporary) relief may often be given by the performance of paracentesis. The removal of even a few ounces of fluid by the aspirator may be sufficient for the purpose.

XV. PULMONARY COLLAPSE. ATELECTASIS.

Causation and morbid anatomy.—Under a considerable variety of circumstances, more or less extensive portions of the lungs become void of air and shrink, assuming an appearance to which the term *carnification* has been commonly applied.

A. The simplest form of this condition is observed in a lung or part of a lung compressed by accumulation of fluid within the pleura. It is much reduced in bulk, wrinkled and livid on the surface, uniformly smooth, dark, and flesh-like on section (the bronchial tubes and larger vessels being compressed equally with the vesicular texture), tough but flabby in consistence, heavier than water, and capable of being reinflated through the bronchial tubes which lead to it.

B. A second form is that which is frequently observed post mortem in the lungs of patients dead of bronchitis, fevers, or other affections in which, from debility or blocking up of some of the smaller bronchial tubes, respiration has been incompletely performed. Here the collapse does not as a rule involve any considerable continuous tract, but affects

scattered groups of lobules, which are usually most abundant and largest in the lower portions of the lungs. In such cases the lungs present considerable irregularity of surface—depressed livid-looking polygonal patches alternating with elevated tracts of comparatively pale and crepitant tissue. On section the depressed areæ are found to correspond to dark-coloured, airless, smooth, tough, carnified patches, which extend to various depths into the substance of the lung. In many cases a few such patches of collapse only are visible; in others the collective bulk of collapsed lung-tissue is very considerable.

Frequently, no doubt, the condition of the affected bits of lung here referred to is identical with that of lungs compressed by pleural effusion; and in nearly all cases inflation may be readily performed. It must not be forgotten, however, that such collapse often takes place in lungs which are already congested or œdematous, when of course the shrinking is less pronounced, and the tissue is more juicy and lacerable than in simple collapse. Nor must it be forgotten that when the collapse is a complication of bronchitis, there is a tendency for it to pass into, or to be associated with, lobular pneumonia; and that then patches of apparently pure collapse are often associated with patches of distinct lobular pneumonia and others presenting gradations between these extremes. The main physical distinctions between lung-tissue consolidated from collapse and pneumonic lung are: the shrunken condition, the smooth, homogeneous, shiny, reddish-black sectional surface, and toughness of the former, the expanded, granular, lacerable, and, for the most part, pale reddish or greyish marbled aspect of the latter.

The explanation of the production of the form of collapse now under consideration is not far to seek. Dr. Gairdner's theory is simple, and has found general acceptance. According to it, the smaller tubes get more or less completely filled with mucus, which acts in each case as a valvular plug, preventing the passage of air beyond it, during inspiration, by becoming then more tightly wedged in the narrowing tube, but allowing the escape of air from the implicated air-cells during expiration by being then driven into the wider portion of tube above. It is not quite clear, however, that mucus is generally capable of performing this twofold action; nor is it essential that it should so act in order that collapse shall be produced. The simple but complete and prolonged obstruction of a small tube is amply sufficient to cause collapse of the portion of lung to which it leads—the obstruction preventing both ingress and egress of atmospheric air, and the tissues beyond gradually absorbing that which is imprisoned within them.

C. A third form of what may also be conveniently termed collapse is known by the name of '*atelectasis*.' This is really the persistence of the fœtal state of lung, in which the condition of things is little, if at all, distinguishable from what obtains in simple collapse.

Symptoms and progress.—In discussing the morbid anatomy and symptoms of collapse, it is so usual to confound it with lobular pneumonia (with which it is frequently associated), that the result is less an account of collapse than of inflammation. Collapse is, for the most part, a mere consequence of other lesions, and gives rise to but few distinctive symptoms. Nevertheless, there are some cases in which, doubtless, its occurrence aggravates the patient's symptoms and diminishes the chances of his recovery. This is more especially the case in bronchial affections, hooping-cough, measles, and the like, occurring in young children. For in them, owing to the yieldingness of the thoracic walls, perfect inflation of the lungs under difficulties is often impossible, the flexible framework of the chest failing to respond to the efforts of the inspiratory muscles. The symptoms to which extensive collapse may be expected to give rise are in the main those which attend congestion and œdema, namely, gradually increasing dyspnœa, with the other consequences of defective aëration of the blood. We have already, in discussing the subject of bronchiectasis, adverted to the fact that collapsed lung-tissue, whether the collapse be of the nature of atelectasis, or due to pressure, or bronchial affection, may remain permanently solid, and pointed out that there is good reason to believe that it is in such conditions that bronchiectasis with globular dilatations not unfrequently takes its origin. It may be added that extensive collapse of lung-tissue is necessarily associated with depression and comparative immobility of the thoracic parietes in relation with the affected tracts; and that hence in young children there not unfrequently results permanent impairment of the form of the chest, the lower half usually becoming contracted in the horizontal plane, while the upper remains normally, or even becomes abnormally expanded.

Treatment.—Pulmonary collapse, apart from the bronchial and other affections which attend it, and the permanent consequences which it sometimes entails, scarcely calls for special treatment.

XVI. HEMORRHAGE. PULMONARY APOPLEXY. HÆMOPTYSIS.

Causation and morbid anatomy.—Hemorrhage from the respiratory organs may be of two kinds:—first, that in which the blood is yielded by some part of the air-passages or cavities in direct continuity with them; and, second, that in which it takes place primarily into the substance of the lungs.

1. The former kind is due to either congestion, inflammation, ulceration, or injury; and may occur in the course of simple or ulcera-

tive inflammation of the larynx, trachea, or bronchial tubes; or attend syphilitic, carcinomatous, or tubercular affections of the same parts; or take place during the process of detachment of the membranes of diphtheria or plastic bronchitis, of the discharge of hydatids or of the opening of abscesses. It may be due also to the rupture of an aneurysm into the trachea, bronchial tubes, or pulmonary vesicles. In these cases the hemorrhage takes place, either from numbers of minute vessels, or from one vessel of comparatively large size which has undergone ulceration or rupture.

If the blood thus effused be small in quantity, it is usually mingled with the sputa in the form of spots or streaks. If it be more abundant, it becomes more generally diffused throughout their mass. When profuse it not unfrequently accumulates in the cavities or tubes which yield it, or finds its way by the effects of gravity or of inspiration into healthy tubes, and even into those of both lungs. Under the latter circumstances it is apt to coagulate and form solid casts of the channels in which it lies. It is said that blood yielded by the bronchial or other passages may be sucked into the air-cells, and thus cause pulmonary apoplexy. This we are not disposed to admit.

2. The second variety of hemorrhage takes place into the interalveolar texture of the lungs and air-cells; and the extravasated blood tends partly to be expectorated, partly to accumulate in the tissues, producing the condition termed '*pulmonary apoplexy*.' This form of hemorrhage attends pneumonia, but is rarely excessive in that disease, or productive of apoplexy. Pulmonary apoplexy is much more common in lobular pneumonia and pyæmia. Its most frequent cause, however, is heart disease, attended with impediment to the passage of blood through the left cavities, or with extreme feebleness of circulation. Further, pulmonary apoplexy not unfrequently complicates Bright's disease in its advanced stages, embolism of the pulmonary artery, and those cases in which, whether from debility or other causes, there is a disposition to the formation of coagula in the vessels generally.

Pulmonary apoplexy is indicated post mortem by the presence of patches of lung tissue of various sizes, from that of a pulmonary lobule up to that of a hen's egg, or larger, which are for the most part distinctly limited by the margins of the outermost of the affected lobules, are distended like pneumonic tissue, are of a dark, reddish black colour like coloured clots unexposed to the atmosphere, contain little or no air, yield a small quantity of sanious perhaps frothy serum on pressure, and are heavy, and more or less brittle or lacerable. The patches which may be present display great variety, both in number and in size; and they may occur in any region of either lung; but are more common below than above. The presence of pulmonary apoplectic effusions is usually associated with that of moulded adherent clots in the arterial branches leading to them, to the formation of which

thrombi, indeed, there is reason to believe that they are often, if not always due. Apoplectic clots undergo decolourisation, as does blood effused into the subcutaneous tissues; and if small may disappear, leaving behind them some brownish granular pigment only. Sometimes they soften and break down; and generally they induce inflammatory mischief in the lung-tissue around them and the pleural surface upon which they abut. The surrounding inflammation is sometimes pretty extensive, and the blending of the two morbid conditions may make it difficult or impossible to determine the relation between them, or to define their respective limits.

3. Besides the above varieties of hemorrhage there are others dependent on constitutional disorders which may affect indifferently the parenchyma of the lungs, and the mucous membrane of the air-passages, or are of uncertain origin. Such are the hemorrhages, which attend purpura, typhus, small-pox, diphtheria, and similar diseases, that which is said to occur vicariously of menstruation, and that due to diminished atmospheric pressure.

Symptoms and progress.—It is not usually a difficult matter to determine whether blood voided by the mouth is derived from the respiratory organs or not. The facts that it is expelled by coughing, and has a florid frothy character; the detection by auscultation of crepitation in one lung, or part of a lung; and (if its source be apoplectic) the recognition of dulness and of the other local indications of pulmonary consolidation, will of course be important aids to diagnosis. Further, careful enquiry into the history of the patient, and examination of his thoracic organs will, in a large proportion of cases of hæmoptysis, reveal the presence of organic pulmonary, cardiac, or arterial disease. At the same time we must not forget to look to the nose and fauces, in order to be sure that the blood is not yielded by these parts, and especially to determine, so far as may be, the condition of the œsophagus and stomach. Still it is quite possible to make mistakes in spite of the most anxious care; and it is important, therefore, to bear in mind the following considerations:—first, hemorrhage may take place from the lungs, and yet no other evidence of thoracic disease be present; second, it may, especially if it be from the larger air-passages, be so sudden and profuse that the blood pours from the mouth without any effort at coughing, and even with more or less of the sensation and appearance of sickness; third, although the blood of hæmoptysis is usually described as frothy and scarlet, when it escapes in large quantities its colour may be that of blood in any other form of hemorrhage, and devoid of marked frothiness; and when it has lain in bronchial tubes or cavities, or has been derived from apoplectic effusions, it not unfrequently is dark-coloured or almost black, or of a more or less dull chocolate or coffee-ground hue; fourth, in profuse pulmonary hemorrhage blood may be swallowed in considerable quantity and subsequently vomited or passed by stool, while, on

the other hand, in hæmatemesis or epistaxis, blood sometimes finds its way into the air-tubes, and is discharged thence by coughing. When pulmonary hemorrhage is slight, as it generally is in bronchitis, pneumonia, and pulmonary apoplexy, the symptoms to which it gives rise are unimportant; when, however, it is profuse, as it often proves in phthisis or carcinoma, or when an artery or vein is perforated or an aneurysm ruptured, the symptoms and prospects are in the highest degree grave. The patient frequently dies suddenly, either choked by the violent outburst, or rendered syncopic from the loss of blood; or sinks at an early period from the effects of repeated hemorrhagic attacks.

Treatment.—Many of the varieties of pulmonary hemorrhage do not call for special treatment, and indeed, in the great majority of cases, the hemorrhage must be attacked through the disease to which it is due. This rule applies in great measure even to cardiac and phthisical hæmoptysis. When, however, the discharge is profuse, or threatens to become profuse, or in any way specially dangerous, it calls for prompt treatment. The patient should be kept perfectly still, in the recumbent posture, and every means of quieting the circulation should be adopted; no exertion, not even that of speaking, should be permitted; he should be placed in a cool room, and be but little oppressed with bed-clothes; ice-bags may be applied to his chest; his food should be unstimulating, and but small in quantity; he should have cold drinks or ice to suck, and for medicinal remedies such as quiet the circulation or contract the smaller arteries, among which may especially be named lead, gallic acid, digitalis, ergot, and turpentine. Local or general bleeding may, in some cases, be justifiable. The treatment of internal hemorrhage is, however, always eminently unsatisfactory.

XVII. PNEUMOTHORAX.

Causation and morbid anatomy.—The presence of air in the cavity of the pleura is probably always referrible to the existence of some communication between that cavity and the external atmosphere. Thus, it may be due to a wound, such as is made in paracentesis or when the pleura is punctured in the carelessly performed operation of tracheotomy; or to the opening into the pleura of an abscess already communicating with the exterior, such as one in the parietes of the chest, or an hydatid or other abscess of the liver. Its most frequent causes, however, are the discharge of an empyema through the lungs or thoracic parietes, and the opening of a tubercular or other pulmonary abscess into the pleural cavity. It is said to be caused sometimes by the rupture of emphysematous vesicles, sometimes by the decomposition of fluid and solid matters occupying the pleural cavity.

Pneumothorax, even if it do not commence from empyema, is probably always followed sooner or later by inflammatory effusion into the pleural cavity, and by the formation of pus. If it affect a circumscribed portion only of the pleura there is little or nothing by which it can be distinguished practically or clinically from a pulmonary vomica. The most striking cases are those in which a sudden and free communication takes place between the lung and a pleural cavity which had previously been healthy. Then air is admitted into the cavity with each inspiratory act, and, not being again expelled, accumulates at the expense of the lung, which undergoes gradual compression, of the mediastinum which becomes displaced to the opposite side, of the diaphragm which is pushed downwards, and of the outer thoracic parietes, which become distended and immovable. The accumulation of air acts, indeed, mechanically in precisely the same way as the accumulation of fluid.

Symptoms and progress.—The symptoms which mark the occurrence of pneumothorax are, for the most part, more or less sudden pain or uneasiness in the affected side, but especially the sudden supervention of severe and increasing dyspnœa. They are identical, as nearly as may be, with those of dropsical accumulation, but are much more rapid in their development. It need scarcely be added, however, that when the pneumothorax supervenes on empyema, or occurs within a limited space, no special symptoms may be developed. The physical signs have been already considered. They are mainly, in addition to distension and immobility of the affected side, hyper-resonance on percussion, absence of respiratory murmur, with cavernous and metallic sounds, and diminution of vocal fremitus. The supervention of empyema, and the accumulation of fluid in the pleural cavity, lead to the production of additional physical phenomena which have already been sufficiently described.

Treatment.—The treatment of pneumothorax is in the main that of empyema. It may, however, be pointed out that, when intense dyspnœa comes on rapidly, it may be necessary to remove the accumulated air by paracentesis.

XVIII. PARALYTIC AFFECTIONS OF THE LARYNX.

These affections are very various in their origin. They may be due to mere functional disturbance, as in hysteria and loss of power of phonation from sudden fright or other mental disturbance; to diphtheria; to cerebral disease; to lesions of the pneumogastric trunk, or of the superior or recurrent laryngeal nerves. Further, from various causes, atrophy of one or more of the laryngeal muscles may take place.

1. *a. Bilateral paralysis of the superior laryngeals* is a rare affection,

and usually due to either hysteria, bulbar paralysis or diphtheria. The superior laryngeals are the sensory nerves of the larynx, but supply motor branches to the circo-thyroid muscles exclusively, and, in conjunction with the recurrents, to the arytenoidei. They are also, according to Von Ziemssen, the motor nerves of the depressors of the epiglottis. The consequences of their paralysis would therefore be, anæsthesia of the larynx, inability to depress the epiglottis, and inability also to make the vocal cords tense. The symptoms would be some degree of hoarseness of voice with incapability of uttering high notes; and, in consequence of the association of loss of sensation with failure of the epiglottis to descend, tendency for the food to enter the rima glottidis, but without the immediate spasmodic choking which naturally attends that accident. Under laryngoscopic examination, we should expect to see perfect execution of the movements of adduction and abduction.

b. Unilateral paralysis of the superior laryngeals may result from the implication of the nerve in various morbid processes. The symptoms would be of the same nature as those observed in the double affection, but less pronounced, and of course the anæsthesia would be limited to one side.

2. *a. Bilateral paralysis of the recurrent laryngeals* arises from the same causes as induce paralysis of both superior laryngeals. The recurrent nerves supply all the intrinsic muscles of the larynx, with the exceptions above specified. There is therefore in this affection loss of power mainly in the muscles which open and close the glottis. Under the laryngoscope therefore the cords will be found to lie nearly motionless, midway between their position in complete closure of the glottis and that which they occupy when the glottis is widely opened. They do not approximate in phonation, but they tend mechanically to fall together during deep inspiration. The symptoms of the disease are: complete aphonia, with undue expenditure of breath during forcible expiration, as when the patient attempts to phonate or coughs; and inability to cough or expectorate with vigour. There is absence of dyspnœa, at any rate during quiet breathing.

b. Unilateral paralysis of the recurrent laryngeals is usually referrible to intra-thoracic disease or tumours occupying the lower part of the neck, especially to aneurysm and carcinoma; occasionally also to enlargement of the thyroid body. It is an affection of tolerable frequency, and of great significance. When it is present the affected vocal cord remains motionless midway between the position of closure and that of complete patency, while the opposite healthy cord tends during phonation to pass beyond the median line, and thus to render the rima glottidis oblique. The voice loses its clearness and purity, and tends to break into a falsetto when the patient tries to speak loud. He is doubtless also liable to some degree of occasional dyspnœa. Indeed, it is generally held that one of the consequences of this form

of paralysis is the occurrence from time to time of spasmodic attacks of intense difficulty of breathing and cough, which are apt to prove fatal. This, however, we believe to be an error, and are ourselves disposed to refer the dyspnœal attacks which are so common in these cases to concurrent compression of the trachea, which is often induced by the same affection as induces the paralysis.

3. *a. Bilateral paralysis of the pneumogastric nerves* is most frequently observed in hysterical patients, and may be induced in them by the occurrence of slight laryngeal inflammation, or under the influence of emotion. It may also be a sequela of acute laryngitis or diphtheria, and of sudden fright, horror, or grief. It is probably never complete, and rather reveals itself by imperfect action of the muscles than by their entire immobility. The symptoms, which include aphonia and more or less breathlessness, need no detailed description. Its duration, curability, and liability to recur depend in great measure on its cause. As, however, it is almost invariably functional, a complete cure may generally be anticipated.

b. Unilateral paralysis of the pneumogastric nerves, that is, palsy involving loss of sensation as well as loss of motion on the affected side, is rarely if ever met with as an element of ordinary hemiplegia. It has been observed, however, in the case of tumours involving the nucleus of origin of the nerve in the medulla oblongata, and may be induced by the accidental division of its trunk in the neck, or its implication in the progress of morbid growths, above the giving off of the laryngeal branches. When the disease is in the medulla oblongata there must almost necessarily be involvement either of the hypoglossal or of some neighbouring nerve, or other paralytic phenomena—a combination which would probably render the diagnosis comparatively easy. In other cases, the existence of some lesion on the corresponding side of the neck would probably explain the nature of the laryngeal affection. The symptoms would be almost identical with those of paralysis of the recurrent laryngeal. Anæsthesia would probably be overlooked, unless attention were specially directed to the possibility of its presence.

4. *a. Bilateral paralysis of the posterior circo-arytenoidei* is of very rare occurrence, and its causes are very obscure. It may come on at any age. These muscles open the glottis; and the consequence of their paralysis is that the adductor muscles being unopposed tend to transform the glottis into a narrow chink, which narrows itself still further during inspiration. The essential symptoms of the affection are the gradual development of a purely inspiratory dyspnœa, generally without catarrh or disturbance of voice. At first the inspiratory stridor comes on only when some unusual bodily exertion is being made; but it gradually becomes permanent, even when the patient is at complete rest. It is especially obvious when he is asleep. At the same time expiration is performed without difficulty, and the voice

remains clear. The disease is always chronic, and rarely if ever curable. Tracheotomy is almost always needed ultimately.

b. Unilateral paralysis of the circo-arytenoidei.—In this case the inner border of the affected vocal cord stands in the median line. The voice is somewhat impure; but it is only during forced inspiration that coarse loud-sounding vibrations are produced.

5. *Paralysis of the lateral circo-arytenoidei (adductors)* causes much the same symptoms as paralysis of the recurrent laryngeals. The vocal cords (one or both) remain wide open; there is absolute aphonia and undue expenditure of air in speaking and coughing.

6. *Paralysis of the arytenoideus.*—This muscle draws the arytenoid cartilages together. When, therefore, it is paralysed, the part of the rima glottidis bounded by the arytenoid cartilages remains open during phonation, forming a triangular chink; while the vocal cords in the anterior two-thirds come into absolute contact. The result is that unvocalised air escapes through the chink when the patient speaks loud, and the quality of the voice becomes impaired or hoarse. This affection is usually the consequence of acute catarrh, but is rare.

7. *Paralysis of the thyro-arytenoidei.*—These are more commonly affected than any of the other muscles of the larynx, in catarrh, over-exertion in speaking or singing, and hysteria. Their paralysis is indicated by loss of tension in the vocal cords, so that when they are brought into apposition an oval slit is still left between them. The symptoms are hoarseness and impurity of voice.

Treatment.—In the treatment of functional paralysis, the general health of the patient must be carefully considered, and as far as possible improved. But local treatment is especially important. For this purpose counter-irritation externally, and stimulating applications to the mucous membrane, are often useful. No local measures are so generally efficacious as the application of galvanism. This may be effected by placing the electrodes on either side of the exterior of the larynx, or (with the aid of Dr. Mackenzie's or some other suitable apparatus) one within and one in contact with the skin over the situation of the thyroid cartilage. The treatment of other forms of paralysis is involved in that of the conditions which give rise to them. They are often incurable.

XIX. SPASM OF THE LARYNX AND TRACHEA.

1. *Larynx.*—Spasm is chiefly known as causing contraction of the rima glottidis. It is rarely an independent affection, but is of common occurrence as a complication of other disorders. It is an essential element in hooping-cough, spasmodic croup, and the true epileptic

seizure; it is readily induced by the inhalation of irritating vapours, or the entrance of solid or fluid matters into the larynx; and it is frequently associated in a greater or less degree with inflammatory affections of the larynx. It may also be a phase of hysteria. Prolonged spasmodic closure of the glottis, or laryngismus stridulus—a kind of epileptic convulsion—is occasionally fatal in young children.

2. *Trachea.*—That spasmodic contraction of the trachea may take place is a physiological fact. How far it is a matter of any importance is another question. We allude to it here, however, because we believe that when aneurysmal or other tumours are compressing the trachea and inducing from time to time spasmodic attacks of dyspnœa, the immediate cause of difficulty of breathing is not unfrequently spasmodic contraction of the muscular tissue of the compressed portion of the trachea, associated, it may be, with more or less hyperæmia and accumulation of mucus.

Treatment.—For the relief of laryngeal spasm the following measures may be serviceable:—namely, the application of leeches, counter-irritants, or hot fomentations to the upper part of the chest in front, or to the neck; or the employment of hot baths, while cold water is dashed into the face; and, for internal use, emetics, sedatives, and the inhalation of chloroform. Tracheotomy may be necessary.

XX. ASTHMA. (*Spasm of the Bronchial Tubes.*)

Definition.—The term asthma is often applied loosely to various forms of difficulty of breathing, and indeed, is very commonly employed to designate the dyspnœa which attends ordinary chronic bronchitis and emphysema, or cardiac disease, or that which is due to the pressure of tumours upon the trachea. We use the term here in its more correct and restricted sense to indicate a specific affection, characterised by the periodic recurrence of general contraction of the bronchial tubes and consequent dyspnœa.

Causation.—Asthma is not unfrequently inherited—asthmatic parents begetting asthmatic children. It has also been observed occasionally to have a similar relation with epilepsy and other spasmodic nervous disorders. It affects males about twice as frequently as females. It may make its first appearance at any period from birth up to extreme old age; but most commonly commences during the first ten years of life. The first outbreak is often traceable to an attack of hooping-cough, measles, or bronchitis; but, in a large proportion of cases, no such explanation of its origin can be discovered. When, however, patients have become asthmatic, paroxysms of dyspnœa may be excited by a wide range of conditions, which are unequally operative in different cases,

and some of which are not improbably capable of originating the disease. Of these some appear to act directly, others indirectly, upon the bronchial tubes. Of the former, Dr. Hyde Salter gives a long and interesting list, which includes: the inhalation of smoke, dust, or pungent vapours; the smell of cats, dogs, horses, rabbits, or other animals; the scent of the rose, privet, or other flowers; the emanations from new-mown hay and powdered ipecacuanha; change of weather, the prevalence of particular winds, and the presence of fog. But the most curious and mysterious of such causes is simple change of locality. Thus, some asthmatics suffer most in a dry, some in a moist atmosphere, some at a high, some at a low elevation, some in inland localities, some by the seaside, some on one side of a street of which the opposite side is innocuous to them. But most find themselves better in London or other large towns than they are in the country; and, as a rule, a moist air is more suitable for them than a dry air, a low site better than an elevated site. Among the conditions which may be supposed to act indirectly are the ingestion of certain articles of diet (which, however, differ so much for different asthmatics that it would be useless to quote examples), distension of the stomach, constipation, disease of the brain, and violent emotions. Dr. Salter considers that when particular articles of food cause asthmatic attacks, they act after absorption, and hence directly on the mucous membrane of the bronchial tubes.

Symptoms and progress.—The asthmatic paroxysm usually comes on without warning. In some cases, however, it is preceded for a shorter or longer time by premonitory symptoms, which are different for different cases, but mostly uniform for each case. These are, sometimes abnormal buoyancy of spirits, sometimes mental depression, sometimes drowsiness, but most frequently a slight degree of the asthmatic state, manifesting itself by tendency to wheeze, constriction across the chest, sense of flatulence, alteration of carriage, and the like. Among the occasional earlier phenomena of asthma are a tendency to pass an abundance of pale limpid urine, and (as Dr. Salter points out) a peculiar troublesome itching under the chin, not relieved by scratching.

The attack may come on at any hour, but is almost always uniform, or nearly so, as to the time of its supervention in each case. It sometimes occurs an hour or two after dinner, sometimes as soon as the patient lies down in bed, but in the great majority of cases between two and four o'clock in the morning, probably after the patient has had a comfortable sleep. There is no doubt (as Dr. Salter observes) that the forenoon is in every respect the most favourable time for asthmatics; their attacks least frequently commence then, and when on them are apt at that time to undergo some remission.

The symptoms of the asthmatic paroxysm are mainly those of intense dyspnœa. The patient is probably roused from sleep with the symptoms full upon him; or else, after a certain time of discomfort passed between sleeping and waking in battling with his augmenting

dyspnœa, wakes to the full consciousness of his condition. He is then compelled to rise from his bed—baring his chest and throwing aside everything that hampers his respiratory movements—in all the agony of impending suffocation. The phenomena of the fully-developed paroxysm are, for the most part, as follows:—The sense of suffocation is terrible; the patient's whole energies are devoted to the performance of the respiratory acts; his breathing is not rapid, often, indeed, slower than natural; but it is effected with the most violent efforts; his mouth is open, his nostrils dilated, his shoulders elevated, his head thrown back; the respiratory muscles harden and stand out, and he places himself in some constrained position which gives them leverage—standing or sitting with his elbows resting on the table or some other elevated ledge and his head buried in his hands, or grasping some unyielding object, generally above his head; he places himself even in the depth of winter at the open window. The expression of his face is one of intense anxiety; the lines are strongly pronounced; his eyes are congested and protruding, his surface pale and ghastly, or livid; copious perspirations break out upon his face, head, and trunk, while his arms and legs, and especially his hands and feet, become cold; the pulse is rapid, small, feeble, and sometimes irregular. The dyspnœa is peculiar: inspiration is comparatively short, expiration greatly prolonged, and both are attended with loud wheezing; no interval exists between expiration and the following inspiration. The chest cavity is greatly expanded; it is large and rounded from elevation of the ribs, elongated from depression of the diaphragm; and this expansion is maintained even at the end of expiration—the fact being that the chest is abnormally distended with air, and that the powerful action of the respiratory muscles effects very little movement in its parietes, and consequently very little interchange of air. Dr. Salter points out, indeed, that this over-distension of the chest begins to take place even before the appearance of manifest dyspnœal symptoms, and that the descent of the diaphragm even at this early stage causes measurable enlargement of the upper region of the abdomen, which may easily be, and often is, mistaken for abdominal flatulence. On percussion the chest is probably abnormally resonant, and the cardiac dulness is diminished in area or effaced. On auscultation there is usually total absence of normal respiratory murmur, but in its place general sibilant rhonchus in all its varieties. The patient speaks with difficulty, bringing his words out pantingly one by one. There is usually no cough, at all events none at the beginning of the attack.

The duration of the paroxysm varies from a few minutes to several weeks—most frequently it is two or three days, or it commences at the usual time and subsides in the course of the following day. When the attack is much prolonged, it is generally made up of a series of shorter attacks, separated from one another by periods of more or less perfect remission. Its disappearance is attended with the gradual

subsidence of the asthmatic phenomena, and the supervention of cough. This is at first dry; but by degrees crepitation replaces wheezing, and the cough is then accompanied by the expectoration of mucus in small transparent pearly pellets, and occasionally by sputa slightly streaked with blood. Frequently, and this is especially the case after short attacks, recovery is rapid and complete. In other cases, the patient suffers subsequently for a longer or shorter time from tightness at the chest and dyspnœa, with more or less cough and expectoration.

Asthmatic attacks have a tendency not only to recur, but in a large number of cases to distinct periodicity of recurrence. Thus in some cases they come on weekly, monthly, yearly, or at other intervals which the patient's experience enables him to foretell. In most cases, however, their recurrence is due to the more or less regular recurrence of those extrinsic causes which determine the attack: such, for example, as the assumption of the recumbent posture, variations in diet, change of residence, and change of season.

The prognosis of asthma is very uncertain. In many cases, especially if it commence in infancy, it disappears during the period of adult life. When it comes on at an advanced age it is probably always permanent. Indeed, in a large proportion of cases, whatever the time of its commencement, its duration is life-long. But then the affection usually undergoes some change of type with the advance of years. This depends in great measure on the slow supervention of organic lesions. Thus, especially if the attacks be frequent and severe, the lungs are apt after a time to become emphysematous, and the right side of the heart hypertrophous—conditions which are usually attended with diminution in the severity of the actual attacks of asthma, but the development of permanent shortness of breath during the intervals between them, and other symptoms of emphysema and chronic bronchitis. Further, asthmatic patients frequently acquire an almost characteristic physical conformation. They are, as a rule, emaciated, with thin furrowed cheeks, high shoulders, body bent forwards, head thrown back, and misshapen chest—the upper part being dilated, the lower compressed, especially in the lateral direction.

Dr. Salter divides asthma into idiopathic or primary, and symptomatic or secondary. The former is the affection which we have endeavoured to describe. But symptoms identical in the main with those of asthma supervene secondarily on other affections, especially dyspepsia, bronchitis, and heart disease, and are termed by him peptic, bronchitic, and cardiac respectively.

Pathology.—The extreme rarity with which death takes place in uncomplicated asthma renders the investigation of the pathology of the disease difficult. There is ample proof, however, that it occurs quite independently of organic lesions of the lungs, heart, or other important organs, and that it is therefore a so-called 'functional' disease. Indeed, the observations of Drs. Gairdner and Salter establish,

almost beyond the possibility of doubt, that its symptoms are essentially dependent on spasmodic contraction of the muscular tissue of the bronchial tubes, and consequent narrowing of their calibre. It is easy to see that such contraction is ample to explain not only the dyspnœal symptoms, but the auscultatory peculiarities, the sudden accession and sudden subsidence of each attack, and even the organic changes which in some cases ensue. At the same time it may be worth while to notice that there is something in the persistence and singular periodicity of the disease, and in the fact of the frequent dependence of the paroxysm on a variety of apparently trivial conditions, to remind us of the skin affections known as urticaria evanida and factitious urticaria, and that urticaria-like swelling of the mucous membrane might equally explain the temporary contraction of the bronchial tubes. Under any view, however, the bronchial affection must be referred to the operation of the nervous system, possibly excited by reflection from the bronchial surface.

Treatment.—In the treatment of the asthmatic paroxysm all ligatures and other impediments to respiration should be loosened, and the patient should be made to assume such a position as will enable him to use his respiratory muscles to advantage. Further, any gastric or other derangement under which he may be suffering, or any condition which may be supposed to have induced or favoured the attack should, as far as possible, be at once remedied or removed. Many drugs are more or less beneficial. Among these may be enumerated tartar emetic and ipecacuanha in emetic doses; tobacco, given so as to produce its characteristic depressing effects, or smoked in the usual way; lobelia inflata also so given, in large and frequently repeated doses, as to cause great depression; datura stramonium or datura tatula, used either by inhalation or in the form of tincture or extract; belladonna, conium, hyoscyamus, and opium (the last, according to Dr. Salter, is injurious in uncomplicated asthma, benefiting those cases only in which there is associated bronchitis); alcohol, ether, strong coffee; nitre paper burnt in the apartment; and chloroform. The effects of chloroform are marvellous, but unfortunately they are for the most part only temporary.

The principles by which the general treatment of an asthmatic patient should be regulated are sufficiently simple: they consist in the avoidance of all the causes which in his case are known to induce an attack; the selection of that locality for residence which experience has shown to be most suitable for his case; and the maintenance of his general health by wholesome food, and the adoption of habits and an employment compatible with health. If the patient have not yet learnt by experience what he can do and what he cannot do with impunity, the rules which we lay down for his guidance must be such as are in accordance with what we know of the general peculiarities of the disease.

XXI. HAY-ASTHMA. (*Hay-fever.*)

Definition.—This term has been applied to a peculiar catarrhal affection coming on in this country during the months of May, June, and July, and commonly referred to the emanations from various flowering grasses, or new-mown hay.

Causation.—A small number of persons only are susceptible, but these suffer annually at the season specified, unless they take precautions against the inhalation of the irritating influence, by either remaining indoors, betaking themselves to some large town, removing to the sea-side, or taking a sea voyage. The tendency to hay-asthma seems hereditary. Conditions closely resembling it are produced in some persons by the smell of ipecacuanha or other vegetable effluvia, or by emanations from various animals, such as cats, rabbits, and dogs. The precise cause of hay-asthma has been a matter of discussion. Helmholtz, a few years ago, discovering, in the mucus discharged from the irritated mucous membrane, lowly vegetable organisms, attributed the disease to their influence. Dr. Elliotson, many years since, suggested pollen as its cause. And Mr. Blackley, himself a sufferer, has recently published a work on hay-asthma, in which, by a series of most careful researches, he appears to have proved the accuracy of Dr. Elliotson's suggestion. He comes to the conclusion that the effects of pollen are partly mechanical, partly chemical, and that it acts locally:—if applied to the eye causing conjunctivitis; if to the nose coryza; if by inhalation to the bronchial tubes asthmatic symptoms.

Symptoms and progress.—The symptoms of hay-asthma are in the main those of violent catarrh;—namely itching, congestion and swelling of the conjunctivæ and eyelids and watering of the eyes; itching, congestion, tumefaction, and copious discharge from the nostrils, attended with much sneezing; great irritation in the throat, fauces, and soft palate; tightness at the chest, and dyspnœa, with cough and expectoration. The symptoms vary in their severity, and generally become aggravated towards the middle and end of June. In the first instance, and in mild attacks, the conjunctival and nasal mucous surfaces alone may suffer. The symptoms are in all cases liable to more or less regular exacerbations.

Treatment.—The most obvious and effectual method of treatment is the avoidance of the cause of the disease; and, indeed, many sufferers have learnt by bitter experience entirely to shun the country and all proximity to grass fields and new hay during the dangerous season. For those who are compelled to expose themselves, the use of a respirator, made, as Mr. Blackley suggests, with six or eight folds of crape or a double fold of cambric, will prove of considerable advantage. Helmholtz has recommended (and his recommendation appears to

have been followed with more or less success) the washing out of the nostrils and throat with a weak solution of quinine, by means of a pipette or nose-douche. Other remedies which have been tried with reputed success are the tincture of nux vomica in ten-minim doses, tincture of aconite, liquor arsenicalis, hydrocyanic acid, and the smoking of tobacco or stramonium.

CHAP. IV.—DISEASES OF THE VASCULAR ORGANS.

THE vascular system comprises the heart, arteries, veins, and capillaries; the lymphatic glands and vessels, together with certain ductless glands; and the blood with its tributary fluids. Of all the parts here enumerated the heart, the centre and presiding genius of the system, is by far the most important, both physiologically and on pathological grounds; and to the morbid conditions of the heart, therefore, we shall first direct attention.

SECTION I.—DISEASES OF THE HEART.

I. INTRODUCTORY REMARKS.

A. *Anatomy and Anatomical Relations of the Heart.*

1. *Dimensions of heart.*—The heart has been estimated somewhat roughly, yet not inaptly, to equal in size its owner's fist. It enlarges with the growth of the body until growth ceases, and then continues to enlarge, though slowly, during the remainder of life. Its average weight in the adult male varies between ten and eleven ounces; in the adult female is about nine ounces. The capacities of its several cavities are probably nearly equal; the auricles, however, are believed to be somewhat less capacious than the ventricles, and the left cavities than the right. The ventricles of the adult heart have, each, a capacity which has been variously estimated at from two to five ounces. The mean capacity is probably three ounces. The thickness of the cardiac walls presents considerable differences: those of the right auricle measure on the average about a line; those of the left auricle about a line and a half; those of the right ventricle (at the base, where they are thickest) very nearly two lines; and those of the left ventricle (midway between the base and apex, where their thickness is greatest) rather more than five lines. Of the cardiac orifices the auriculo-ventricular are larger than the arterial, and those of the right side larger than those of the left side to which they respectively correspond. The following table gives Dr. Peacock's circumferential measurements:—

		MALES. Inches.	FEMALES. Inches.
Auriculo-ventricular	Right	4·74	4·56
	Left	4·	3·99
Arterial	Pulmonic	3·55	3·49
	Aortic	3·15	3·02

2. *Relations of heart to pericardium.*—The heart, which occupies the middle mediastinum, is contained within the fibrous pericardium—a somewhat conical bag, of which the base corresponds to a portion of the central tendon of the diaphragm, the apex to the ascending arch of the aorta, the pulmonary artery, and superior cava; with the parietes of which vessels, as also with those of the pulmonary veins and inferior cava, its fibrous tissue blends. The serous pericardium, on the one hand, lines the fibrous pericardium in its whole extent; on the other hand, closely invests the heart, forms a tubular sheath around the trunk of the pulmonary artery and the ascending aorta, furnishes incomplete investments to the proximal extremities of the pulmonary veins and venæ cavæ, and is reflected thence to become continuous with the parietal lamina. The pericardial cavity extends from the upper margin of the second costal cartilage above (at which point the ascending arch ends), to the central tendon of the diaphragm below.

3. *Relations of heart to chest-walls and surrounding organs.*—The heart occupies an oblique position. Its base, which includes the points of attachment of all the large arteries and veins, and is formed mainly by the auricles, is directed upwards, backwards, and to the right, and extends vertically from the fourth to the eighth dorsal vertebra. Its apex points in the opposite direction, namely downwards, forwards, and to the left, and usually impinges between the fifth and sixth costal cartilages, a little internal to a line drawn vertically through the nipple. The position of the apex varies, however, a little in different healthy persons, and differs a little also with change of posture, and with the respiratory movements. If it beat in the fifth interspace during recumbency, it probably beats against the sixth cartilage when the upright position is assumed. The anterior aspect of the heart, which also faces upwards, is formed below and to the right by the right auricular appendage and right ventricle; above and to the left by the left auricular appendage and left ventricle. The greater part of this aspect, however, is constituted by the right ventricle. The posterior aspect, which is also directed downwards, and rests mainly on the diaphragm, consists exclusively of the two ventricles—the left ventricle forming by far its larger proportion. About two-thirds of the heart are situated to the left of the mesial plane of the body, the remainder to the right.

The relations of the heart and great vessels to the surface of the chest are important. The outer margin of the left ventricle extends from the left second intercostal space (midway between the osseous ribs and sternum) outwards and downwards to the apex in the left fifth interspace. The outer margin of the right ventricle commences at the sternal end of the right fifth costal cartilage, and passes thence downwards and to the left to meet the former line in the apex. The left auricular appendage slightly overlaps the upper edge of the left ventricle, making its appearance in the lower part of the second inter-

costal space. The right auricular appendage extends from about the same level above (commencing, however, at the mesial line of the sternum) downwards to the point at which the margin of the right ventricle begins. Between these points it presents a semilunar form—the one limiting line taking a nearly straight course beneath the sternum, the other limiting line being convex, and extending in the third and fourth interspaces half-way from the sternum to the osseous ribs.

Of the valves, the pulmonic is the highest and most superficial; it is situated immediately to the left of the sternum—perhaps a little beneath it—in the second interspace; the aortic, which is deeper-seated than the pulmonic, and indeed partly overlapped by it, is subjacent to the junction of the left third cartilage with the sternum and to the adjoining half of the sternum; the tricuspid lies beneath the sternum, its centre midway between the sternal ends of the fourth costal cartilages; the mitral, which lies deepest of all the valves, is situated behind the pulmonic and aortic, and on a lower level than they, its central point probably corresponding to the left third interspace, a little external to the sternum.

A small portion only of the heart is in actual contact with the anterior walls of the chest, the remainder being separated from them by the thin edges of the lungs. In ordinary tranquil inspiration the lungs almost meet in the mesial line of the sternum from above down to a point midway between the sternal ends of the fourth costal cartilages. From this point the edge of the right lung still continues vertically downwards, while that of the left retreats to the junction of the left fifth cartilage and rib, where it forms a notch just before its termination in the basal edge. A triangular interval is thus produced, situated wholly to the left of the mesial line of the sternum, bounded on either side by the edges of the lungs, and below by the diaphragm, to which the left lobe of the liver is immediately subjacent. In the outer angle of this space a small portion of the apex of the left ventricle becomes superficial, the rest of the triangle being occupied by the right ventricle.

Laterally, the heart is bounded by the lungs, from each of which it is separated by both pleura and pericardium; behind, it is limited by the posterior mediastinum, with the roots of the lungs above, and the œsophagus and thoracic aorta in its whole extent, separating it from the vertebræ; below, it lies on the diaphragm, which divides it from the liver, and partly, to the extreme left, from the stomach. Above, it is continued into the large vessels, namely the pulmonary artery, aorta, and vena cava.

The ascending aortic arch, covered at first by the pulmonary arterial trunk, and then by the right auricular appendage, passes upwards and to the right beneath the sternum, and extends for about a quarter of an inch beyond the edge of the sternum into the right second and first intercostal spaces. The superior cava extends half an

inch farther in the same direction. The transverse arch corresponds as nearly as possible to the lower half of the manubrium. The pulmonary artery passes between the two auricular appendages upwards, backwards, and to the left, and, having crossed the commencement of the aorta, lies to the left of that vessel, becoming superficial at the inner part of the left second interspace, just before it retreats under the aortic arch to divide into its two branches.

B. *Physiology of the Heart.*

1. *Action of heart.*—The function of the heart is to maintain the circulation of the blood in both the systemic and the pulmonary vessels. To effect this it undergoes a series of alternately active and passive movements, which are rhythmical and follow one another with greater or less rapidity. To commence with the ventricular contraction:—The ventricles, already distended with the blood transmitted to them from their respective auricles, contract suddenly and actively, propelling their contents into the aorta and pulmonary artery respectively, and causing at the same time the closure of the auriculo-ventricular valves. Whilst this contraction is in progress the auricles, which were contracted at the moment of its commencement, gradually dilate, and by the time the ventricles have got completely empty, have attained their full dimensions and are full of blood. The contracted ventricles now relax and in their turn expand, the arterial valves close, and the auriculo-ventricular valves open and allow of the flow of blood through the still dilated auricles into the ventricles. Soon the passively dilating ventricles are almost filled, when suddenly the hitherto torpid auricles contract, adding their contents to those of the ventricles which thus become distended. Immediately after the contraction of the auricles, and indeed almost by a continuous peristaltic action, the contraction of the ventricles takes place, and the cycle of events above described recurs.

It is important to add, in the first place, that the actions of the two sides of the heart are, as nearly as possible, synchronous; and in the second that the closure of the auriculo-ventricular valves takes place at the beginning of the cardiac systole, that of the arterial valves at the beginning of the cardiac diastole. It must be added, too, that the force with which the ventricles act is always exactly equal to the resistance which they overcome; that (other things being equal) contraction of the arterioles calls for increase of cardiac exertion, their dilatation for its diminution; and that (again other things being equal) increased quickness of the ventricular systole implies greatly augmented exercise of cardiac force, and conversely.

The contraction of the heart is attended with distinct pulsation in the præcordial region. The area over which this extends varies somewhat with the form of the chest, and considerably with different

degrees of thinness or plumpness of the thoracic parietes. Generally it is limited to the apex, where it is always most intense, and covers not more than a square inch of surface. A certain amount of epigastric pulsation, due to the movements of the right ventricle, is compatible with health.

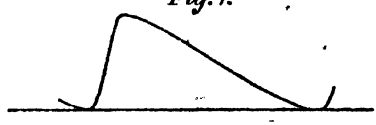
2. *Sounds of heart.*—The contraction and dilatation of the ventricles are each attended with a characteristic sound, which marks the commencement of the act, and is followed by a short interval of silence. These constitute respectively the first and second sounds of the heart. The first, or systolic sound, varies in character in different persons; it is, however, always deeper in tone and longer in duration than the other; it is also more or less compact, beginning and ending with a certain amount of abruptness. It is audible over the whole of the cardiac region, but is most pronounced over the apex of the left ventricle. The second, or diastolic sound, is short, perhaps half the length of the first, sharp and sometimes ringing. It is heard with greatest distinctness at the base of the heart, and more especially in the right second interspace immediately adjoining the sternum. The loudness of the sounds and the extent of surface over which they are respectively audible are subject to great variety.

Many causes have been assigned for the cardiac sounds. There is, however, now no doubt that the second sound is due to the sudden closure of the arterial valves which takes place at the commencement of the ventricular diastole; and there is little doubt that the first sound is mainly attributable to the generally less sudden closure of the auriculo-ventricular valves which attends the commencement of the ventricular systole. But it is pretty certain that the first sound is reinforced by that due to the contraction of the muscular tissue of the cardiac walls. For the most part, as has been already pointed out, the two sides of the heart act in unison; and hence the two arterial valves usually concur in the production of the second sound, the two auriculo-ventricular in that of the first; but, inasmuch as the action of the left side of the heart is far more powerful than that of the right, the valves of that side take the chief share in causing the cardiac sounds. It is owing to this fact that the second sound is usually loudest towards the base of the heart over the right half of the sternum, and that the first is usually most obvious where the left ventricle becomes superficial, namely at the apex. When the sides do not act in perfect unison, a more or less obvious reduplication of the cardiac sounds takes place.

3. *Pulse.*—The intermittent injection of blood from the heart into the arteries produces the phenomenon known as the pulse. The beats of the pulse correspond as a rule in number and rhythm to the contractions of the cardiac ventricles; and, like them, follow one another, for the most part, with remarkable regularity; although liable, in different persons, and under different circumstances, to present

great variations as to rate and force, and always presenting slight relative increase of rate and force during inspiration, and slight relative decrease during expiration. The character of the pulse, although depending mainly upon the action of the heart, is largely modified by the condition of the arteries in which it occurs, and by that of the capillary arteries and capillaries, and of the venous system beyond. During the whole period of the contraction of the left ventricle its contents are being propelled into the aorta; and the force which is thus exerted within the arteries is expended, partly in driving the blood already within the vessels onwards, partly in stretching the elastic walls of the arteries. The consequent arterial tension attains its maximum with more or less rapidity, and then diminishes before the systolic action is completed. As soon as, with the cessation of the systole, the propulsion of blood into the aorta ceases, the distended arteries contract upon the blood within them, still propelling it onwards, but with gradually diminishing force, until they have attained their former calibre or until their contraction is interrupted by the next cardiac systole. The period here adverted to corresponds to the ventricular diastole. If the pulse presented no other elements than those immediately due to the phenomena which have just been considered—namely, the systolic distension of the artery, on the one hand, and its diastolic collapse on the other, the sphygmographic trace of the pulse would have some such form as that represented in fig. 1.

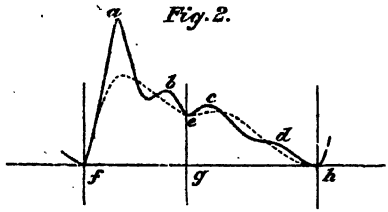
Fig. 1.



There would be a more or less sudden rise, presenting a rounded summit, the highest point of which would correspond to the moment of highest arterial tension; and this would be followed by a more or less gradual fall. But for the most part the sphygmographic tracing displays other elements besides these. In the first place the line of ascent is usually prolonged vertically upwards and then suddenly falls, forming a very acute angle, before it merges in the convex summit above indicated; and in the second place the line of diastolic collapse is for the most part interrupted at its commencement by a more or less distinct rise and frequently after a short interval by a further and less distinctly marked wave, or a diminishing series of waves. The typically complete tracing would thus present not less than four successive waves, of which at least two would correspond to the systole of the heart, and at least two to the diastole. The first of these waves, which is known as the *primary* or *percussion wave*, is generally attributed, not to any actual addition to the quantity of blood which the

artery presenting it already contains, but to the impulse which is supposed to be transmitted along that blood by the shock of the commencing systole; and is supposed to precede by a scarcely appreciable interval the *secondary* or *tidal wave* which follows it. Dr. Galabin, however, shows that this explanation is incorrect, and 'that the percussion and tidal waves form in the artery but one wave, and are only separated by the sphygmograph. Owing,' he says, 'to the inertia of the long lever it is carried up a little too high, and when in falling it meets the true arterial wave it is again tossed up, and thus forms the tidal wave.' The third, or the *dicrotic wave*, has like the first, been variously explained. It has been attributed by many to the shock of the sudden closure of the aortic valves, an opinion in which Dr. Galabin concurs; and again to the recoil of the hitherto distended

Fig. 2.



a, Primary or percussion wave; *b*, secondary or tidal wave; *c*, dicrotic wave; *d*, fourth wave; *e*, aortic notch; *f g*, duration of cardiac systole; *g h*, duration of cardiac diastole.

The dotted line represents the tracing which would be drawn if the instrument followed the movement of the artery with perfect accuracy.

Copied, with slight modification, from Dr. Galabin's diagram.—*Thesis for the Degree of M.D., Cantab.*, 1873.

arteries. But the cause is probably that which Dr. Sanderson assigns for it. He points out that as the wave due to the injection of the ventricular contents into the aorta takes a certain time to reach the capillary arteries, and as hence the period of greatest movement in the latter vessels must take place distinctly later than that in the aorta, so the subsidence of this wave and the period of comparative rest which marks the end of systole and the whole of diastole is likewise delayed in transmission to the peripheral vessels; and that consequently there is a moment at which, while the blood is almost stagnant in the aorta, it is still flowing rapidly through the minuter vessels, and a later moment at which the blood in the capillaries also becomes comparatively quiescent. But this arrest in the capillaries, accompanied as it is by the contraction of the elastic arterial coat upon the diminished contents of the vessels, produces a virtual distension and a sudden increase of pressure throughout the arterial system. The dicrotic wave is the expression of this arterial tension. The fourth wave has probably, as Dr. Galabin considers, the same relation to the dicrotic wave as the tidal to the percussion wave.

Let us now briefly consider the significance in the order of their occurrence of the more important of the several factors of the pulse-tracing which have been enumerated. The initial rise will necessarily be largely determined as to its amplitude by the suddenness and violence of the cardiac systole; but will obviously be also influenced more or less considerably by the condition of the arteries—flaccidity aiding it; tension, on the other hand, opposing it. The presence of the tidal wave as a distinct event depends mainly upon the duration of the tension of the arteries due to the ventricular systole. If the tension be of short duration the percussion wave falls rapidly and continuously until its fall is arrested by the dicrotic rise; if it be long sustained, then the second rise becomes developed, varying in its form according to the condition of the artery. The breadth of the combined summits of these two curves is, therefore, a measure of the duration of the tension here adverted to; the breadth of their bases, as determined by a horizontal line drawn from the commencement of the systolic rise to the end of the systolic fall, is a measure of the duration of the cardiac systole; and the lowest point of the notch which separates the tidal from the dicrotic wave indicates the moment of closure of the aortic valve. The third rise (the dicrotic wave) may be regarded as a measure of the completeness of the check which the systolic wave experiences in the smaller vessels during the diastolic period, and is indicative, therefore, either of comparative feebleness of the heart's action or of high tension of the venous relatively to that of the arterial system. The duration of the diastolic period is measured by the horizontal line which may be drawn from the aortic notch to the commencement of the next systolic ascent.

The character of the pulse varies in health, not only in different individuals, but in the same person at different times and under different circumstances. It may be *frequent* or *infrequent*, conditions which may be recognised as well by the finger and the watch as by any more complicated machinery. It may be *long* or *short*. These terms apply, not to the whole interval between the commencement of one pulsation and that of the pulsation which next follows (for in that case they would be synonymous with 'infrequent' and 'frequent' respectively) but to the duration of the systolic wave. These qualities may be roughly recognised by the finger, but are demonstrated with accuracy only by the sphygmographic tracing. It should be noted here that when a pulse becomes increased in frequency, this increase is due mainly to curtailment of the diastolic period. It may be *large* or *small*. These terms are employed somewhat loosely; the former should perhaps be used of that state in which a considerable volume of blood is propelled into the arteries at each systole, and the latter of the converse condition. We are apt, however, to term that also a large pulse which occurs in dilated arteries, such as those of elderly persons, and that a small pulse in which the arteries are simply contracted. These different forms of

largeness or smallness are often combined, and are indicated respectively in the sphygmographic trace by relative amplitude of the systolic waves. The pulse may be *strong* or *weak*, or in other words *hard* or *soft*. The former resists compression by the finger, the latter is easily obliterated by it. The best test, however, of strength of pulse is again the sphygmograph, by means of which the amount of pressure necessary to procure obliteration can be estimated with some degree of accuracy. It may be added that when there is high arterial tension it is necessary to use considerable pressure in order thoroughly to develop the characteristic tracing.

C. *Pathology of the Heart.*

The heart and structures associated with it may, as is the case with all other organs, become the seat of inflammatory or other processes, which will then produce the local and general symptoms commonly belonging to such processes. But the heart is an instrument of extreme delicacy, and is liable, under the influence of these and other conditions, to have its mechanism more or less seriously deranged. Its derangements, which may be structural or functional, or both combined, produce various local and remote or general consequences which are the characteristic symptoms or sequelæ of heart disease. We propose to consider these derangements and their consequences briefly, apart from the intimate nature of the pathological lesions from which they spring, and apart also from the special symptoms due to the specific nature of these lesions.

1. *Mechanical and Structural Derangements.*

The local conditions which interfere with the healthy action of the heart may either be seated external to the organ; or involve its muscular walls; or be connected with its valves; or be situated within its cavities. It may of course happen that two or more of such lesions are associated.

a. Conditions external to the heart.—Simple displacement of the heart is met with under many different circumstances. Occasionally, in company with the other viscera of the chest and abdomen, it is found transposed. Ascites or abdominal tumours may displace its apex upwards and to the left; aneurysms of the arch, and other tumours of the upper part of the chest or posterior mediastinum, may cause it to descend. Serous or other effusions into either pleura commonly push it over towards the opposite side; while the contraction of the lung and side which so often attends cirrhosis and the absorption of pleuritic fluid tends to attract it more and more towards the affected side. It may be observed that the displacements which result from the last conditions are generally much more noticeable when they take place towards the right than when they take place towards the left side; in distension of the left pleura it is not uncommon to find the heart beating

wholly between the right nipple and right edge of the sternum. In spinal curvature, also, the position of the heart is often much modified, being then greatly determined by the relative sizes of the two halves of the chest and by the degree and form of the curvature; sometimes it lies wholly to the right of the sternum, sometimes beneath it. Occasionally, when the patient is markedly pigeon-breasted, the heart occupies the whole of the space which lies between the two nipples. The most remarkable displacements of the heart, however, are those which result from the growth of intrathoracic tumours. In such cases the apex of the organ has been detected beating in the right axilla.

In reference to displacements, it is important to recollect, that the base of the heart is comparatively firmly fixed above and posteriorly; and that, although no doubt the parts contained in the posterior mediastinum, and the base of the heart which is incorporated with them, are often displaced to a greater or less extent, it is that portion of the heart which lies free in the pericardium that is chiefly apt to suffer in this respect, and is often the only part so affected—the free or ventricular portion of the organ moving at its base as on a pivot.

Affections of the pericardium are much more serious causes of cardiac disturbance than those just considered. They act mainly by compressing the heart and thus interfering with the efficient performance of its duties. The affections here specially adverted to are such as are attended with effusion of fluid and those in which fibrous and other forms of solid material accumulate. The effusion may be merely dropsical, or inflammatory and associated with the formation of false membrane, or purulent, or hemorrhagic. When fluid is poured forth into the cavity, it gradually distends it, enlarging it in all its dimensions, but chiefly in those situations in which its walls are least resistent. The cavity becomes rounded and at the same time elongated, especially in the upward direction along the ascending arch and pulmonary artery; and moreover by its distension it displaces the diaphragm downwards, and the lungs laterally. At the same time the heart is necessarily carried with the portion of the parietal pericardium to which it is united backwards, and consequently away from the anterior thoracic walls. The quantity of fluid which accumulates within the pericardium is sometimes enormous. Two and even three quarts have been met with. The larger quantities are generally the result of chronic disease, which allows of the gradual distension of the pericardial cavity to a much greater extent than is possible in acute cases.

b. Conditions involving the muscular walls.—The muscular walls of the heart are liable to many changes. Among these we may especially enumerate hypertrophy and atrophy. Hypertrophy always takes place in response to some increased work which the heart is called upon to perform, is in general compensatory, and (apart from other associated cardiac defects) prevents rather than promotes cardiac embarrassment. Simple atrophy almost invariably occurs in the course of chronic wasting

disease, and in some sort of proportion to the concurrent atrophy of the rest of the organism; and hence the dwindled heart still remains sufficiently strong to perform the offices required of it, and as a rule gives rise to no untoward symptoms. Generally associated with hypertrophy, and occasionally with simple atrophy, dilatation of the heart's cavities takes place. The extremest dilatation is met with in cases of great hypertrophy; but there is no necessary relation between dilatation and the amount of muscular tissue present. Dilatation is on the whole an evidence and cause of cardiac weakness. Other causes of cardiac debility are fatty and other forms of degeneration, inflammation, and syphilitic, sarcomatous or other infiltrating growths.

c. Conditions involving the valves.—By far the most serious derangements on the whole are those connected with organic disease of the valves. Valvular defects may be of two kinds:—namely, *obstructive* (*stenosis*), that is of a nature to impede the direct flow of the blood; or *regurgitant*, in which the closure of the valves is imperfect and reflux of blood is hence permitted into the cavities behind them. The affections which produce valvular lesions are very various. We may here advert to the fact that the mitral valve, and still more the tricuspid, occasionally even in health admit of regurgitation from the ventricles into the auricles when the ventricles are over-distended with blood. In a large proportion of cases the lesions are due to inflammatory changes:—namely, infiltration and thickening of the tissues of the valves, the formation of beaded or warty masses upon their surfaces, and consequent adhesion, ulceration, or laceration. In many cases the valvular affection is consequent upon atheromatous and calcareous degeneration of the several structures connected with the valves. Occasionally it is the result of accidental violence, and occasionally of congenital malformation. Further, incompetence is sometimes due to dilatation of the valvular orifices—a condition which is apt to go along with dilatation of the ventricles, and to affect mainly the auriculo-ventricular apertures. In these cases incompetence may be aggravated by comparative shortness of the carneæ columnæ, or chordæ tendineæ. We will briefly discuss the principal defects which the several valves are apt to present.

Obstructive aortic valve disease may be due to the following causes:—(i.) adhesion of the several segments to one another (as a congenital defect it is not uncommon to find two contiguous cusps blended as far as their corpora arantii, the coalesced sides forming a more or less obvious vertical frænum, which divides the upper aspect of the compound organ into two halves; more rarely the three valves are thus blended, and when the blending is pretty complete they form between them a conical funnel with a narrow orifice in the apex which is directed upwards): (ii.) the accumulation of inflammatory granulations: and (iii.) atheromatous or calcareous changes. The last are often attended with great thickening and at the same time great rigidity of the valves, the orifice becoming in some cases converted into a mere chink.

Regurgitant aortic valve disease may depend (i.) on contraction and puckering of the free edges of the valves, in consequence of which they fail to meet; (ii.) on ulcerative destruction or contraction of the valves at their angles, which allows the intermediate free edges to form pendulous or everted flaps; and (iii.) on rupture or actual perforation of the curtains. We do not of course refer here to the fenestræ so commonly observed in the lunulæ, which, as is well known, do not in any degree impair the efficiency of the valves.

Obstructive mitral disease may depend (i.) on cohesion of the edges of the curtains (this is often congenital, the valve then presenting a more or less funnel-like character, with its apex pointing towards the ventricle, and formed by a narrow, button-hole slit; in most cases of this kind the valve is thickened, the chordæ tendineæ are short and thick, and the smaller branches, which radiate into the valves, apt to be more or less completely blended with them and with one another; in cases of inflammation and degenerative change there is a similar tendency to the production of the conditions here enumerated): (ii.) on inflammation, which causes thickening and at the same time granular excrescences; and (iii.) on atheromatous and calcareous changes.

Regurgitant mitral disease may depend (i.) on mere contraction of the free edges of the cusps; (ii.) on shortening or rupture of the chordæ tendineæ; and (iii.) on perforation of the valves.

The morbid conditions to which *the valves of the right side* are liable are identical with those which involve the corresponding valves on the left side. The pulmonic and tricuspid valves are, however, comparatively rarely the seat of other than congenital disease.

d. Conditions involving the contents.—Coagulation of blood in the heart's cavities not unfrequently takes place during life, more especially during the period in which the patient is moribund, when it must be regarded as a normal accompaniment of the process of dying. Older clots are also occasionally met with, such as the adherent rounded softening clots, or so-called 'polypoid concretions,' and the laminated clots which are also common in sacculated aneurysms. The causes of these different varieties of coagula, their anatomical peculiarities, and their effects, will be discussed at length under the head of thrombosis and embolism.

2. *Functional Derangements.*

a. Motor derangements reveal themselves by undue feebleness or force, frequency or infrequency of action, by intermission or irregularity, or by sudden arrest of action from spasm or paralysis. Several of these are only exaggerations of conditions which are compatible with health. *Feebleness* of the heart's action attends most wasting diseases and the later period of many febrile and other acute affections. It is common also in mitral disease, and in some other morbid conditions of the heart. It is characterised, by weakness of apex beat; diminished

intensity of the cardiac sounds, especially the first, which may be absolutely abolished; feebleness of pulse, which is undulatory, thready, or markedly dicrotous, and may be imperceptible at the wrist, even while maintaining a distinctly dicrotous character in the larger arteries; and, lastly, a great tendency to variation in the rate of the pulsations under the slightest disturbing influences. *Increased force* of cardiac action tends to take place whenever any impediment to the flow of blood occurs either at the aortic orifice or in the course of the arteries, in the capillary vessels or in the venous system. It is common therefore in aortic valve disease, in the presence of rigid arteries, and in Bright's disease. It may also occur in inflammatory disorders, during muscular exertion, and under the influence of nervous excitement. The indications of this condition are, violence and extension of the cardiac impulse, which is often prolonged and heaving, and may be felt in the back and even shake the entire body; loudness of the cardiac sounds; and hardness of pulse. *Increased frequency* of pulsation occurs under many various conditions; such as nervous excitement, debility, febrile disease, and so on. The individual pulsations may be weak or strong, and the characters presented by the cardiac movements, its sounds, and the arterial pulse will correspond. *Diminished frequency* of pulsation is common in convalescence from acute disorders. It is also met with in some cerebral affections and in some cases of cardiac disease. The pulsations of the heart may mount up to 200 or even 260 in the minute, and they may fall to 20 or even 12. The term '*palpitation*' is commonly used of those conditions in which, under the influence of nervous excitement, the pulsations of the heart, arteries, or both, are painfully evident to the patient himself; the beats are frequent, sudden, and violent, and the pulse often attended with marked dicrotism. *Irregularity* of the cardiac rhythm is occasionally observed in gout and indigestion, but is most frequently associated with various forms of heart disease, and especially with affections of the mitral valve. It is manifested by inequality of the successive pulsations, both as regards their force and fulness, and the length of the interval which elapses between them. *Intermission* of action is a form of irregularity which is mostly functional; it is common in dyspepsia, and is occasionally a constitutional peculiarity of the patient. In intermission the general rhythm of the heart's action is not impaired, but at regular or irregular intervals a pulsation is dropped, as it were. At the wrist it is wholly absent; on listening to the heart, however, the intermission is represented by an abortive three, followed by a pulsation of greater intensity than those which follow next. Occasionally such abortive strokes may occur alternately with effective ones, and the pulsations at the wrist be half as numerous as the cardiac strokes. *Sudden arrest* of the heart's action and consequent death may be caused by shock, or syncope, and is uncommon in certain forms of heart disease.

b. Abnormal sensations are frequently associated with [illegible]

tions. In palpitation the pulsations of the heart and often of the larger arteries are distinctly felt and complained of by the patient. When intermission takes place the sufferer generally experiences a kind of throb, or tumble in the region of the heart, or a choking sensation which may be attended with momentary faintness. A feeling of oppression at the chest, or fulness, or aching, is not uncommon. And sometimes the pain may be intense, prolonged, and indeed unbearable, extending over the whole cardiac region, or limited to some definite part of it, and often radiating thence to various parts of the trunk and to the extremities, especially down the arms.

3. *Effects of Cardiac Derangements on the Walls and Cavities of the Heart.*

The effect of continued over-exertion of the heart, as of all other muscles, is hypertrophy. Its muscular tissue increases in quantity and its walls consequently in thickness; and at the same time its cavities almost invariably undergo more or less dilatation. The hypertrophic walls are, as a rule, denser than those of the healthy heart, and often present, in addition to augmentation of muscular fibres, augmentation of connective and other tissues. Nevertheless it not unfrequently happens that, as disease advances, the hypertrophied muscle becomes enfeebled in consequence of fatty or other degenerative changes. With this reservation, however, the degree of hypertrophy may be taken as a measure of the increased labour which the heart has been called upon to perform. Although probably in some degree dilatation always accompanies hypertrophy, and owes its origin to the same cause, it must be regarded, not as an evidence of strength, but as the result of weakness—of the yieldingness of the heart's walls to the increased internal pressure to which they are subjected—and hence, although accompanying hypertrophy, as antagonistic to it. It will thus be readily understood that, other things being equal, a heart intrinsically weak will become dilated more than hypertrophied under the stimulus of over-exertion; that a heart intrinsically strong will, under similar circumstances, become hypertrophied more than dilated. It must be added that dilatation, which is sometimes the primary organic change in hearts which are simply feeble, not only impairs efficiency, but actually furnishes an incentive to cardiac exertion and overgrowth. In the great majority of cases the dilatation of the cardiac cavities is uniform; occasionally, however, the thinner parts of the walls, and especially softened or weakened areæ, yield disproportionately.

The above remarks are general. We will now apply them. Whenever any persistent obstacle exists to the passage of blood through the aortic orifice, along the arteries, or through the capillary network beyond, the left ventricle gradually gets hypertrophied, and in a greater or less degree dilated as well. For a time this hypertrophic change is

almost purely compensatory; the increased force of the cardiac contractions almost exactly counterbalances the effects of the obstacle; the heart acts regularly, the ventricle empties itself completely at each systole, the mitral valve acts perfectly, and the auricle experiences no difficulty in the transmission of its contents into the ventricle. So far all the morbid changes are confined to the left ventricle; but after a longer or shorter period disproportion arises between the hypertrophy of the ventricle, on the one hand, and its dilatation and the impediment to be overcome on the other; the ventricle fails to act efficiently, probably does not wholly expel its contents at each beat, and the auricle consequently begins to experience some difficulty in getting rid of its contents, and now in its turn becomes dilated and hypertrophic. The same sequence of phenomena follows that virtual impediment to the aortic circulation which results from aortic valve incompetence. In this case, however, dilatation doubtless precedes hypertrophy, and the auricle probably becomes stimulated to over-exertion at a comparatively early period.

Whenever disease, whether it be obstructive or regurgitant, exists at the mitral orifice, blood tends to accumulate in the left auricle, while increased force is needed for its propulsion thence; the cavity, therefore, of the auricle becomes enlarged, and its walls thickened. But inasmuch as no valves exist at the orifices of the pulmonary veins, or in any part of the course of the pulmonary vessels, the augmented pressure of blood which commences within the auricle speedily spreads backwards throughout the entire pulmonary system. And hence arise impediment to the escape of blood from the right ventricle and its consequent hypertrophy and dilatation.

Disease affecting the pulmonic orifice, equally with increased blood-pressure within the pulmonary artery, necessarily causes dilatation and hypertrophy of the right ventricle, which are presently followed by similar affections of the right auricle.

Disease of the tricuspid orifice provokes like changes in the right auricle, and in connection therewith accumulation of blood in the systemic veins and dilatation of these vessels, to be gradually followed by similar conditions in the systemic capillaries and arteries and consequent obstruction to the escape of blood from the aortic orifice.

It will be understood from the foregoing observations that when disease (actual or virtual) exists at any valvular orifice, first the cavity behind it becomes hypertrophied and dilated, and subsequently the same conditions gradually involve cavity after cavity in the backward direction, until possibly every one becomes thus affected in a greater or less degree; and, further, that in the extension of these conditions from the left auricle to the right ventricle, or from the right auricle to the left ventricle, the pulmonic or systemic vascular system, as the

case may be, necessarily suffers from undue accumulation and pressure of blood within it.

The form of the heart is greatly, and often characteristically, modified, by the hypertrophic and other changes above discussed. In general hypertrophy and dilatation, such as are met with in chronic albuminuria, the shape of the heart is altogether unchanged. When aortic valve disease alone is present, the left ventricle is alone, or chiefly, enlarged; and not only does its left edge extend further than usual to the left, so that more of the ventricle is exposed when we look at the organ *in situ*, but the apex projects far beyond that of the right ventricle. In mitral valve disease the left auricle becomes enlarged, but the right ventricle soon shares in the enlargement; the left ventricle, on the other hand, is relatively small; generally it either remains stationary or dwindles, or if it enlarges its enlargement is inconsiderable; the heart consequently acquires a nearly globular form, and the right apex either comes to share equally with the left in constituting the heart's apex, or forms it exclusively. The same shape of heart results from disease of the pulmonic orifice, or from chronic pulmonary disease; but here the left auricle remains small.

It must not be forgotten that a constantly palpitating heart becomes dilated and hypertrophied in consequence of its palpitation; and that not only temporary violent muscular effort, but also habitual sustained exertion, induces similar organic changes. Dr. Allbutt, who has investigated this subject with close attention, believes that in such cases the hypertrophy and dilatation commence on the right side of the heart, and are thence propagated to the opposite side.

4. *Effects of Cardiac Derangements on the General Organism.*

The disturbance to the circulation which results from cardiac affections cannot long continue without causing more or less serious disturbance of other organs or groups of organs. In connection with the pulmonary stasis which attends mitral disease and other equivalent conditions, we observe congestion and œdema of the lungs, effusion of blood into the pulmonary tissue (pulmonary apoplexy), thrombosis of the pulmonary arteries, which is generally associated with and is probably the cause of pulmonary apoplexy, tendency to inflammation, and all the symptoms—lividity, dyspnœa, and the like—which flow from such affections. In connection with systemic stasis there arise: general dilatation of veins and capillaries, with congestion and tendency to hemorrhage; anasarca, especially of dependent parts; dropsy of serous cavities; and thrombosis. Moreover, the liver becomes congested and indurated, and assumes the well-known nutmeg character, and jaundice and other consequences of hepatic disorder ensue. The kidneys get similarly affected, and the urine grows scanty and albuminous; and

not unfrequently the gastro-intestinal tract undergoes functional disturbance, or becomes the seat of hæmorrhagic effusion or organic lesions. The central nervous system is especially apt to suffer; from insufficient supply of blood to it arise attacks of syncope and epileptiform convulsions; from hyperæmia, drowsiness and coma; besides which, head-ache, vertigo, and delirium are not uncommon; and occasionally cerebral apoplexy from laceration of vessels ensues. There are several phenomena in connection with the systemic venous circulation, generally observed in cases of disease of the right side of the heart, and not unfrequently in mitral disease, which call for special notice. These are: dilatation and pulsation of the larger veins; pulsation of the liver; and bulbous enlargement of the terminal phalanges of the fingers and toes. *a.* Dilatation and pulsation of the veins at the root of the neck, along the neck, and even down the arms, is not uncommonly observed. The dilatation is due to their over-repletion with blood; the pulsation, as a rule, to the fact, that owing to this dilatation the venous valves allow of regurgitation, and that at the same time regurgitation takes place, during the contraction of the right ventricle, through the tricuspid valve. The pulsation in marked cases may be not only seen but felt, and the sphygmographic tracing displays a double rise, of which the second is far more ample than the first, a circumstance which is due to the fact that the latter is caused by the auricular contraction, the former by that of the ventricle. It must not be forgotten, however, that pulsation of the veins at the root of the neck may be caused simply by auricular contraction when the veins are full, and that it may be simulated by the rhythmical distension of the veins which takes place during expiration, and by pulsation transmitted from neighbouring arteries. *b.* It was first shown by Friedreich, and has since been confirmed by other observers,[1] that, under similar circumstances to the above, hepatic pulsation not unfrequently occurs. It seems to be due to regurgitation into the vena cava ascendens, and thence into the hepatic veins. The pulsation (which must be distinguished from ordinary epigastric pulsation, due to the direct influence of the action of the heart, or of the abdominal aorta) is visible over the whole extent of that portion of the abdominal surface with which the distended liver is in immediate relation, and may in many cases (especially if the enlargement of the liver be considerable) be felt, in grasping the hepatic zone with the two hands, to be distinctly expansile. The sphygmographic tracing which may be obtained from the pulsating organ presents the same characters as those yielded by the venous pulse, and indicates a like origin. *c.* In cases of long-continued venous obstruction (and especially, therefore, in heart disease of congenital origin), the last phalanges of the toes and fingers become more or less livid and swollen or bulbous—a condition which is also observed in phthisis and several other affections. Finally, we must

[1] Dr. Frederick Taylor, 'Guy's Hospital Reports,' 1875.

not forget to allude to the consequence of the detachment of cardiac vegetations, or particles of atheromatous or calcareous detritus, or of the escape of the contents of softened clots—namely, embolic obstructions of the arteries of various organs, but more especially of those of the brain, spleen, kidneys, liver, and lungs.

5. *General Diagnosis of Cardiac-Derangements.*

So far as physical diagnosis is concerned, the most important points to which we have to attend in the investigation of cardiac diseases, are: first, alterations in the form of the præcordial region; second, alterations in the area over which dulness on percussion extends; third, changes of resistance; fourth, the situation, extent, and character of the cardiac pulsation, together with the presence or absence of vibration or tremor; and, fifth, the presence of abnormal sounds.

a. Alterations in the form of the præcordial region can generally be easily recognised by mere inspection. Frequently, when the heart is enlarged, and still more when there is effusion into the pericardium, this region becomes distinctly prominent over a greater or less extent of surface. This change is much more readily produced in the child than in the adult. When much pericardial effusion is present the intercostal depressions of the implicated region are apt to be smoothed away and effaced; sometimes, indeed, they bulge. The degree and extent of protrusion may be determined: partly by comparing by actual measurement, or by the cyrtometer, the horizontal circumference of the two halves of the chest; partly by comparing, on the two sides, the relative distances between any corresponding pairs of points, as, for example, those between the nipples and the mid-line of the sternum, the width of corresponding intercostal spaces, and the like.

b. Alterations in the area of cardiac dulness must be ascertained either by the use of the pleximeter and hammer, or by the ordinary mode of percussion. Dulness over the præcordial region, like other forms of dulness, varies in quality in different individuals (according to the quantity of fat or flesh present in the thoracic parietes, and the condition of the bony framework) from the almost imperceptible sound produced by percussing the thigh, or the sharpish click which may be elicited from the patella or the forehead, to the comparatively dull thud which is yielded by the sternum. Even in the same individual, the dulness elicited over the sternum is markedly different from that obtained over the costal cartilages; and indeed the presence of dulness must be determined less by the absolute sound which is elicited than by comparing it with the sounds yielded by percussion of neighbouring parts, and more especially of corresponding parts on the opposite side of the chest. Increased area of dulness may depend either on

pericardial effusion, on hypertrophy or dilatation of the heart, on the presence of tumours, or on the retraction of the free edges of the lungs. On the other hand, extension of dulness in cardiac disease may be counteracted or annulled by the presence of pulmonary emphysema.

c. Increased resistance is, for the most part, due to the presence of thick, dense, pericardial adhesions, or of solid growths of other kinds. It may occasionally be detected by mere pressure of the hand, but is most strikingly revealed by the entire absence of that yieldingness which is generally so obvious on percussion of the thoracic parietes.

d. Pulsation and thrill.—The apex of the heart changes its position under various circumstances. In hypertrophy, it is found beating below and external to its normal site. In pericarditis with effusion the apex is somewhat elevated, and this elevation is said to be maintained to some extent even after adhesion has taken place. Sometimes the cardiac pulsation extends over a large surface. This is especially observable in cases of hypertrophy; and in hypertrophy of the right ventricle pulsation becomes strikingly obvious in the epigastrium. Extension of pulsation is also manifested in cases of pericardial effusion; but the pulsation is then due mainly to the undulations which the heart excites in the surrounding fluid, and is not strictly synchronous with the cardiac movements. Again, the cardiac pulsation varies in character. In hypertrophy it is prolonged and heaving, in palpitation it is short and violent, and in some cases of associated hypertrophy and dilatation, a distinct impulse or jog attends the diastole as well as the systole. In the last case, too, it often happens that the systolic impulse of the apex is accompanied by an obvious subsidence of the rest of the præcordial region. But, besides these, certain adventitious movements of the thoracic parietes are sometimes present in cardiac diseases, such as tremor or fremitus—trembling or vibratile movements, which are occasionally likened to the purring of a cat. They are sometimes present in pericarditis, but still more obviously in certain forms of valve-disease, more especially regurgitant aortic and direct mitral.

e. The production of abnormal sounds by the movements of the heart, or of the blood which passes through it, may be recognised either by the ear applied to the cardiac region, or by means of the stethoscope. These sounds are usually termed 'murmurs,' or 'bruits.'

i. *Pericardial murmur*, or *friction sound*, is produced by the attrition of the roughened surfaces of the heart and parietal pericardium. It varies in character: being sometimes a uniform to-and-fro sound, like that produced by rubbing two pieces of paper together; sometimes a more or less uniform crackling, rumbling, or creaking; sometimes a series of irregular jogs, which are generally more numerous than the sounds of the heart, and rarely synchronous with them, and depend on the fact that the equable movement of the opposed surfaces on one another is interfered with by the obstacle which their

roughness or stickiness interposes. Pericardial sounds, especially if of limited extent, are not always distinguishable from endocardial murmurs; they rarely, however, present much intensity, are probably never musical, and are scarcely perceptible except immediately over the part at which they are developed. ii. *Endocardial murmurs* may arise at any one of the four valvular orifices of the heart, during either the systole of the ventricles or their diastole. Thus, at the aortic or the pulmonic orifice murmurs may be developed, either while the blood is flowing from the ventricle into the artery; or during the period of diastole, in consequence of the reflux of blood from the artery into the ventricle. The former murmurs are known as *systolic* or *direct*, the latter as *diastolic* or *regurgitant*, of the respective arterial orifices. And so also, at the mitral or the tricuspid orifice, a murmur may arise, either during the systole of the ventricle in consequence of regurgitation of blood from the ventricle into the auricle, or during the diastole of the ventricle while the blood is pursuing its normal course from the auricle into it. The former murmurs are respectively *systolic mitral* and *systolic tricuspid*, and are at the same time *regurgitant*; the latter are *diastolic* or *direct*. Cardiac murmurs either replace the normal sounds of the heart or are superadded to them. They are necessarily loudest at the points at which they are developed; but in consequence of the intervention of cardiac structures which are not implicated, or of the free edges of the lungs, they are not necessarily loudest at those portions of the chest surface which are nearest to these points. Again, they are carried, as might be supposed, by the blood-stream, and are hence louder in the course of that stream than in the opposite direction. Endocardial murmurs present a wide range of character, dependent on differences of intensity, quality and pitch. As to intensity, they may be so soft as to be barely detectable, or so loud as to be distinctly audible by the unaided ear at a short distance from the pericardial region. As to quality, they may resemble a simple whiff, a whispered vowel, a whispered *r*, or a prolonged *s*; they may be harsh and rough, or grating, or they may have a more or less distinctly musical character. And lastly, the musical note may vary in pitch from bass to treble, from a deep hum or buzz to a whistle. They are often compared to sounds with which we are familiar, such as blowing, cooing, sawing, rasping, and the like; and, apart from such special qualities, are usually distinguishable from the normal heart-sounds by their greater prolongation, and by the fact of their comparatively gradual subsidence. Endocardial murmurs are the result of molecular vibrations produced in the blood as it traverses one or other of the cardiac orifices. Molecular vibrations are of course always present, whether in health or disease, but they are only rendered sufficiently intense to evolve sound either when the blood is driven with unnatural velocity through one of the orifices (as sometimes happens at the healthy aortic or pulmonic orifice in anæmia or palpitation), or

when it meets in its course with some impediment, or encounters some roughness or projection, or some pendulous vibratile body, or when, as in regurgitation, opposing streams meet and cause eddies. It is not always possible, nor is it important, to determine the conditions on which the different qualities of murmurs depend. It may, however, be remarked that roughness or hoarseness of sound implies for the most part roughness or irregularity (however produced) at the orifice at which it is developed, and that musical quality may be determined by extreme narrowness of orifice, and especially by such conditions of the edges of an orifice as permit them to perform regular vibrations. The roughest and most grating murmurs probably are the consequence of partial detachment of valves, or of rupture of chordæ tendineæ, which allows the implicated valve to flutter loosely in the blood-current. The most distinctly musical sounds are chiefly observed in murmurs due to regurgitation, as might be supposed from the combination of narrowness of orifice and of vibratile edges which is then commonly present. There can be no doubt that the quality of cardiac murmurs is often very distinctly modified by the resonance of the blood-containing ventricular cavities; in fact, that murmurs not otherwise musical are thus rendered more or less musical, and that musical murmurs have some of their harmonics developed by this means with disproportionate power. It is obvious that such modifications must occur mainly while the ventricles are filling or full, and hence specially affect murmurs developed during the ventricular diastole. It is probable that the deep tone of so-called 'præsystolic' murmurs is in some measure due to this circumstance; and that the different qualities of the same murmur, as heard over the aortic orifice where it is created, and at the apex whither it is conveyed, are similarly explicable.

Reduplication of the sounds of the heart, though occasionally observed in health, is, for the most part, an indication of disease. It may be heard under various conditions; but reduplication of the first sound is chiefly met with in connection with hypertrophy of the heart and high arterial tension, especially, therefore, in chronic Bright's disease; and reduplication of the second sound is not unfrequently observed in affections of the mitral valve.

f. Venous murmurs.—It may be added here that venous murmurs, consisting of a continuous humming or buzzing, whistling or hissing, are not uncommon in the larger veins when they are partially obstructed, and especially in anæmic patients. They may generally be best detected in the neck, particularly on the right side.

6. *Special Diagnosis of Cardiac Derangements.*

a. Pericardial effusion is indicated locally: by bulging of the præcordial region, with more or less distinct effacement of the correspond-

ing intercostal spaces; by diffused undulatory pulsation and elevation of the cardiac apex; by extension of dulness, which assumes a triangular form, and, taking place mainly upwards, may reach from the clavicle above to the diaphragm below, and be bounded to the left by an oblique line passing from the junction of the left first rib and cartilage downwards and outwards through or beyond the left nipple, and on the right by a line running for the most part vertically somewhere between the right nipple and the median line of the sternum. The effect of pericardial effusion on the heart is to embarrass its action, to cause it to beat quickly, weakly, and irregularly, and to deaden its sounds. The pulse becomes correspondingly affected. The patient suffers from shortness of breath, pain or difficulty in breathing and palpitation, and not unfrequently, owing to interference with neighbouring organs, complains of fulness in the throat and difficulty of swallowing.

b. Pericardial adhesion, which is commonly the consequence of pericarditis, cannot always be recognised by either local or general signs. If it occur simply in patches, or if, being general, it be caused by a delicate adventitious lamina, there will probably be nothing whatever to indicate its presence. If, however, the accumulation of fibroid material be thick or dense, there will necessarily remain more or less permanent increase of the area of cardiac dulness, more or less disturbance of the heart's action, and more or less tendency to the development of the ordinary symptoms of chronic heart-disease. The local indications of adherent pericardium are mainly permanent extension of præcordial dulness, elevation of the apex of the heart and displacement to the left, and, it is said, recession of the thoracic walls over the apex of the heart at the time of systole in place of the normal protrusion.

c. Hypertrophy of the heart (as has been already shown) is probably always associated with more or less dilatation, always originates in overwork, and is in a very large proportion of cases developed, in obedience to its exciting cause, more largely on one side of the heart than the other. From these statements, it will be seen that the presence of hypertrophy and its distribution may generally be predicted from a knowledge of the existence of one of the recognised causes of hypertrophy; and further it may be gathered that cardiac hypertrophy seldom, if ever, exists in an uncomplicated form. The presence of hypertrophy is generally indicated by extension of præcordial dulness, prominence of the præcordial region, and powerful, heaving, diffused, cardiac impulse. If the hypertrophy be general, or involve mainly the left side, the apex of the heart gets displaced downwards and outwards, and may be found as low as the seventh interspace or eighth rib, and an inch or two outside the nipple; moreover, the pulse becomes hard and sustained, and the arteries get tense, and manifest a tendency to degenerate and yield. If hypertrophy affect the right side only or in chief part, epigastric pulsation becomes a prominent feature, and the apex beat is diffused and ill-defined. In this case the pulsation of the

systemic arteries is not necessarily affected, the tension is limited to the pulmonary vessels, and it is these which are, after a time, apt to become dilated and to degenerate. The cardiac sounds, and more especially the first, are said to be duller than natural in simple hypertrophy, but to become much increased in loudness when dilatation is associated with hypertrophy.

d. Feebleness of the heart is a consequence of numerous different kinds of lesions, such as dilatation, and degenerative changes of its walls; and is a late result of most organic affections of the organ. It is attended with more or less feebleness of the cardiac sounds and beats, and a corresponding condition of the pulse, which is sometimes increased in frequency, sometimes slower than normal, and often irregular. The patient moreover has difficulty of breathing, and palpitation, especially under excitement or on exertion; probably cardiac neuralgia; liability to faint; and venous congestion with tendency to rapid supervention of dropsy and the other usual consequences of heart disease. The symptoms are scarcely distinguishable from those of incompetence of the mitral valve, with which lesion, indeed, debility of the heart is often associated. Enfeeblement of the heart is one of the recognised causes of sudden death.

e. Aortic valve disease.—Obstructive disease or *stenosis* is characterised by the presence of a murmur which commences with the commencement of the heart's systole, and is continued onwards during the systolic silence. It is usually loudest over the right half of the sternum at the level of the third cartilage or third interspace, is very distinct over the ascending arch, and sometimes even in the back along the descending arch and upper part of the thoracic aorta; and it diminishes in force as it is traced from the base of the heart to the apex. The extent of its diffusion depends largely upon its loudness or pitch; when feeble it may be audible only over the valve and ascending arch. It is synchronous with the carotid pulse and cardiac impulse. The diagnosis of aortic valve disease is aided by the hypertrophic condition of the heart which attends it, and by the prolonged elevation of the systolic element of the pulse. In *aortic regurgitation*, the murmur which is produced commences with the second sound of the heart, which in some cases it entirely replaces, and is generally much prolonged, sometimes up to the very commencement of systole. It is usually most distinctly audible in the neighbourhood of the aortic orifice, and is carried thence downwards by the refluent stream towards the apex, often more particularly along the sternum, diminishing, however, in intensity in its passage, and sometimes undergoing some change of quality. Occasionally it is most distinct over the lower part of the sternum. It is in general rapidly lost along the ascending arch. If feeble, it may be detectable only over the valve and the adjoining portion of the ventricle. It occurs alternately with the carotid pulsations and the cardiac impulses. Its diagnosis is assisted by the fact of the heart being dilated and hyper-

trophied and by the character of the pulse. The latter has usually a peculiar jerky quality, which is due to a combination of sudden violence of the systolic wave with an equally sudden collapse at the beginning of the diastolic period—the latter being so sudden and extreme that the dicrotous rise is almost or entirely suppressed. This variety of pulse is usually termed Corrigan's, or the 'water-hammer' pulse.

f. Pulmonic valve disease.—A *systolic murmur* produced at the pulmonic orifice is heard loudest over the left edge of the sternum, or about the level of the third costal cartilage. It is heard also over the trunk of the pulmonary artery, namely, at or about the left edge of the sternum, as high as the upper border of the second cartilage. But it is inaudible, or nearly so, to the right of the sternum and along the ascending aortic arch, and fades away as it is traced downwards over the right ventricle. Organic murmurs at this orifice are rare, excepting as the result of congenital disease. The most common by far are anæmic. *Regurgitant murmurs* from defect of the pulmonic valve are of extreme rarity. They would naturally be best heard over the diseased valve, and thence downwards towards the right apex.

g. Mitral valve disease.—Of all murmurs the *systolic mitral*, or that due to regurgitation through the mitral orifice, is the most common. It attends the systole of the heart, and, therefore, like the direct aortic is synchronous with the carotid pulse. It is usually heard most distinctly, not immediately over the valve, but over that part of the left ventricle which is most superficially placed, namely, the apex. If feeble it may be audible in this position only, but, if loud it is often heard over the whole of the præcordial region. In the latter case it generally diminishes in force from the apex to the base; but occasionally increases again over the aortic orifice, or at that part of the left ventricle which, next to the apex, approaches nearest to the surface of the thorax. A systolic regurgitant murmur is carried back with the refluent blood into the left auricle; and partly on this account, partly because of the situation of the left ventricle to the left and back of the heart, it is generally distinctly audible about the angle of the left scapula, and along the horizontal line passing from this point to the apex of the heart—a fact of great importance in the recognition of this murmur. *Direct mitral* murmurs occur during the diastolic period, and until of late years were generally overlooked or misinterpreted. They are often absent because, although obstructive disease is not uncommon, the force with which the blood passes from the auricle into the ventricle is generally insufficient to generate a murmur. It is well known, however, to physiologists that during the earlier period of the ventricular diastole the blood is flowing almost passively through the auricle into the ventricle, and that it is only at the last, just before the ventricle itself contracts, that the auricle contracts and propels its blood with vigour. It is at this moment, therefore, that a murmur is most likely to be developed. It need scarcely be added that, when the auricle has become, as it soon does, dilated and

hypertrophied, and the time occupied in discharging its contents more or less protracted, the murmur is likely to be rendered both more intense and of longer duration. A diastolic mitral murmur, then, is audible during the ventricular diastole, but generally nearer its end than its beginning, sometimes indeed running up to the systolic sound, and apparently blending with it. More commonly the rhythm of the heart appears to be altered at the apex. The interval between the murmur and the first sound is so short that there is frequently a tendency, on listening at the apex, to reckon the murmur as the first sound, the true first sound as the second, and, from its indistinctness in the neighbourhood of the apex, either to disregard the true second sound, or to look upon it as a mere reduplication; or, if there be a systolic murmur, to take the second sound for an accentuated portion of it. From the usually peculiar relation of the diastolic mitral murmur to the ventricular systole, it is often termed '*præ-systolic.*' From the fact of its being determined by the auricular systole, Dr. Gairdner names it auricular systolic. There seems no good reason, however, why the name diastolic mitral should not be retained for it. This murmur is generally of short duration, somewhat deep-toned and rough, and to be heard over a very limited area at the apex of the heart, or a little to its inner side. It is very seldom audible in the back or at the base. It is important to note, that a præ-systolic murmur is often attended with a sensible thrill or purring sensation, that it is apt to be very irregular or unequal in its production, and that, above all murmurs, it is liable to disappear when the circulation is tranquil, and to become distinct when the heart's action is excited. In order to identify the præ-systolic murmur, it is essential either that the pulse should be felt while the heart is being auscultated, or that the sounds at the base and apex should be simultaneously examined by means of a double stethoscope.

In both mitral regurgitation and mitral obstruction, the ventricle tends to propel a comparatively small quantity of blood into the aorta at each systole, and consequently the pulse tends to be small and feeble, the arterial tension to be diminished, and more or less distinct dicrotism to be manifested. Further, the action of the heart, and consequently the pulse, soon become irregular.

h. Tricuspid valve disease.—Disease of the tricuspid valve is rare; it is also rare to have a murmur produced at this orifice. A *direct* murmur, or one attending the ventricular diastole, is of exceedingly infrequent occurrence. A *regurgitant* or *systolic* murmur is much more common; but this is more frequently due to over-distension of the ventricle or comparative shortness of the musculi papillares, and consequent inadequacy of the valves, than to their structural disease. It is sometimes observed in the displaced hearts of persons suffering from angular curvature of the dorsal vertebræ, in whom also the right ventricle is sometimes much hypertrophied. The murmur is generally somewhat low-toned, audible most distinctly about the ensiform car-

tilage, diminishing thence towards both the left apex and the base, and absent at the back of the chest. Tricuspid obstruction and regurgitation are attended with more or less obvious fulness of the systemic veins, especially those of the neck and upper arm; and not unfrequently distinct pulsation, apparently synchronous with that of the ventricle, may be distinguished in them and in the liver.

i. Hæmic murmurs.—But besides those due to valvular incompetence or impediment, other murmurs of functional origin are not infrequently observed. They are for the most part temporary, and are especially apt to occur when the heart is acting with unwonted violence, and in persons—more particularly young women—who are markedly anæmic. They are always systolic, and generally chiefly audible at the base of the heart, either to the right or to the left of the sternum. Occasionally they are observed at the apex. When heard at the base they probably arise in the commencement of the aorta or pulmonary artery. It is maintained, however, by Naunyn, and his views are adopted by Dr. Balfour, that these murmurs are really mitral regurgitant, and that they are heard in fact over the left auricular appendage, into which they are carried by the refluent blood-stream. If this were true, however, we ought to hear ordinary regurgitant mitral murmurs best in the same situation. When the murmurs are at the apex they are certainly regurgitant, and due probably to temporary dilatation of the ventricle, and consequent incompetence of the mitral valve.

7. *Prognosis of Cardiac Derangements.*

a. Our prognosis of pericardial effusion must depend largely upon what we know of its cause; and its causes we need scarcely say are numerous. We may point out, however, that when effusion takes place rapidly, as it does when an aneurysm or the heart itself ruptures into the pericardium the effects are remarkable: the cavity becomes rapidly distended, and the heart presently ceases to act, mainly, if not entirely, from its inability to contend against the compressing force to which it is subjected. When, however, effusion takes place slowly, the parietal pericardium undergoes gradual distension, and enormous accumulation may then ensue with only moderate embarrassment of the heart's action.

The consequences of adhesion of the pericardium are various; in many cases no influence whatever is exerted upon the muscular parietes or the action of the heart, and the patient continues in good health; in many cases, however, especially if the adhesions be abundant or thick, the action of the heart becomes more or less seriously embarrassed, and this embarrassment involves in some cases hypertrophy and dilatation, in some atrophy of the organ, and in either case aggravation of the patient's symptoms.

b. Hypertrophy is in most cases compensatory, and therefore

rather a benefit than an injury to the patient; dangers, however, follow in its train, the more important of which are dilatation of cavities, incompetence of valves, and degenerative changes in the muscular tissue of the heart itself and in the arterial system—all of them indications and sources of failing strength.

c. Whenever a diseased heart becomes also enfeebled, the symptoms from which the patient suffers are greatly aggravated. Weakness of the heart, indeed, whenever it occurs apart from and out of proportion to weakness of the general system, is always of grave import.

d. In attempting to estimate the relative prospects of life of patients suffering from the various forms of valvular lesions, many different matters have to be taken into consideration. Thus, if the affection be due to rheumatic inflammation, we know that the patient has special liability to a recurrence of his rheumatism, and consequently to aggravation of his cardiac malady; if the disease be the consequence of senile changes, we know that the valve affection must, in the nature of things, be progressive; and both in these and in other cases there is often something in the condition of the valves, only to be guessed at during life, which renders the danger of embolism always imminent. Again, the constant bodily or mental labour to which many sufferers are condemned necessarily influences symptoms unfavourably and hastens death; further, any conditions of failing health which tend to enfeeble the muscular walls of the heart tend, on this very account, to affect injuriously in a disproportionate degree the due action of the organ, and to expedite the fatal issue; and lastly, inflammatory and other affections of the lungs, which embarrass the pulmonary circulation, form especially serious and dangerous aggravations of all forms of heart disease.

But, putting aside all these sources of danger, which are more or less accidental, and common to most varieties of heart disease, the question remains, 'what, *cæteris paribus*, are the relative prospects of life of those suffering from the different valvular lesions?' and (it may be added) 'what are the special dangers to which they are respectively liable?' Obstructive disease at a valvular orifice is a much less serious matter than regurgitant disease, inasmuch as the hypertrophy of the muscular walls of the cavity behind becomes for the most part accurately adjusted to the increased work which is thrown upon them. The adjustment is often so accurate in the case of aortic valve obstruction, that persons thus affected occasionally live for years unconscious of the presence of disease. Indeed, this is certainly the least serious of all valvular lesions. Obstructive mitral valve disease, again, unless it be extreme, is pretty successfully counteracted by hypertrophy of the left auricle. Compensative hypertrophy of the auricle, however, can scarcely be so efficacious as that of the ventricle, since the absence of valves at the entrance of the veins allows the increased blood-pressure to be easily propagated backwards through the pulmonary vessels. It is certain, indeed, that in a large proportion of these cases symptoms

of cardiac disease manifest themselves before long; but, on the other hand, it is also certain that many persons who labour under congenital constriction of the mitral orifice live for many years, and for a large portion of their lives suffer little. No degree of hypertrophy can neutralise the effects of regurgitation. Indeed, it is questionable whether the hypertrophy which always follows on regurgitation is in any degree compensative of that regurgitation; whether, indeed, it is not to be regarded as the result of an effort to neutralise the virtual weakness which the dilatation, always attending regurgitation, causes. Aortic regurgitant disease is probably the most serious and rapidly fatal of all forms of valvular lesion. Regurgitant disease of the mitral is certainly less serious than the last, and patients often labour under it for many years; nevertheless it is probably more dangerous than obstructive disease of the same orifice. The order of danger in which Dr. Peacock places the four lesions which have just been considered, and we concur with him in this matter, is as follows: first, aortic regurgitant; second, mitral regurgitant; third, mitral obstructive; and fourth, aortic obstructive. It need scarcely be remarked, however, that this order is necessarily often departed from; that regurgitation (although productive of a murmur) may be so slight as to be of comparatively little moment; that obstruction may be so extreme as to lead to the rapid destruction of life. Diseases of the right side are so rare, and when present so often associated with lesions of the left side, that it is impossible, excepting theoretically, to estimate their relative degrees of danger.

We have previously discussed the various consequences of heart disease; and from what was then said the causes of death in patients suffering from valvular lesions may for the most part be determined. Sudden death, which was formerly so largely attributed to heart disease, is not a common sequela of valvular lesion. It is most common in regurgitant aortic disease, and in that case is due to syncope, or perhaps, as some maintain, to cardiac anæmia from non-filling of the coronary arteries.

8. *Treatment of Cardiac Derangements.*

a. The treatment of pericardial effusion will be best considered with the various morbid conditions on which it depends; that of embarrassment of the heart from adherent pericardium resolves itself mainly into that of enfeeblement of the cardiac walls, which will be referred to further on.

b. The treatment of simple cardiac hypertrophy is a matter of simplicity. We can only remove hypertrophy by removing or obviating the lesion which has provoked it, by maintaining the circulation in an equable and quiet condition by the avoidance of mental and bodily excitement or over-exertion, and by careful attention to the healthy maintenance of the functions of the body generally. It is, however, of

the highest importance to delay or prevent the supervention of that enfeebled condition of heart in which hypertrophy so commonly and disastrously ends; and this must be effected by promoting the general health of the patient; for which purpose iron and other tonics, change of air, and nourishing diet are often necessary.

c. The treatment of cardiac debility differs little, if at all, from that needed in the later stages of valvular, and more especially mitral valvular disease, a subject presently to be considered.

d. In treating valvular diseases we must never forget that we are dealing with affections which, in the nature of things, are incurable; that valvular defects tend, on the whole, to increase; that their ill effects tend gradually to become augmented by the changes which take place secondarily to them in the walls and dimensions of the cardiac chambers, and are always liable to serious aggravation by the presence of any condition, be it normal or morbid, which embarrasses the circulation. Our primary object must, therefore, be to prevent, or at all events to delay, the supervention of those numerous morbid processes and symptoms which have already been adverted to as the consequences of heart-disease. We cannot repair the injured valve. We cannot, and would not if we could, prevent the compensatory hypertrophy which ensues; we may, however, by forbidding excessive muscular exertion, or taking precautions against mental excitement, or other provocatives of increased cardiac action, prevent in many cases that hypertrophy from becoming excessive, and therefore injurious. We cannot prevent a certain amount of dilatation from taking place in association with hypertrophy; but by the same measures by which we counteract the one we tend also to counteract the other; and, further, since dilatation is to a large extent dependent on impairment of muscular strength, we may, by maintaining the general strength, maintain also to some extent that of the heart itself. Lastly, we may often succeed by careful attention in preventing the recurrence of inflammatory attacks, in arresting pulmonary and other congestions which react deleteriously on the heart, and in maintaining the quality and quantity of the blood in a fairly normal condition.

Hence a patient whose heart is diseased should abstain from all forms of violent and sustained exertion, and should never push even what seems to be moderate exercise to the extent of causing shortness of breath, or palpitation, or uneasy feelings of any kind, or even fatigue. His pursuits and surroundings should be such as do not entail mental excitement. He should be protected by proper clothing and other precautionary measures against cold. His bodily health should be maintained by the use of wholesome, nutritious, but not too abundant food, by the cautious employment of stimulants, and by carefully regulating the action of his emunctories.

But, notwithstanding the greatest care, a time comes sooner or later, and comes soon to those who are compelled to work hard for

their livelihood, when the consequences of the cardiac lesion become painfully apparent. The patient begins to suffer from palpitation, irregularity of pulse, shortness of breath, dropsy, jaundice, albuminuria, pulmonary apoplexy, angina. But even in these cases it is remarkable how often, under the influence of perfect rest and the other items of treatment which have been enumerated, all unfavourable symptoms subside. Indeed, in the treatment of the symptoms and consequences of valvular disease there is no doubt that absolute rest is of far more value as a remedial agent than anything else that can be named. But in aid of rest other agents may often be beneficially employed. Frequency of pulsation, and especially irregularity, are almost invariably connected with feebleness and irritability of the heart's action. To remedy this condition it seems desirable first to give strength to the heart's contractions, and next to diminish their frequency. For the former of these purposes iron and the vegetable tonics, and possibly nux vomica, are valuable; for the latter probably no drug, at any rate in mitral valve disease, is superior to digitalis. A combination of digitalis with iron is often of very great value. Belladonna is by many preferred to digitalis in the treatment of lesions of the aortic valve. To relieve the overloaded venous system, to which so many of the resultant phenomena of valvular disease are due, we may employ diaphoretics, diuretics, and purgatives, and besides these in some cases the removal of blood by leeches or cupping, or by venesection. Further, to relieve shortness of breath or engorgement of the lungs, or præcordial uneasiness, ether, ammonia, lobelia, stramonium, squills, ipecacuanha, or other expectorants, opium and counter-irritants may all of them, under slight modifications of circumstances, be of use.

II. PERICARDITIS, MYOCARDITIS, AND ENDOCARDITIS.

A. *Pericarditis.*

Causation.—Inflammation of the pericardium is evoked in various ways: by extension from the muscular walls of the heart when these contain abscesses; by extension from the pleura, peritoneum, cellular tissue of the neck, posterior or anterior mediastinum, or any other neighbouring part which is the seat of inflammation; by local injuries, such as penetrating wounds of the pericardium, or the opening of sinuses from hepatic or other abscesses into it; and by the rupture of aneurysms, hydatid cysts, and the like. The most frequent and important cause of pericarditis, however, is exposure to cold, especially if that exposure results in the development of rheumatic fever. Pericardial inflammation not unfrequently occurs in association with, if

not in dependence upon, chronic albuminuria, scarlatina, chorea, pyæmia, and occasionally in connection with tubercular, syphilitic, and carcinomatous or other malignant growths.

Morbid anatomy.—Inflammation of the pericardium, like that of all other serous membranes, is characterised in the first instance: by dilatation of the blood-vessels and consequent hyperæmia; effusion of their fluid contents into the substance of the serous membrane, and into the subserous tissue; and tendency to proliferation of the endothelium. At first, little more than simple congestion and œdematous thickening of the membrane is present. But soon inflammatory exudation takes place, consisting partly of fibrine, which as it is secreted coagulates upon the surface, and remains adherent to it or blended with it; partly of serum, which, containing dissolved albumen and fibrinogen, accumulates in the pericardial cavity, and separates one surface of the membrane from the other; and partly of inflammatory corpuscles, derived either from the proliferating endothelium or from errant leucocytes of which the majority remain entangled in the coagulating fibrine.

The relative quantities of solid and fluid exudation, their characters and the changes which they undergo, present great varieties. In some cases of pericarditis, which is thence often termed 'dry,' the whole surface becomes covered with a greater or less abundance of false membrane, but there is little or no accompanying serous effusion. In most cases, however, a few ounces of fluid are poured out in the course of the affection. And occasionally the accumulation amounts to one, two, or even three pints.

The solid exudation or false membrane forms in the early stage of its production a thin, slightly coherent lamina, which is scarcely distinguishable except from the fact that it robs the serous surface of its normal smooth glistening aspect. But it soon increases in quantity by the addition of fresh inflammatory matter to its free surface, and may thus by degrees attain the thickness of paper, cardboard, or of $\frac{1}{4}$ or even $\frac{1}{2}$ inch. As its thickness increases, so also as a rule do the density and closeness of adhesion of its deep surface, and the irregularity of its free aspect. At first the latter is merely faintly granular, but it soon gets more or less villous or tuberculated, or pitted with irregular and deepish holes. It is difficult to give in a few words a notion of the different appearances which may be presented; in some cases the surface is honeycombed; in others it is ribbed like the sand which the waves have just left; in others it has the aspect which may be produced by separating two hard smooth surfaces which have been stuck together with a layer of butter; in others again the exudation has been clearly rolled by the to-and-fro movements of the heart into cylindriform pellets, which remain irregularly attached to one or both surfaces of the pericardium. And further, irregular bands, festoons, or laminæ of the same material not unfrequently

extend in greater or less abundance between the visceral and parietal layers.

The pericardial fluid is sometimes limpid and colourless, almost like water, sometimes more or less opaline, and occasionally distinctly tinged with blood.

In many cases, no doubt, inflammation commences at some one spot or circumscribed area of the serous membrane; and, indeed, in mild cases it not very unfrequently remains thus limited, or at all events does not become general. More frequently the whole of the pericardium is involved.

In the great majority of cases of pericardial inflammation, resolution takes place after a longer or shorter period. The fluid which has been effused undergoes gradual absorption; the false membrane becomes organised, contracts, hardens, and ultimately is converted into a more or less imperfect form of connective tissue. In some instances circumscribed inflammatory patches result in the formation of those opaque, white, cicatrix-like thickenings which are so commonly met with on the surface of the right ventricle, and are known as 'milk-patches.' In some such cases the opposed pericardial surfaces become adherent at one or two points, or over a small area. But in by far the larger number of cases, when the inflammation has been general, the absorption of the fluid and the coming together of the inflamed surfaces end in their more or less complete coalescence, and in the obliteration in an equal degree of the pericardial cavity. The characters which the resulting adhesions display depend largely of course upon the quality and quantity of the false membrane from which they have arisen. Sometimes they are thin and delicate, and differ little from ordinary connective tissue. Sometimes they are thick, fibrous, and perhaps œdematous, and measure then maybe $\frac{1}{4}$ or $\frac{1}{2}$ an inch or more in thickness. Sometimes they are almost cartilage-like in density and hardness. Sometimes they become the seat of calcareous formations, which may constitute bands or patches of considerable extent.

In the course of pericarditis other results besides those which have been enumerated may take place. In some cases the newly-formed blood-vessels of the false membrane become ruptured, and blood in greater or less quantity is effused into its substance, or (if the opposed surfaces be not yet adherent) into the pericardial cavity. This hemorrhage may be so copious as to cause death. In other cases the inflammation becomes suppurative, and the pericardial cavity is converted into an abscess, which may ultimately contain two or three pints of pus. Suppurative pericarditis is often very chronic in its progress and sooner or later the pus may point and discharge externally in the præcordial region, or extend in other directions beyond the limits of the pericardium.

The inflammatory processes of pericarditis, when the attack is slight, are probably limited to the serous membrane exclusively; but

when the inflammation is intense or assumes a chronic form, it invades the deeper tissues, which then get congested and œdematous, and often, if muscular, degenerated and enfeebled. Hence it happens that the integuments of the præcordial region become in many cases distinctly œdematous; and it is perhaps occasionally owing to involvement and consequent enfeeblement of the intercostal muscles that the intercostal spaces are observed to bulge. It is a more important fact that, in a large number of cases, the outer layers of the muscular walls of the heart become to a greater or less depth obviously degenerated, softened, and weakened.

Symptoms and progress.—The symptoms of pericarditis are so commonly associated with those of the malady in the course of which it arises, and with those of endocarditis, which is so often developed in common with it, that it is not altogether easy to disentangle them entirely from those belonging to these other conditions. Pericarditis is in many cases so mild a disorder that it is attended with few or no symptoms of any importance. In other cases it is one of the most perilous maladies with which we have to deal, and its symptoms are correspondingly severe. But, between these extremes, cases of all grades of intensity are met with.

In its mildest form, pericarditis often entirely escapes detection, or is recognised only by the accidental discovery of pericardial friction; in most such cases, however, there is at some time or other some slight præcordial pain or uneasiness, together with extension of cardiac dulness and more or less obvious febrile disturbance. Most cases of what are termed 'latent' and 'dry' pericarditis belong to this group.

In describing the symptoms of more aggravated cases of pericarditis, it will be convenient to divide them into local and general, and to discuss these *seriatim*. The local symptoms are due directly to the condition of the pericardium and its influence on surrounding parts. The patient generally complains of pain and tenderness in the region of the heart. He winces if pressure be made over the præcordium, and still more if it be made in the epigastric region. The pain varies in character, is aching, cutting, burning, or a mere sense of soreness, and occasionally extends from the heart to the left shoulder and down the left arm. It is usually augmented by movement of the diaphragm, and hence the patient tends to breathe rapidly, shallowly, and with little abdominal motion. When the pain and tenderness are very severe, he usually lies upon his back, and, while moving his limbs with tolerable freedom, keeps his trunk almost entirely still. The roughening of the pericardial surface which takes place at the commencement of the disorder is attended with distinct friction sound, the characters of which have already been described. This usually commences at the base or apex, or along the right side, but soon becomes general; and having lasted for an uncertain time—a few hours, a day or two, or longer—slowly or rapidly vanishes. The further progress of the case

will alone determine whether this disappearance is due to adhesion having taken place, and is therefore permanent, or whether it depends on increase of fluid effusion and consequent separation of the pericardial surfaces from one another. In the latter case, the friction reappears with the absorption of the fluid, and its final disappearance, due to adhesion, is a subsequent event. It must be added that pericardial friction-sound is usually rendered more intense by the application of pressure to the præcordium, that its intensity is often distinctly modified by the movements of respiration, and, further, that pleuritic sounds developed along the edges of the præcordial region often have a distinct cardiac rhythm impressed upon them. Other phenomena of more or less importance which may often be observed are: œdema of the integuments over the cardiac region; a perceptible thrill, arising from the grating of the two rough pericardial surfaces upon one another, to be felt by applying the open hand to the cardiac area; and more or less complete masking of the normal heart-sounds by those of pericardial friction. It is scarcely necessary to add that all the phenomena (local and general) which have been previously described as belonging to pericardial effusion, are commonly added with typical completeness to those which have now been detailed, and indeed that they constitute an essential element in the clinical description of pericarditis.

The *influence of pericarditis* on the action of the heart and on the pulse is various. Early in the disease the heart itself may be little affected; more commonly its movements are increased in frequency, and the pulse is at the same time harder and fuller than natural. With the increase of effusion the beats of the heart become accelerated and diminished in strength; the pulse consequently gets small and feeble, and often irregular. Moreover, its rate is peculiarly apt to be increased by any slight excitement or muscular effort.

Among the general symptoms referrible to pericarditis are the following: first those of inflammatory fever, namely, increase of temperature, dryness of the tongue, thirst, loss of appetite, and scanty high-coloured urine; second, shortness of breath, often amounting to dyspnœa or orthopnœa, and frequent short, hacking cough; third, vomiting, a general aspect of distress, a look of anxiety, with pinched features and a pallid, or sometimes congested, countenance, weariness, want of sleep, tossing of the arms, irritability, rambling, and occasionally (especially towards the close of fatal cases) maniacal delirium, convulsions, or coma. The latter phenomena, however, which are certainly not unfrequently associated with pericarditis, seem almost invariably to have been observed in cases where the pericarditis was distinctly rheumatic, and where, therefore, it is possible that they may have been due to some other cause. Tetanic spasms and risus sardonicus also have occasionally been noticed in rheumatic pericarditis. Further it may be mentioned that in cases attended with much effusion, difficulty of swallowing from pressure on the œsophagus, congestion of

the head and neck from obstruction of the superior cava, and aphonia from compression of the left recurrent laryngeal have been observed.

Recovery from simple pericarditis is attended with the gradual subsidence of the symptoms which belong to the disease. In slight cases convalescence is often very rapid and complete. Generally, however, when there has been much pericardial effusion, and the symptoms have been severe, the amendment is slow; and more or less permanent ill-health is apt to remain. Pain, tenderness, cough, difficulty of breathing while the patient is at rest, and fever, gradually subside, the patient's appetite improves, and he begins to enjoy refreshing sleep. But the pulse frequently remains for a long while preternaturally quick, or on the other hand becomes slow and intermittent, and the præcordial prominence and increased dulness still continue excessive. Moreover, under these circumstances the patient often remains incapable of taking active exercise on account of the persistent ready development of cardiac uneasiness, palpitation, and shortness of breath. These symptoms also may in their turn subside more or less completely.

Adhesion of the pericardium can rarely be diagnosed with certainty in the absence of a distinct history of pericarditis. It is often attended, however, with more or less persistence of enlarged area of dulness, and permanent and unalterable elevation and displacement outwards of the apex beat, together perhaps with palpitation, dyspnœa, and some of the general symptoms of cardiac disease. Other occasional diagnostic indications are, retraction at the apex and of the præcordial intercostal spaces during the ventricular systole, and an impulse corresponding to the diastole. Moreover, a pericardium which has once been inflamed is apt under the influence of exciting causes again to become inflamed, notwithstanding the complete obliteration of its cavity.

Pericardial suppuration generally takes a chronic course. The commencement of suppuration may be attended with rigors and elevation of temperature. The former may recur from time to time; the latter probably continues; and soon the fever assumes a distinctly hectic type. The local phenomena are not always very well marked; there will probably be some persistence or increase of pain and tenderness, gradual extension of præcordial dulness, and augmenting distension of the præcordial region, with distinct and increasing œdema of the integuments.

Severe pericarditis not unfrequently ends sooner or later in death. If death occur during the height of the disease it may be the result of one or other of the cerebral complications which have been enumerated, or of asphyxia due to pulmonary complication; but in the majority of cases it is the consequence either of slow asthenia or of an attack of syncope. When death takes place at a later period, it is not unfrequently dependent on the gradual supervention of the ordinary consequences of heart disease—namely, pulmonary congestion with pulmonary apoplexy, or systemic venous congestion with anasarca, and

affection of the liver, kidneys, and other organs. Suppurative pericarditis is generally fatal.

B. *Myocarditis.*

Causation and morbid anatomy.—Inflammation of the muscular tissue of the heart rarely occurs except in connection with peri- or endo-carditis. In pericarditis, as we have already pointed out, a greater or less thickness of the muscular walls in contact with the inflamed serous membrane is often distinctly implicated; and there is no doubt that their inner aspect may be similarly involved during the course of an attack of endocarditis. It may even happen that in some situations the cardiac walls become thus affected in their entire thickness. Occasionally no doubt idiopathic inflammation arises, independently of inflammation of the serous membranes. It is said then to occur chiefly on the left side and towards the apex. It may however be more or less general. Sometimes pyæmic abscesses, or abscesses due to embolism, are found studding their substance. These are mostly small. But abscesses of considerable bulk have been described.

Inflammation of the muscular tissue of the heart presents the same pathological phenomena as inflammation of muscular tissue elsewhere. The affected parts become injected, there is a tendency to proliferation of the stationary protoplasmic elements, and to the escape of leucocytes and red corpuscles; and in connection with these phenomena the muscular fibres rapidly lose their striation, become granular and opaque, and break down. Not infrequently indeed this affection of the muscular fibres, together with more or less mottling and softening of tissue, irregularly distributed, is the only obvious indication of myo-carditis.

The early effects of inflammation are to diminish the cohesion of the affected tissues and to render them less resistant than natural. But subsequently, if resolution do not take place, they become contracted and hardened, and assume a cicatricial character. Under either of these conditions, especially if the morbid processes be circumscribed, yielding of the affected walls may take place, and the foundation of cardiac aneurysm be laid. When abscesses form they may burst into the pericardium, exciting inflammation of that membrane; or into the cardiac cavities, and thus evoke the phenomena of embolism or pyæmia. In many cases, no doubt, the inflamed muscle becomes completely restored.

Symptoms and progress.—It is impossible to assign any specific symptoms to myocarditis. Among those which are most likely to be present, are: more or less fever; debility of the heart with feebleness of impulse, of first sound, and of pulse; tendency to faint; difficulty of breathing, with oppression and uneasiness in the præcordial region; and nervous phenomena such as restlessness, giddiness, delirium, convulsions and coma. Death usually occurs suddenly from collapse or syncope.

C. *Endocarditis.*

Causation.—The causes of inflammation of the lining membrane of the heart's cavities are to a large extent identical with those which excite pericarditis and myocarditis. Most of the local causes, however, to which pericarditis may be due, can scarcely be operative upon the endocardium. Endocarditis is occasionally the result of the accidental rupture of valves or chordæ tendineæ; more commonly it depends on exposure to cold; but by far its most frequent cause is the presence of rheumatism. It may also be caused by extension from abscesses in the muscular parietes. Again, like pericarditis, it is often developed in connection with chorea and scarlet fever. A chronic form of endocarditis also may occur in connection with the syphilitic cachexia, chronic alcoholism, Bright's disease, and other affections inducing persistent dyscrasia.

Morbid anatomy.—In the great majority of cases endocarditis is limited to the left side of the heart, and to the valves or their immediate vicinity. Its presence is indicated by increased vascularity of the affected areæ; infiltration and inflammatory overgrowth of tissue, and consequent increase of thickness; and development of warty growths or granulations upon the surface. The thickening, which is mostly attended with opacity and softening, varies in degree, and, when it involves the thin curtains of the valves or the delicate chordæ tendineæ, causes them to become puckered or contracted. The granulations are in the first instance mere points; but they soon increase in size, sometimes becoming small bead-like bodies, sometimes papillary excrescences, sometimes rounded masses from the size of a tare up to that of a filbert. Frequently the neighbouring outgrowths coalesce to a greater or less extent, forming warty, botryoidal, or cauliflower-like masses, and in some cases pendulous fringe-like but irregular processes, which may attain a length of one or two inches. During the inflammatory process it is not uncommon for ulceration to take place. If this affect the valves it leads to their partial detachment, their attenuation at points, and the production of valvular aneurysms, or to their perforation; if it involve the tendinous cords, to their laceration.

When inflammation attacks the aortic valve the granulations which characterise it first appear as a fringe along the festooned inner margins of the lunulæ, but with the extension of disease they may cover to a greater or less extent the whole of the under surface of one or more of the cusps and even extend downwards on to the septum. They often, indeed, at length hang from the free edge of the valve, which then usually is thickened, contracted, and irregular in form. The aortic aspect of the valve is rarely the seat of granulations.

When the mitral valve is inflamed, granulations appear on its

auricular aspect a little within the free edge, whence they may extend over the greater part of that surface and thence on to the auricular walls. With the development of granulations there is usually more or less thickening and contraction of the free edge of the valve, and at the same time some contraction of the valve at its base, in virtue of which the orifice becomes diminished in capacity. The chordæ tendineæ also are apt to be the seat of granulations, to undergo thickening and shortening, and to become blended to a greater or less extent with the valvular curtains. Granulations are rarely met with on the ventricular surface of the valve.

Inflammation, when it attacks the valves on the right side of the heart, produces exactly similar effects to those above described.

Inflammation of the endocardium is not always acute, or always limited to the valves. In regurgitant aortic disease the surface of the septum ventriculorum, for half an inch or an inch below the valve, generally presents more or less cicatricial thickening, and occasionally marked contraction. The thickening is the result of chronic inflammation probably due to the constantly recurring impact of the refluent blood-stream against the ventricular walls in this situation. Again, we occasionally find, especially in connection with some forms of so-called 'atheroma' of the arteries, the lining membrane of the left ventricle studded with irregular patches of opaque thickening. These are due to hypertrophy, with more or less degeneration, of the endocardium, and are doubtless also of inflammatory origin.

Symptoms and progress.—The symptoms of endocarditis, apart from those of the disease (if any) with which it is associated, and of the lesions to which it gives rise, are neither striking nor serious. The symptoms, indeed, which are usually ascribed to this affection, are mainly made up of those of acute rheumatism and valvular obstruction or incompetence. And it must be admitted that it is by the development of the valvular lesions, which are an almost invariable accompaniment of endocarditis, that we mainly assume its presence and trace its progress. It is needless to say that the discovery of valvular mischief is no proof of the presence or even of the pre-existence of endocarditis. But if, in the progress of any one of those diseases of which endocarditis is a common complication, we detect a cardiac murmur which had not previously existed; and if further observation proves this to be a permanent phenomenon; or if changes in it indicative of increasing mischief take place; or if additional murmurs become developed; we cannot reasonably doubt that endocarditis is present. The same conclusion may be fairly arrived at when a young person, who is known to have been hitherto healthy, presents vague symptoms of ill-health, and reveals under the stethoscope a newly-developed and persistent valvular murmur. It is very important, however, to note that, in forming a judgment with respect to cases of this kind, there are many sources of fallacy to be avoided. We must

be careful, that we do not mistake a pericardial rub for an endocardial murmur; that we do not hastily assume that a murmur which we hear for the first time has not existed from some previous attack of rheumatism or from birth; and that we do not take a functional or anæmic murmur for one of organic origin. On the other hand, we must not too readily take it for granted that, for example, in a case of rheumatism, in which the heart is known to have been injured in some previous attack, the cardiac disease which we recognise is all of old date; we must not forget that direct murmurs due to granulations occasionally disappear; and, further we must always recollect that inflammatory vegetations may be formed on the valves, and more particularly on the auricular aspect of the mitral, which never impair the action of the heart and never give rise to abnormal sounds.

The remaining indications of the presence of endocarditis are slight and fallacious. From the position of the inflamed areæ it is scarcely possible that præcordial tenderness should be present; and, indeed, it is rarely if ever observed. More or less uneasiness or pain in the region of the heart may, however, be complained of. From the smallness of the extent of the inflamed surface we should scarcely expect much febrile disturbance; nor, as a rule, is simple endocarditis attended with marked fever. Still there may be elevation of temperature, thirst, scanty urine, and other indications of the febrile condition. Again, here, as in pericarditis, we may naturally look for some excitement or other modification of the action of the heart. It generally acts more frequently and powerfully than natural.

The prognosis of endocarditis is very serious. It is rare indeed for perfect recovery to take place. Moreover, the patient remains, for the most part, liable to fresh attacks of inflammation, and consequent increase of valvular lesion. The results of endocarditis are mainly those which have already been considered under the head of valvular disease, and will, of course, vary according to the valve affected, and the degree and kind of its affection, and need not be again discussed. But it must not be forgotten that it is in connection with endocarditis and its local consequences far more than with any other form of disease involving the endocardium, that detachment of solid particles or masses takes place which are conveyed as emboli to the brain, liver, spleen, kidneys, lungs, and other organs; and that the liability to this detachment has little or no obvious relation with the severity of the cardiac lesion. The subject of embolism will be fully discussed further on.

D. *Treatment of Inflammation of the Heart and Pericardium.*

In most cases of the several forms of cardiac inflammation which have been passed in review, the affection is developed in the course of other diseases, such as rheumatism, Bright's disease, and pyæmia; for

which the patient is already under observation. The treatment, therefore, of these maladies forms an essential element in the treatment of the heart affections which complicate them. It is important, however, to consider whether any, and if so what, additional measures may be adopted in reference to the cardiac lesions.

In the treatment of pericarditis the abstraction of blood is generally regarded as a most important remedial measure. Blood may be taken by venesection from the arm; but it is probably most conveniently, and best, removed from the præcordial region by cupping or leeching. To be efficacious, blood-letting should be performed early, while the symptoms are yet acute; and should be, so far as is compatible with the patient's age and condition, free, in order to obviate as much as possible the necessity for its repetition. A dozen or twenty leeches may be applied to the chest of an otherwise healthy adult, and the bleeding subsequently encouraged by fomentations or poultices. In slight cases at an early period, and in severe cases after removal of blood, counter-irritation is of considerable value. It relieves pain and uneasiness, and probably promotes the absorption of fluid. A large mustard plaister, or cotton-wool saturated with turpentine or spirits of wine, and covered with some impermeable tissue, may be applied to the præcordium; or iodine paint or blistering fluid may be painted over the part; or simple fomentations, as hot as the patient can bear them, may be persisted in. There is, it may be observed, a practical objection to the use of applications which blister the surface: namely, that they interfere with that frequent examination of the cardiac region which is so important. Of the value of opium in this, as in almost all other inflammatory affections, there can be no doubt. It may generally be safely administered, and in large doses; excepting, perhaps, when the heart shows signs of great enfeeblement, when the circulation is embarrassed, the respirations rapid and shallow, and the skin dusky. When these latter phenomena supervene, ammonia, ether, alcohol, and other stimulants are indicated. In order to reduce inflammation, and remove the products of inflammation, it was formerly deemed essential to put patients under a course of mercury or iodide of potassium. These remedies, however, are probably inefficacious except in certain constitutional conditions. Again, diuretics and purgatives have been largely advocated for the purpose of removing fluid accumulations from the serous cavities. But there is little proof that they have any appreciable influence in this respect. It may, nevertheless, be useful when febrile temperature is present to employ some of those agents—namely, aconite, veratrum, or quinine—which are known to reduce temperature. But the most efficient means of effecting the removal of dropsical accumulations is to improve the patient's general health. And on this and other grounds it is always important to bring him under the influence of tonic treatment as soon as the condition of the digestive organs allows of its employment.

The above remarks as to treatment relate more immediately to pericarditis. But they are to some extent applicable to endocarditis. It must be borne in mind, however, that local bleeding and local medication of all kinds are necessarily less efficacious in endocarditis than in the other; and, further, that as endocarditis is (except in its remote consequences) a far less dangerous, and severe affection than pericarditis, a far less active plan of treatment is generally needed.

When, in pericarditis, the accumulation of fluid appears to be seriously interfering with the action of the heart, especially if it persist despite all treatment; or when we have reason to suspect the presence of pericardial suppuration; the question whether paracentesis should be performed for the removal of the fluid will perforce present itself. The operation is one which has been performed neither frequently nor with much success; moreover, it is an operation of considerable delicacy and difficulty; still it can scarcely be doubted that it should be attempted under the above circumstances. The chief danger to be avoided is that of puncturing the heart, the next that of wounding the internal mammary artery. To avoid the former danger it is important first to determine accurately the lateral boundaries of the distended pericardium, and next to satisfy oneself, by the presence or absence of sensible impulse, over what area (if any) the heart is in contact with the anterior thoracic parietes, and then carefully to make an opening into that part of the pericardium from which the heart seems to be remote. The mammary artery runs down behind the costal cartilages, a little outside the sternum. The most eligible spot for puncture is usually towards the inner extremity of the fourth or fifth intercostal space close to the sternum. It is probably the safest plan to divide the soft tissues with the scalpel one by one until the parietal layer of the pericardium is reached, and then to puncture carefully with a fine trocar and cannula. If serum escape the entrance of air should be prevented; if pus, it may be advisable to wash out the cavity, and even to inject a weak solution of chlorinated soda or Condy's fluid. In some cases it may be well to make a preliminary puncture with a fine aspirating needle.

III. MORBID GROWTHS AND PARASITES.

A. *Fatty Growth.*

The presence of a small quantity of fat upon the surface of the heart, mainly in the course of the transverse and longitudinal sulci, is extremely common, especially in persons who have attained middle life, or who present a general accumulation of fat throughout their

connective tissue. This condition is of no importance. But occasionally, in persons of great obesity, fatty growth becomes excessive, and encroaches seriously upon the substance of the heart, not only investing the organ more or less completely, but invading the substance of its walls, separating the muscular fibres from one another, and imparting to the walls in places (more especially in the right ventricle) the softness and general aspect of simple fat.

The *symptoms* referrible to this affection (which is sometimes described as a form of fatty degeneration) are those of cardiac feebleness and incompetence.

B. *Tubercle.*

Tubercle is of infrequent occurrence, and generally takes place in connection with widespread distribution of the disease. Miliary tubercles are occasionally found imbedded in the substance of the muscular walls. Their most common seat, however, is the pericardial serous membrane. In this situation they may occur in small scattered groups only, or may be thickly and pretty generally distributed; and, especially in the latter case, are often associated with more or less abundant inflammatory exudation. Cheesy tubercle in considerable masses, and generally associated with thick and dense adhesions, is also occasionally observed in the pericardium.

The *symptoms* of cardiac and pericardial tuberculosis are generally lost in those of more advanced tubercular disease of other organs. If, however, they be sufficiently pronounced to attract attention, they are indistinguishable from those of subacute or chronic pericarditis.

C. *Syphilis.*

Syphilitic affection of the heart is not uncommon. The condition which is now very generally regarded as such is characterised by the presence of fibroid infiltration, of greater or less extent, of the cardiac walls; with imbedded caseous masses, somewhat closely resembling the so-called 'knotty' tumours of the liver; and with more or less indurated thickening and adhesion of the pericardium. True gummata of recent formation have also been observed. Microscopically, the diseased tissues present, as do those of gummata developed in voluntary muscles, overgrowth of the interstitial connective tissue, with more or less fatty or caseous conversion of certain parts, in which the involved muscular fibres share. The disease may implicate any part of the heart, but most commonly affects the ventricular walls. Sometimes it forms tumours, which project from the outer aspect of the heart, or encroach upon its cavities; sometimes it leads to thinning of certain parts, and to aneurysmal dilatation. It must be added that fibroid change of the cardiac walls may be due to other causes than syphilis, to chronic

inflammation for example, and that the specific origin, therefore, of all such cases must not be hastily assumed.

The conditions here spoken of may, at any rate in a clinical point of view, be combined. They are chronic in their progress; and are not unfrequently associated with adhesion of the pericardium, lesion of the valves, and hypertrophy, dilatation or other modifications of the walls or cavities of the heart. The symptoms, therefore, which they induce, although liable to considerable variety of detail, are essentially those of chronic heart disease, and mainly of those conditions or stages of disease in which the heart is enfeebled and incompetent to carry on the circulation efficiently. Dropsy is of common occurrence, and sudden death not unfrequent. The disease occurs almost exclusively among persons of middle or advanced age.

D. *Malignant disease.*

This affects the pericardium, as it does other serous membranes, only much less frequently. It may occur here in the form of miliary granulations, circular plates, or nodulated outgrowths. It is always secondary, and probably never attains sufficient proportions to cause obvious symptoms. Malignant disease of the muscular walls of the heart is also not common, and is probably always of secondary origin. Generally it occurs there in the form of small imbedded tumours, which are of no practical importance. Occasionally, however, it forms masses, as large as a hen's egg or orange, which encroach on the cavities or orifices of the heart, and constitute a more or less serious impediment to the circulation. In some instances, sarcomatous and other growths, originating in the posterior mediastinum, involve the heart by continuity; they steal, as it were, along the vessels at the base, and then gradually infiltrate the muscular parietes of the auricles and ventricles, separating the muscular fibres from one another, and causing general increase of thickness. In these cases no tumours may be developed, and microscopic examination may be needed for the detection of the nature of the morbid process which has been going on. Among the varieties of malignant disease which have been found involving the heart and pericardium may be mentioned scirrhus, encephaloid, melanotic cancer, lymphadenoma, and sarcoma.

Malignant disease of the heart and pericardium has rarely, if ever, been diagnosed during life, and indeed rarely gives evidence of its presence by symptoms referrible to the heart. It is obvious, however, that the symptoms to be looked for are those indicative of cardiac obstruction and weakness, and that the supervention of such symptoms in the progress of malignant disease might suggest the possibility of cardiac involvement.

E. *Parasites.*

These are very seldom met with in connection with the heart. The *trichina spiralis* has never been found in it. The *cysticercus cellulosæ* has been discovered there, but not as productive of symptoms. *Hydatids* also have occasionally been observed, varying from the size of an orange downwards, and either imbedded in the substance of the muscular walls, or occupying the subserous tissue of the visceral pericardium.

The *symptoms* to which hydatids when they give rise are those: either of interference with the due performance of the cardiac functions; of suppuration, to which such cysts are liable, of pericarditis, dependent on extension from the inflamed cyst, or on its rupture into the pericardium; or, lastly, of the discharge of the hydatid contents into the interior of the heart.

F. *Treatment.*

It is impossible to lay down rules in regard to the treatment of cases in which the heart is involved in adventitious growths or the seat of parasites. The symptoms which they are likely to induce are mainly those of cardiac debility and incompetence, and the treatment must be adapted to the symptoms which are present. It may be said, however, generally, that diffusible stimulants and tonics are indicated.

IV. DEGENERATIONS.

A. *Degenerations of the Muscular Walls.*

Causation and morbid anatomy.—We have already pointed out that, under the influence of starvation and various wasting diseases, more especially phthisis, the heart becomes remarkably diminished in bulk. But this change is due to atrophy alone, the muscular fibres undergoing simple attenuation, without structural change.

Of actual degeneration, three varieties are generally described:—namely, *fatty* or *yellow* degeneration, *granular* or *brown* degeneration, and *fibroid* degeneration.

1. *Fatty degeneration* in an advanced condition is indicated by softness of the affected tissues, opacity, a peculiar pale buff colour, and, it may be, obvious greasiness. Under the microscope, the muscular fibres are found to have lost, in a greater or less degree, their natural striation, to be studded with minute refractive oily molecules, and to be, as a rule, more friable than in health. In the early stage it sometimes happens that the oily particles occur only at the poles of the nuclei of the muscular fibres, or arranged in longitudinal strings; but with the

progress of the disease they get more numerous; and in extreme cases the fibres lose all their normal characteristics and are converted into opaque, irregular cylinders of accumulated fatty particles.

Fatty degeneration occurs under various conditions. It is frequently the result of inflammation, and when developed in connection with pericarditis occurs more especially in the layer of muscular fibres immediately subjacent to the visceral pericardium. It is sometimes observed in acute diseases, especially in certain fevers, and in poisoning by phosphorus. We have seen it remarkably developed in a child that died of acute purpura. It is a common condition of advanced life, especially if this be attended with certain diseases or morbid tendencies, such as heart disease, chronic bronchitis, Bright's kidney, hepatic disease, arterial degeneration, or gout. It is common also, mainly in old age, as an immediate consequence of obstructive disease of the coronary arteries or of any other morbid condition impairing the vitality of certain portions of the organ.

When the degeneration occurs in connection with inflamed serous membrane, the affected lamina appears to the naked eye anæmic, and in other respects but little altered. When it is due to general disease or to disease influencing the heart generally, the whole organ may become pallid and softened; but more frequently the tissues are mottled with fattily degenerated spots or patches—a condition which is very often peculiarly distinct in the carneæ columnæ and on the inner surface of the ventricles. When the degeneration is secondary to obstructed arteries, it usually occupies a more or less distinctly circumscribed region which presents, as a rule, remarkable softness and friability.

2. *Granular degeneration* is generally distributed uniformly throughout the muscular tissue of the heart, which assumes a brownish hue. The muscular fibres are studded with longitudinal strings of brownish particles, the exact chemical constitution of which is not known. The circumstances which determine this form of degeneration seem to be the same with those to which general fatty degeneration is also due.

3. *Fibroid degeneration* affects portions only of the cardiac walls, and is comparatively common on the right side. The affected tracts are greyish, dense, and hard—changes which are due in different degrees to overgrowth of fibroid tissue and to wasting of the muscular fibres, and their conversion into, or replacement by, fibroid tissue. The change is probably often undistinguishable from the consequences of syphilis; but is sometimes a sequela of myocarditis. Again, hypertrophy of the heart, and especially that form of it which is secondary to Bright's disease, is often made up partly of overgrowth of muscular tissue, partly of overgrowth of the intervening connective tissue; and in some cases the latter element becomes disproportionately abundant, and the heart consequently, in a sense, degenerate and enfeebled.

Symptoms.—The symptoms of degenerative affections of the muscular walls of the heart are mainly those of cardiac weakness and in-

competence; such especially as dyspnœa, lividity, tendency to syncope, indistinctness of the first sound of the heart and weakness of pulse, which may be quick, slow, irregular or variable. To these must of course be added the other usual consequences of defective or impeded circulation. Enfeeblement from degeneration is one of the recognised causes of sudden death; and it is an important fact that sudden death is liable to occur in those in whom degeneration is not yet far advanced, and who have not yet presented definite symptoms of cardiac disease. Rupture of the heart is not uncommon in those cases in which local softenings from arterial obstruction are present.

B. *Degenerations of the Valves and Endocardium.*

Causation and morbid anatomy.—Fibroid, fatty, and calcareous changes, or degenerations of the endocardium, are among the most frequent causes of heart disease. For the most part they come on with advancing years, and may be regarded (with the corresponding conditions of the arterial system) as some of the chief consequences and indications of senile decay. They are apt, however, to manifest themselves even in early adult life, especially in those who have lived intemperate or over-laborious lives, or have suffered from syphilis, or are the subjects of chronic Bright's disease. They are also apt to supervene on ordinary endocarditis; and hence it is often difficult (except from the history) to distinguish between degenerative lesions of primary origin and such as are the consequences of bygone acute endocardial inflammation. It must be remembered, however, that the changes, which are here roughly grouped together as degenerations, probably for the most part take their origin in a form of chronic endocarditis—a subject which will be more fully discussed when we come to speak of endoarteritis and degeneration of arteries.

Degenerative changes may manifest themselves at any point of the endocardial surface; but far more frequently involve the valves than other parts. The lining membrane of the left ventricle is more commonly affected than that of the other cavities; and the aortic and mitral valves far more commonly than the valves of the right side. In some cases the valves present simply a few opaque, buff-coloured (atheromatous) patches; in some they manifest more or less general fibroid thickening—a condition which is usually accompanied by a greater or less amount of contraction, and often by some fatty or calcareous deposit; in some cases they are rendered thick, nodulated, and irregular, from the accumulation of combined fibroid, fatty and calcareous deposit, and then, if the disease be far advanced, project as more or less rigid processes across the orifices to which they belong, become blended to a greater or less extent with one another at their bases, and reduce the valvular aperture to a mere chink; in some cases, again, the degenerate tissue undergoes erosion, excavations form, and

finally perhaps the valve gets perforated or ruptured. These changes generally are not strictly limited to the valves; they are apt to be prolonged from the aortic to the aorta or the septum ventriculorum, and from the mitral to the chordæ tendineæ, which become thick, short, and sometimes incorporated with one another. The chordæ tendineæ, like the valves, occasionally get lacerated.

Symptoms.—It is obvious that the conditions here described may produce all varieties of valvular defects, singly or in combination; and more especially the same defects as commonly result from acute endocarditis—namely, obstructive and regurgitant disease of the aortic and mitral orifices. The changes are chronic, and the symptoms which they induce creep on insidiously; so that it often happens that a patient has had the disease upon him for years before its presence is distinctly revealed. Indeed, the first clear indication of heart disease is sometimes due to the sudden rupture of a valve, or some other untoward complication or event; and we are often astonished to find post mortem how extreme a degree of contraction of the aortic or mitral orifice has been compatible, not merely with life, but with life passed in comparative ease and comfort.

The early symptoms of degenerative disease of the valves are usually vague, comprising, perhaps, some degree of irregularity of the pulse, more or less shortness of breath, occasional neuralgic pain or uneasiness in the region of the heart, attacks of giddiness or faintness, and not unfrequently more or less impairment of the digestive functions. It must be added that, inasmuch as the cardiac affection is usually associated with degenerative changes in the arteries and even in other tissues, the symptoms due to these become mingled with those of the heart disease, and may to some extent aid our diagnosis of the actual condition of the heart. Among such indications may be mentioned the presence of rigid or otherwise diseased arteries, as revealed by the condition of the pulse or by cerebral symptoms, and the existence of the arcus senilis. The symptoms of the declared disease are mainly those of the valvular lesions which have been already fully considered. The chief practical point to be remembered is that, however slow the symptoms may have been in attaining serious development, the morbid processes on which they depend are in the nature of things progressive and tend surely to a fatal issue.

C. *Degenerations of the Coronary Arteries.*

The coronary arteries and their branches are peculiarly liable to all those degenerative changes which affect the lining membrane of the heart and arterial system. Their parietes consequently become thickened with fatty or calcareous deposit, and their channels reduced in size or obliterated. The latter conditions involve the imperfect nutrition of the parts to which the affected vessels lead, and induce those

localised fatty changes, attended with discolouration of tissue and softening, which have already been adverted to.

No specific *symptoms* can be referred directly to disease of the coronary arteries. Angina pectoris has been asserted to occur with special frequency in these cases. But it must be recollected that arterial degeneration is usually present in a greater or less degree in persons advanced in years, and is then usually associated with other cardiac degenerations.

D. *Treatment.*

Degenerative conditions of the heart, as of other organs, call for all measures—tonic, alimentary, and hygienic—calculated to maintain or improve the general health; but they also need special precautions and special items of treatment, according to the particular phenomena and dangers which each case presents. These have been sufficiently indicated on an earlier page under the head of the treatment of valvular derangements.

V. ANEURYSM OF THE HEART.

Causation.—In addition to that general dilatation of the heart's cavities which has been previously considered, partial dilatations or aneurysms are occasionally met with. They have been oftener observed in men than in women, and for the most part at an advanced period of life. They are not uncommon, however, during middle age, and occur, indeed, though with extreme infrequency, in children.

Localised dilatation obviously depends on comparative feebleness of that portion of the cardiac wall which undergoes dilatation, and its inability to resist successfully the internal pressure to which it is subjected. The cause of weakness is doubtless different in different cases. In some dilatation seems to arise in ulcerative destruction of the lining membrane, or in laceration and breaking down of more or less of the muscular wall; but in most it is apparently due to the presence of one of those forms of enfeeblement which have just been passed in review, namely, fatty, fibroid, or some other variety of degenerative change. It seems obvious, therefore, that it may be a consequence of endocarditis and myocarditis, either in their acute or chronic forms, and of syphilis. Not improbably also it occasionally originates, as do arterial aneurysms, in the effects of very violent muscular exertion or of violence inflicted from without.

Morbid anatomy.—Cardiac aneurysms now and then occur in the right ventricle and even in the left auricle, more especially at the foramen ovale; but by far their most common seat is the left ventricle.

They are generally said to affect chiefly the apex of this cavity; but they may originate at any spot within it. In size they range from that of a pea to that of the heart itself. In form they may be: a simple hemispherical expansion of the apex or some other part; or flask-like, communicating by a comparatively small orifice with the ventricular cavity; or sacculated, consisting of a series of intercommunicating chambers imbedded in the substance of the walls, and extending over a more or less considerable area. Their parietes vary in thickness, and are sometimes as thin as paper; and generally (especially if the aneurysm be of large size or old date) consist, more or less completely, of dense fibroid material, with little or no trace of muscular tissue. Occasionally they undergo calcification. Cardiac aneurysms sometimes are empty, sometimes contain laminated or other forms of coagulum. As regards their results, they seem occasionally, after having reached a certain size, to remain stationary, or nearly so; but they tend ultimately to undergo laceration, and thus to cause communication between the left ventricle and one or other of the auricles, the right ventricle or the pericardium. In their progress towards the surface they not unfrequently cause pericardial inflammation, and adhesions, which both delay rupture and limit its effects.

Among cardiac aneurysms must be included those of the valves and coronary arteries. Valvular aneurysms occur chiefly in the aortic and mitral valves, but occasionally in the tricuspid, as the result of inflammatory or degenerative weakening or erosion; and they constitute bulgings of various sizes, which, in the case of the aortic valve, project into the ventricle, in the case of either of the auriculo-ventricular valves into the auricle, and usually sooner or later rupture, and thus allow of free regurgitation.

Aneurysms of the coronary arteries are rare. They are generally developed in the trunks, at a short distance from the aorta, and form small tumours in the transverse sulci. Occasionally numerous small aneurysms stud not only the trunks, but also many of the larger branches. Like cardiac aneurysms, they may open into the pericardium, cardiac cavities, or large vessels at the base of the heart.

Symptoms.—Cardiac aneurysms for the most part are never suspected to be present until the occurrence of rupture causes either grave symptoms of cardiac disease, or death from escape of blood into the pericardial cavity. There are no special symptoms by which their presence is indicated. They are of course frequently attended with some of the usual symptoms of chronic heart-disease; and, no doubt, when the tumour is large and so situated as to come into relation with the anterior thoracic parietes, the presence of a pulsating tumour distinct from the heart may occasionally be recognised.

VI. RUPTURE OF THE HEART. EFFUSION OF BLOOD INTO THE PERICARDIUM.

Causation.—Perforation of the muscular walls of the heart may be due to accidental or other violence; with such cases, however, the physician has little or nothing to do. Spontaneous rupture is an affection almost exclusively of advanced age; it sometimes occurs in the floor of an aneurysm, sometimes in a heart generally weakened by degenerative changes, but more frequently in a circumscribed patch of softening, due to atheromatous disease and obstruction of the artery, which supplies it; and it is generally immediately traceable to some muscular effort or mental disturbance. Men are more liable to it than women.

Morbid anatomy.—Spontaneous rupture occurs almost without exception in the walls of the left ventricle, and mostly in front. It generally forms in the direction of the muscular fibres an irregular rent, or series of rents, which pass irregularly through the walls, and present considerable differences of size, form, and position, on the inner and outer surfaces respectively. The lacerated tissue, moreover, is generally infiltrated to a greater or less extent with blood. The consequences of laceration of the heart, though in all cases death ultimately ensues, present a good deal of variety. In some instances (especially in cardiac aneurysm) the actual rupture into the pericardium is often preceded by the formation of pericardial adhesions; in some the rupture occurs primarily into the connective tissue beneath the visceral pericardium; and in both of these cases the effusion of blood is at first circumscribed, and the patient may sink, not however suddenly from copious hemorrhage, but slowly with the symptoms of pericarditis. In some instances the rupture occurs directly into the pericardial cavity, which then becomes more or less rapidly distended with blood. The pericardium is then found post mortem to be full of blood—partly serum, partly a bag of undecolourised coagulum in which the heart is enclosed, and by which it is concealed; the heart, moreover, is found empty, flattened, and more or less wrinkled on the surface, as if it had been subjected to considerable pressure.

Symptoms and progress.—The symptoms of rupture of the heart are far from uniform. In a large number of cases the patient is attacked with severe pain in the region of the heart, gasps for breath, faints, and dies in the course of a few minutes, or even a few seconds. In some cases he is also attacked with sudden severe cardiac pain, faintness, and dyspnœa, but rallies to some extent; and then, passing into a condition of extreme collapse, attended with remarkable feebleness of pulse, coldness of extremities, profuse sweats, anxiety and restlessness, sighing respiration or extreme dyspnœa, and great op-

pression, constriction, or pain at the chest, dies at the end of some hours. In some cases again (and these are they in which adherent pericardium or other circumstances delay or prevent the impletion of the serous cavity with blood) the symptoms which mark the occurrence of laceration subside, and the patient returns apparently to a state of more or less complete health; upon which, at the end of a few hours, or perhaps a few days, either sudden death occurs from the discharge of blood into the pericardium, or pericarditis becomes developed, and sooner or later carries him off.

It need scarcely be said that the phenomena which attend the rupture of aortic aneurysms into the pericardial cavity are identical with those which have just been described.

Other ruptures of the heart besides those of its outer muscular walls may take place; thus, either the septum of the ventricles or that of the auricles may become perforated, the musculi papillares or chordæ tendineæ may be broken, or the aortic, mitral, or other valves torn from their attachments or split. Such lacerations occur spontaneously probably in those cases only in which there has been previous weakening from disease. The aortic valve chiefly suffers in this respect, and the tendinous cords of the mitral. The consequences of these lesions are obvious: in the first two cases, a more or less free communication will be established between the auricles or ventricles; and in the others regurgitation of blood from the arteries into the ventricles, or from the ventricles into the auricles will be set up or augmented. The symptoms here will be mainly those of advanced valve disease; and the nature of the accident on which they depend may possibly be diagnosed, partly by the sudden occurrence or aggravation of the patient's symptoms, partly by the circumstances under which this sudden occurrence or aggravation took place, and partly by auscultatory signs.

VII. HYDRO-PERICARDIUM.

Dropsy of the pericardium, like hydro-thorax or ascites, is one of the incidents of general dropsy. It may depend also on local causes, such as obstruction of the coronary veins, and the growth of tubercles or cancer. A greater or less degree of it is of common occurrence. The amount of serous fluid present rarely exceeds half a pint, and is often not more than one or two ounces. It is insufficient, indeed, as a rule, to cause obvious symptoms or to be discoverable during life. Hydro-pericardium, however, like other varieties of dropsy of serous cavities, may become excessive, and hence not only embarrass the movements of the heart, but reveal its presence by the physical indications (which have been already discussed) of fluid accumulation

in the pericardial cavity. When, however, it becomes thus extreme, there is generally reason to suspect its association with some degree of pericardial inflammation.

Hydro-pericardium rarely, if ever, demands special treatment. Counter-irritation of the præcordial region, and the treatment of the condition on which the dropsy depends, are the chief measures to be adopted. It is conceivable that paracentesis might be needed.

VIII. SYNCOPE.

Causation.—The ætiology and symptoms of syncope have been discussed in an earlier part of this work, to which we refer the reader. With reference, however, to the heart's share in its production we may make a few additional observations here. The cardiac failure (which always takes place to some extent) is commonly referrible to causes, mental or physical, operating through the nervous system; the heart becomes more or less completely paralysed, and contracts feebly or not at all upon its contents. In some cases, however, its failure to act depends upon the presence of some mechanical impediment to its action, as when it is compressed by rapid serous effusion into the pericardium, or by the escape of blood into that cavity, or as when sudden obstruction of one of the cardiac orifices by a clot or embolus takes place, or the patient is suffering from obstructive valve disease. Hearts enfeebled either by dilatation or by fatty or other forms of degeneration, or by abundant or dense pericardial false membranes, are especially liable to failure of action, and are necessarily more liable than others to suffer under the influence of those causes of failure which have been previously enumerated.

Treatment.—A patient suffering from syncope should be placed in the horizontal position, all ligatures should be removed from the neck and elsewhere, and he should be freely exposed to cool fresh air. Ammonia, or other such stimulants, should be held to the nostrils; ammonia, ether, or alcohol administered by the mouth; or, if they cannot be swallowed, these or turpentine should be given in the form of enemata; cold water should be dashed in the face, either from a jug or by means of a wetted cloth or towel, and sinapisms applied to the epigastrium and to the limbs. If death seems imminent, it is important to promote the action of the lungs and heart by frictions, and it may be necessary to employ artificial respiration, to stimulate the heart by galvanism, or, if the veins be distended, to bleed from the external jugular vein. If syncope be the result of profuse hemorrhage, the question of transfusion naturally arises. Whenever the syncopic condition assumes a chronic form it is important to maintain the bodily temperature and

to prevent the patient from making any kind of exertion. Then, too, the gradual improvement of the patient's vital powers by the judicious exhibition of nourishment, and the assuagement of vomiting and all other symptoms which tend to impede this improvement, become objects of the highest importance. The value of iron and other tonics in promoting restoration to health, and of opium or chloral hydrate in remedying sleeplessness, excitement, or delirium, need scarcely be insisted upon.

IX. PALPITATION. GRAVES'S DISEASE. (*Exophthalmic goitre.*)

A. *Palpitation.*

The phenomena of palpitation, so far as they involve the heart and vessels only, have already been adverted to. They comprise increased frequency of cardiac action, suddenness of impulse, together with, not unfrequently, some irregularity or intermission.

The symptoms which attend palpitation are throbbing of the heart and arteries, noises in the ears, muscæ, giddiness, faintness, hurried respiration, præcordial uneasiness and anxiety, flushing of face, coldness of extremities, clamminess of surface, together with which are often associated rushing sounds or murmurs at the cardiac orifices, in the larger arteries, and even in the larger veins of the neck.

The conditions under which palpitation occurs are very numerous. Among them may be mentioned: mental excitement; excessive bodily exertion; indigestion; the influence of certain articles of diet or luxury, more especially strong tea, and tobacco; anæmia and debility, however produced; hysteria; gout; and, besides these, the presence of actual cardiac disease. In many of the cases here enumerated the palpitation is occasional only, and disappears wholly with the removal of the condition on which it depends. But sometimes it assumes a chronic character. The heart is then apt to get dilated and hypertrophied; and these very changes tend to maintain or aggravate the conditions out of which they arose.

B. *Graves's Disease.*

Definition.—The most remarkable cases of persistent palpitation are those described by Graves and Basedow, in which, together with palpitation, there is enlargement of the thyroid body, and exophthalmos or protrusion of the eye-balls.

Causation.—These associated phenomena are most commonly met with in young women above the age of puberty; they are seldom observed in girls of younger age, and seldom originate in advanced life. Men are affected much less frequently than women. The patients are,

in some cases, anæmic or hysterical, but by no means invariably so. Sometimes the commencement of the disease dates from an attack of fever, or is attributed to mental shock or over-exertion. Occasionally it ensues on organic lesions of the heart. By some it has been contended that the cardiac disturbance precedes and is the cause of the goitre and exophthalmos. But against this view is the fact that palpitation, more or less long-continued, is constantly met with in persons who never have any apparent tendency to affection of either the orbit or the thyroid body. Others have regarded the goitre as the primary lesion, and have referred the cardiac and other symptoms to its influence, exerted either by pressure on the arteries of the neck or in some less obvious manner. It is sufficient, however, in opposition to this view, to point out that Graves's disease is sometimes present without thyroid enlargement, and that palpitation and exophthalmos are not specially common among the goitrous inhabitants of goitrous districts. The proximate cause, indeed, of the disease is very obscure. Nevertheless there are many circumstances which render it probable that the collective symptoms are due to some affection of the sympathetic system, which allows of passive dilatation of the vessels of the neck, thyroid body and orbit, and at the same time of excited action of the heart. Many of the symptoms, in fact, closely accord with those producible either by paralysis or by functional disturbance of the sympathetic. Moreover various observers have described, in fatal cases of the disease, morbid conditions of the cervical sympathetic.

Morbid anatomy, symptoms and progress.—The symptoms of Graves's disease may come on suddenly or gradually. In the latter case the patient probably first complains of violent and frequently repeated cardiac palpitation, together with distressing pulsation of the arteries in the neck. After these phenomena have existed for an indefinite period, changes are observed in the eyes and thyroid body. The affection of the eyes, if not actually prior in point of time to that of the thyroid body, is generally perceived earlier. At first the change is slight, and evident only to those to whom the patient's healthy aspect is familiar. The eyes are a little more prominent, glistening, and staring than they were. But gradually their prominence becomes more and more pronounced, until they protrude so far through the eyelids that these are unable to close in sleep, and even at ordinary times are so widely separated that the corneæ are visibly encircled by the sclerotics. Occasionally even the insertions of the recti muscles can be clearly distinguished. The exophthalmos is generally equal on both sides. Occasionally, however, it begins unilaterally, and, even when both eyes get involved, continues most pronounced on one side. It is a curious fact that inflammation rarely attacks the insufficiently protected eyeballs; and that sight remains for the most part unaffected, excepting, perhaps, that the patient is troubled with muscæ, becomes

long or short sighted, and suffers from fatigue in using the eyes. The protrusion of the eyeballs is often variable to some extent, increasing under the influence of palpitation, or excitement, and at the menstrual periods; and it appears to be due either to accumulation of fat, or of fat with increase of connective tissue, in the orbits, or to dilatation of the orbital vessels, or to these conditions combined in various proportions. It is often attended with aching or throbbing in the orbits, and not unfrequently subsides wholly after death.

The enlargement of the thyroid body is for the most part very gradual; and attention is generally first directed to it by the continued presence of pulsation in the lower part of the neck. It then causes merely a slight fulness in the usual situation of the gland, but more especially on the right side, and is subject to variations in degree; sooner or later however a manifest tumour results. This may be symmetrical, or may continue to be a little larger on the right than on the left side, but very rarely attains a large size, or produces injurious effects by pressure on neighbouring parts. This form of goitre is generally softer than ordinary goitre, and is often attended with a thrill or distinct pulsation, perceptible to the patient as well as to the examiner, and with more or less distinct arterial or venous murmur. It has, indeed, more than once been mistaken for aneurysm. Its size, like the prominence of the eyes, is liable to change.

The condition of the palpitating heart varies somewhat. For the most part its action is violent and rapid, and its sounds loud and ringing; its area of dulness is often somewhat increased. In most cases it is at first structurally healthy, and so it may continue. Often, however, the persistence of palpitation induces more or less hypertrophy and dilatation, especially of the left ventricle. A functional systolic murmur is not unfrequently audible at the base, and murmurs are often audible also in the arteries and veins of the neck. Occasionally, as has been already intimated, the phenomena of Graves's disease supervene on actual cardiac disease; and it may be added that, in most cases in which post-mortem examinations have been made, more or less atheromatous change has been detected in the arterial system.

The phenomena above described are not the only ones that are commonly presented in this affection. It has been especially observed that the patient is liable to be irritable, fretful, peevish, incapable of application, and to suffer from sleeplessness; that her appetite is capricious, often voracious; that she suffers from flatulence, and at one time from constipation, at another from diarrhœa; that there is a disposition to febrile excitement, with elevation of temperature by one or two degrees, and that this condition may be associated with the presence of Trousseau's 'cerebral macula;' and that there is generally amenorrhœa, and not unfrequently leucorrhœa. Anæmia and cachexia are also sometimes present. In some cases enlargement of the spleen has been observed; and in some, enlargement of the mammæ.

It should be added that, in the early stage of Graves's disease, palpitation, with throbbing of the vessels in the neck, may be present without obvious thyroid-gland or eye affection, and that in some cases the goitre, in some the exophthalmos, may never become developed.

Graves's disease is not usually dangerous to life. Occasionally patients recover entirely; more commonly there is partial amendment only; and in a large number of cases the disease is slowly progressive, and at best after a while becomes stationary. When death takes place it is mostly the consequence of some intercurrent affection, more especially of the lungs. In rare cases the thyroidal tumour causes death by pressure on the trachea.

Treatment.—There is considerable difference of opinion as to the treatment of this disease. Some recommend, Trousseau condemns, the use of iodine. Iron is generally strongly advocated; both Trousseau and Von Gräfe, on the other hand, regard it as injurious. Depletory measures, and even the removal of blood, have been lauded. Digitalis in largish and frequent doses is said to be exceedingly valuable in promoting contraction of the dilated and pulsatile vessels, and in thus relieving and curing the disease. On the same principle ergot of rye or lead may be supposed to be indicated. Belladonna again seems to act beneficially. Cold applications to the thyroid body and to the præcordial region are said to be very serviceable. If the patient be anæmic, or suffer from amenorrhœa, want of sleep, or any other condition calculated to cause or maintain ill-health, special treatment will of course be needed.

X. CARDIAC NEURALGIA. ANGINA PECTORIS.

Causation.—The causes which induce cardiac neuralgia are numerous, but for the most part such as affect the circulation either through the nervous system or by muscular exertion. Among them are: mental excitement, such as anger, or any sudden impression of pain or pleasure; intemperance in eating or drinking; active exercise, especially ascending a hill or staircase, and straining at stool; in some cases even a blast of cold air. The attacks are often brought on by mere walking exercise, and not unfrequently occur during sleep. Cardiac neuralgia is of frequent occurrence in heart disease and cases of aortic aneurysm, and under these conditions may be met with at any age and in either sex.

Pathology, symptoms, and progress.—Neuralgia of the heart is referred primarily and mainly to the præcordial region, and occurs therefore chiefly to the left of the sternum, but sometimes involves the sternal and right mammary regions as well. It varies in severity; is aching, burning, or indescribable; but is generally attended with a sense

of constriction, dread of breathing deeply, and anxiety. It may radiate down into the lower part of the abdomen, up into the root of the neck, or backwards to the spine; but is specially characterised by a tendency to extend to the left shoulder, and thence downwards along the inner side of the upper arm to the elbow. Not unfrequently it spreads to both shoulders, thence to both elbows, and thence again to the wrists and even to the tips of the fingers. Occasionally it involves the lower extremities similarly. The abnormal sensation which extends along the arms and lower limbs is sometimes an aching, sometimes a sense of tightness or constriction, sometimes a tingling, and not unfrequently a mere numbness. In connection with these symptoms, the affected limbs, as also the face, suddenly become pale and cold; to which conditions venous congestion and clammy sweats are apt presently to succeed. During the height of the attack the patient often becomes giddy and faint, and sometimes falls into a state of insensibility which may be attended with convulsions.

Attacks of cardiac neuralgia vary in their intensity, duration, and frequency of recurrence, and in the conditions under which they occur. They may be so slight as to consist in nothing more than a momentary pain or uneasiness in the region of the heart, with some extension of uneasiness to one or both shoulders. They may be so severe that the patient suffers, and has the appearance of suffering, indescribable agony, with the overpowering dread of impending death. He suddenly becomes still, fearing even to breathe; clutches whatever is near him for support; or, assuming some strange attitude which experience has taught him, grovels on all fours, lies upon his chest, or sits astride a chair with his face to the back, and his head bent over it. The attacks may last from a few seconds to many hours. In the latter case, however, their continuance is due to the repetition of paroxysms which are for the most part of no great intensity. Sometimes a patient has one attack only; or he has a succession of attacks at intervals, and then no more; sometimes the first is fatal; more commonly the affection commences comparatively slightly, with attacks succeeding one another at long intervals, but gradually the intervals become shorter, and the attacks more severe, and recur on slighter and slighter provocation.

When cardiac neuralgia occurs independently of distinct cardiac lesion, it has received the name of '*angina pectoris*.' This is rarely met with below the age of forty or fifty, and is far more common in men than in women. In the majority of cases, too, it has a marked tendency to recur at gradually shortening intervals and with increasing severity, and sooner or later to prove fatal. Occasionally, however, the disease manifests itself in young persons; and occasionally also (and more particularly in them) complete recovery takes place eventually. After death from angina pectoris, various lesions have been detected; and these (the more important of which are calcification of the coronary vessels, and fatty and other degenerative affections of the muscular

tissue of the heart) have been regarded as its cause. In other cases the heart has been found to be perfectly healthy. But it is obvious that such lesions as are here adverted to can only act, if they act at all, as predisposing causes. What, then, is the proximate cause? It has been assumed to be spasm or cramp of the muscular tissue of the heart; and in favour of this view it may be observed how intense is the agony which may be produced by the spasmodic action of the bowels, uterus, or voluntary muscles. The character of the pulse has been described as being sometimes weak and scarcely perceptible; at other times, slow, full, and strong. There is reason, however, to believe, both from the pallor and coldness of surface which attend the onset of the attack, and from sphygmographic observation, that an essential feature of the disease is sudden and extreme contraction of the systemic arteries, which both prevents the free passage of blood to the capillaries, and, damming it up, as it were, in the heart, excites that organ to unwonted but more or less fruitless efforts.

Treatment.—The treatment of cardiac neuralgia, or angina pectoris, must be partly prophylactic, partly directed to the relief of the spasmodic attacks. It is of the greatest importance that the patient should avoid or obviate all those conditions which are apt to produce the affection; that he should eschew all mental and bodily exertion or fatigue; and that indigestion and all other functional derangements should be as far as possible prevented by careful attention to diet and appropriate remedial measures. For the treatment of the anginal attack various remedies have been suggested. Among the most valuable are the diffusible stimulants—ammonia, ether, and brandy—and narcotics, such as opium and belladonna. During an attack, diffusible stimulants are probably the most useful. Faradisation to the cardiac region has been attended with good results. Dr. Brunton, guided by the fact of the spasmodic contraction of the arteries which attends, if it do not cause, angina, has tried the inhalation of nitrite of amyl (which relaxes the muscular walls of these vessels) during the paroxysm with striking benefit. He applies five or six drops to the nostrils on a rag or piece of blotting paper. This method has since been largely employed and with marked success.

XI. CYANOSIS AND MALFORMATIONS.

A. *Cyanosis.*

Causation.—Lividity or blueness of the skin is a frequent symptom of those diseases or conditions in which the due aëration of the blood is interfered with, and especially, therefore, of some forms of lung and heart disease. It may be met with, consequently, in all cases in which impediment exists to the passage of air along the larynx or trachea;

in all cases also in which there is obstructive disease of the bronchial tubes, whether it be bronchitis or any other affection; and in all cases in which, whether from emphysema or other organic lesions, or from pulmonary congestion or œdema, the free transmission of blood along the pulmonary capillaries, or the free admission of air into the air-cells, is interfered with. It is a striking characteristic of cholera; in which disease, either from alteration in the blood or from contraction of the smaller branches of the pulmonary artery, the blood ceases to pass in quantity through the pulmonary capillaries. Lastly, it is very frequently observed in cases of heart disease, more especially of the right side, and in cases of congenital malformation.

Symptoms and progress.—It is in the last class of cases, indeed, that the condition commonly known as cyanosis is most frequently present —cases in which the blueness first manifests itself at birth, or within a few weeks, a few months, or very rarely a few years after that event. We shall describe it as it presents itself in these cases. The blueness of surface varies in depth, but is always most pronounced in the cheeks, lips, tongue, and extremities of the fingers and toes. Here the natural rosy hue may merely present the slightest possible inclination to purple, or the parts may be purple, blue, or almost black. The general surface is dusky, or livid, and ghastly. The tint varies from time to time; it gets intensified under the influence of exertion, mental excitement, exposure to cold, or catarrhal or other like affections of the respiratory organs; and in some cases it almost entirely subsides during times of comparatively good health and perfect quiescence. The conjunctivæ are mostly congested, œdematous, and glistening; the lips, and perhaps the nose and eyelids, are tumid; but the most remarkable degree of tumefaction is always manifested by the terminal phalanges of the fingers and toes, which become strikingly thickened and enlarged, or bulbous. The circulation is feeble, the surface (especially that of the extremities) generally cold, and the patient disinclined, and, indeed, unable to engage in active exercise. He is liable to paroxysmal attacks of difficulty of breathing, during which his cyanosis increases, and he not unfrequently passes into a state of syncope; and he is very apt to suffer from congestive and inflammatory affections of the respiratory organs. He is generally sluggish in body and mind, and his temper is for the most part irritable and fretful. Dr. Peacock says that the internal temperature of cyanotic patients is not below that of healthy persons.

Pathology.—It was not unnatural to assume that the cyanosis of malformation is due to the admixture of arterial and venous blood, which takes place in the great majority of these cases, through an incomplete ventricular septum, a patent foramen ovale, or a persistent ductus arteriosus. But cyanosis has been proved to exist in an intense form in cases of malformation where no such admixture was possible, and to be absent from many cases of malformation in which the com-

munication between the venous and arterial sides of the heart was unusually free. We are hence driven to the conclusion that cyanosis must in the main be due to the same causes as determine lividity in other forms of heart disease, namely, impeded transmission of blood through the lungs, and consequently insufficient aëration, with over-accumulation of blood in the systemic veins. If this be the true explanation, it may fairly be asked what are the distinctive marks by which typical cyanosis is distinguishable from ordinary cardiac lividity? And it must be acknowledged that the differences are of degree or detail only, and are probably due to the fact that the veins of young children yield more readily under the continued strain to which they are exposed than do those of adults. Cardiac lividity in adults rarely attains that depth of colour which we often meet with in cyanotic children; and the bulbous enlargement of the fingers and toes which is so common in the latter cases is seldom observed as a consequence of acquired heart disease.

B. *Malformations.*

Causation and morbid anatomy.—The subject of cardiac malformations is one of great interest and extent, and impossible of adequate discussion in a work like the present. Yet it cannot be wholly ignored. We proceed, therefore, to make a few remarks upon it.

The auricles form originally a single cavity, and the separation between them is effected by the development of a vertical septum, of which the fossa ovalis represents the last-formed portion. This septum may be wholly absent; or the fossa ovalis alone may remain more or less patent, as it is at birth; and between these extremes every degree of defect may be observed. The ventricles, also, constitute, in the first instance, one cavity, which, in the course of development, becomes divided into two by the growth of a partition from the apex of the organ upwards, the last-formed part therefore being that which lies just below the arterial orifices. This septum also may be wholly or in part absent. In the latter event the deficiency is almost always found immediately below the valves. The bulbus arteriosus, again, in the first stage of its development is a single cavity continuous with that of the common ventricle, and becomes like that, by the growth of an independent septum, divided into two portions, of which one becomes the aorta, the other the pulmonary artery. It is possible for this separation never to be completed; it is possible that one of the arteries may be imperfectly developed or get impervious; it is possible also for them to be transposed, so that the pulmonary artery becomes continuous with the left ventricle, the aorta with the right. Further the ductus arteriosus, which is patent up to the time of birth, and allows the aortic blood to be distributed freely to the branches of the pulmonary artery, may remain patent.

Various valvular defects, for the most part causing obstruction, are also of frequent occurrence. Lastly, many of these malformations may coexist, and indeed the appearance of one defect in the course of development usually necessitates the supervention of others at a later period.

Defect of either the ventricular or the auricular septum to a slight extent does not necessarily allow of any material admixture of venous and arterial blood, or involve discomfort or danger to life; if, however, the communication be free, the aërated and non-aërated blood-streams become more or less considerably commingled, and serious symptoms may result. It is obvious that similar consequences will ensue, in a more or less aggravated form, under various other circumstances; as, for example, when the tricuspid orifice is contracted or obliterated, and all the blood that enters the right auricle has consequently to pass through the foramen ovale into the left auricle, and thence into the left or it may be common ventricle, previous to its distribution; or when, owing to relative displacement of an imperfect septum ventriculorum and of the orifices of the pulmonary artery and aorta, both vessels seem to spring from the right ventricle; or when, assuming also the septum of the ventricles to be incomplete, the aorta or the pulmonary artery is contracted or impervious, and in the one case the pulmonic circulation is effected from the aorta through the medium of the ductus arteriosus, in the other the systemic circulation is maintained through the channel, afforded by the trunk of the pulmonary artery and the ductus arteriosus, between the heart and the descending arch of the aorta.

Symptoms and progress.—In the various forms of malformation which have here been passed in review, there is very often some disproportion in point of size between the ventricles, and some hypertrophy of their muscular parietes; consequently there is generally during life some increase of præcordial dulness, some modification of its form, and some increase in the area and force of the cardiac pulsations. Further, there is, in a large number of cases, a more or less loud and rough systolic murmur, audible with greatest distinctness over the left third costal cartilage or somewhere between this point and the left nipple, and, according to its degree of intensity, perceptible over a restricted area only, or over the whole præcordial region and beyond it.

The general symptoms which attend malformations of the heart are (if certain valves only be affected) those mainly of obstructive disease of those valves; if however, in addition to valvular obstruction, there be other congenital defects, or if, independently of valvular lesions, these other defects are sufficiently serious to cause symptoms, the patient presents in a more or less aggravated form the phenomena which have been described under the head of cyanosis.

The prospects of life in children born with malformed heart are very gloomy. The great majority die in the first few weeks after

birth. A small proportion of them survive to the period of puberty. Very few, however, who are markedly cyanotic attain adult life. The chief causes of death, according to Dr. Peacock, are: cerebral disturbance resulting from defective aëration of the blood and congestion of the brain; and imperfect expansion, collapse, and engorgement of the lungs.

C. *Treatment.*

The treatment of cases of malformation should be mainly hygienic and prophylactic. Patients should be protected by warm clothing against vicissitudes of temperature, debarred from all active bodily exercise and mental excitement, and sustained by nourishing diet. Their digestive organs and emunctories should be maintained as far as possible in a healthy condition.

Section II.—DISEASES OF THE ARTERIES.

I. ARTERITIS.

A. *Periarteritis.*

Causation and morbid anatomy.—The outer tunic of the arteries, and to some extent the middle and even the internal tunic, may be regarded as merely modified portions of the general connective tissue. They are directly continuous with it, and, as might be supposed, readily share in its diseases. Hence, when a district of the body is in a state of inflammation, the walls of the arteries which are comprised within it also become inflamed; and occasionally, indeed, inflammation may attack these more violently than other parts, and may travel along them far beyond the limits of the primarily affected area. Such inflammation is usually limited to the outer tunic, and involves the others (if at all) comparatively late and to a slight degree. It is characterised by congestion, infiltration, and thickening of the affected parts; is sometimes attended with the development of pus in and around the outer arterial coat; and occasionally with ulcerative destruction or necrosis of the middle and inner coats, and consequent perforation. From the very slight extent in which usually the internal coat is implicated, it but rarely happens that the lining membrane loses its polish, or that thrombosis takes place.

The *symptoms* to which this form of arteritis gives rise are more or less pain and tenderness, hardness and induration, along the affected vessel, and some degree of inflammatory fever. The formation of abscesses, the plugging of the artery, and its perforation, would severally

produce special symptoms. These, however, are matters which will be more conveniently discussed hereafter.

B. *Endoarteritis.*

Causation and morbid anatomy.—But, besides that form of inflammation which commences from without, we not unfrequently meet with inflammation which originates in the lining membrane, and tends to remain limited to that membrane, or at least involves the outer coats later and by simple extension only.

The causes of primary endoarteritis are somewhat obscure. In some cases it is due to the irritation of a thrombus or impacted embolus; in some to the effect of long-sustained excessive blood-pressure (as in Bright's disease), or to the continued violence of the impact of the blood-stream on certain points. It appears, too, in many cases to depend on cachectic conditions of the system, referrible to long-continued exposure, deficiency of food, intemperance, syphilis, and the like. Indeed, it may be asserted that syphilitic disease of arteries is, at least in many cases, scarcely if at all distinguishable from endoarteritis.

Endoarteritis of the larger vessels is indicated by the development in the substance of the internal coat of translucent wheal-like thickenings which project to a greater or less extent into the vascular channel. They have rounded or irregular margins, and often coalesce so as to form patches of considerable extent, which then present nodulated surfaces. They may be scattered singly in small numbers, or may involve extensive tracts, rendering the surface of the vessels remarkably uneven; and they are particularly apt to appear at the points of bifurcation of vessels, or at the points of junction of branches with the trunks from which they spring. When the affection is the consequence of thrombosis, and when it occurs in minute arteries, it often causes uniform thickening of considerable superficial extent. The thickening is due to inflammatory proliferation of the protoplasmic elements of the internal arterial tunic; and it may be observed that, according to Cornil and Ranvier, the acute form of the disease is distinguishable from the chronic by the fact that in it the proliferation begins at the surface, which is consequently roughened, while in the latter it takes place chiefly in the substance of the tunic. After a while, the muscular coat becomes involved, degenerates and loses its contractile power; and when the inflammation implicates the whole thickness of the vessel the walls become generally thick and translucent.

Syphilitic endoarteritis, more especially as it occurs in the cerebral arteries, has been closely studied by Heubner, who shows: that it begins with proliferation of the endothelium; that gradually a growth forms which encroaches on the channel of the vessel, and consists of more or less stellate and flattened cells; that the innermost of these are in close contact and arranged as an endothelium, while the outer ones are more

loosely aggregated, and separated from one another by some amorphous matter, and a greater or less number of leucocytes; that after a time capillary vessels, continuous with those of the external arterial tunic, are developed in the growth; and that it ends in cicatricial contraction, and does not, like ordinary endoarteritis, undergo fatty change. The morbid process is limited to certain arteries, and affects them irregularly. Dr. Greenfield [1] shows, that the disease may attack the external as well as the internal coat, and that it may spread thence to the middle tunic.

Symptoms.—Endoarteritis may, as has been hinted, be acute or chronic, but there are no special symptoms by which its acuteness or chronicity can be distinguished; and, indeed, endoarteritis becomes chiefly important and distinguishable by the consequences, mainly mechanical, to which it leads. These (which will be elsewhere more fully considered) are referrible to irregularity, rigidity, degeneration and weakening of the arterial walls, diminution or occlusion of the channel, dilatation or aneurysm, ulceration and rupture; to which may be added the consequences of the deposition of fibrine upon the roughened surface, and of the formation of granulations or pendulous fibrinous polypi. When endoarteritis occurs in superficial arteries, in consequence of thrombosis or embolism, considerable pain and tenderness are experienced in the course of the affected vessel. It follows, therefore, that pain may be a symptom of the endoarteritis of deep-seated vessels.

II. DEGENERATION OF ARTERIES.

Causation and morbid anatomy.—Primary fatty degeneration of arteries frequently comes on with the advance of years. It is recognised by the presence of irregular opaque yellowish spots, apparently in the substance of the internal membrane, which may be sparsely scattered, or so abundant as to produce a general mottling. But although it commences in the internal coat, it soon involves the middle coat to a greater or less extent. Microscopically it is found that the cells of the affected regions are the seat of more or less abundant fatty deposit. They gradually become entirely destroyed, and with the progress of the disease the intervening tissues, including the elastic elements and muscular fibres, undergo disintegration.

But more frequently fatty degeneration constitutes a late stage of endoarteritis. The translucent or cartilage-like nodules become more or less opaque, generally in their interior, owing to the fatty transformation of the cells of which they are in so great a degree composed. And after a while the interior of the growth may break down into an

[1] Path. Trans., Vol. xxviii., p. 249.

opaque pulp, containing abundant fatty molecules, degenerate remnants of tissue, and cholesterine. A small abscess-like cavity is the result. Or, as in the primary affection, the fatty degeneration may commence superficially and thence gradually invade the whole of the diseased patch.

Whether the degeneration be primary, or secondary to arteritis, there is a tendency after a while for the degenerated structures to break down and be discharged into the vascular channel. When the disintegration begins superficially, the affected surface gets eroded, and an ulcer-like cavity results. When, on the other hand, the softening mass is at first separated from the blood-stream by a layer of coherent tissue, perforation after a while takes place, and the escape of the detritus through the orifice results in the formation in the substance of the arterial walls of a flask-like cavity, which maintains a free communication with the arterial channel.

But fatty degeneration is not the only degenerative change which occurs. In a large number of cases, more especially chronic cases or those of persons advanced in years, the precipitation of calcareous matter accompanies the fatty process. Calcareous molecules are deposited in the tissues which intervene between the fattily degenerating cells; and the result may be the formation either of amorphous tuberculated calcareous lumps, or more frequently of thin, more or less transparent plates, which are curved in conformity with the curvature of the vessel, and which, though usually covered in the first instance by a thin membranous lamella, soon get denuded. Further, they tend to separate at the margins, and after a time to be shed wholly or in part, and to leave ulcer-like excavations behind. Calcareous plates may be scattered irregularly and in small numbers, or may be so numerous and large as to render the vessel in which they occur a rigid bone-like cylinder.

There is yet another form of calcareous degeneration which is occasionally met with in arteries of medium and small size. It is not attended with, or consecutive to, fatty degeneration, but is due to calcareous transformation of the muscular cells of the middle coat. The capillary arteries occasionally undergo complete conversion into calcareous cylinders.

The degenerative processes above described, although for the most part originating in, and implicating mainly, the internal coat, tend sooner or later to involve the middle coat also; and, even if this present no visible structural change, it becomes after a time more or less impaired as to contractile power and capability of resistance. With certain exceptions which have been specified, degeneration affects the aorta (especially its arch) far more frequently than other vessels. Yet none enjoys immunity. The pulmonary artery, however, is comparatively rarely affected.

Symptoms.—The presence of arterial degeneration cannot always be recognised with certainty. It causes rigidity and therefore loss

both of elasticity and of contractile power. If superficial vessels be implicated, their condition may often be readily recognised by the finger; if the larger and deeper-seated trunks be involved, the loss of their elasticity renders the systolic throb of the pulse prolonged and its cessation sudden; and, further, this same loss of elasticity adds to the resistance which the heart has to overcome, and tends to induce hypertrophy of that organ. The more serious and striking consequences of arterial degeneration are the same as have already been adverted to in connection with arteritis, and will be best discussed under subsequent headings.

III. ANEURYSM. (*Dilatation of Arteries.*)

Definition.—The terms dilatation and aneurysm are of common use as applied to diseased arteries. By dilatation we generally mean a uniform or somewhat uneven enlargement of the channel of some considerable length of vessel; by aneurysm, a comparatively abrupt enlargement of a more or less distinctly circumscribed tract. The term aneurysm is, however, also applied to certain tumours which consist of bundles or convolutions of simply dilated arteries.

Causation.—Aneurysms, in the more restricted sense of the word, are bulgings caused by the pressure of the blood within vessels on walls which have been weakened either by the effects of accidental or other injury, or by the progress of the degenerative changes which have just been considered. The pressure which the blood within the arteries ordinarily exercises on their walls is amply sufficient to cause bulging and aneurysm at points in which their resisting power is impaired. It need scarcely be added that when that pressure is greatly increased, as it is habitually in Bright's disease, and intermittently in violent muscular efforts, its effect on diseased arteries is necessarily proportionately augmented; and indeed, under some such conditions, tracts of even healthy arteries may undergo considerable and permanent dilatation. The influence of violence in the production of aneurysm is very important, whether we regard it as acting through the medium of the blood-pressure, or directly on the vessels by strain. Its importance is shown: by the frequency with which aneurysms occur in those vessels which from their situation are specially exposed to violence; the frequency with which they occur in those persons whose avocations demand excessive muscular exertion; and the frequency also with which the origin of aneurysms is distinctly traced back to some unwonted effort or injury. The starting-point of the aneurysm is then some laceration, probably of the middle coat, or, if the artery be already diseased, some injury to the degenerated tissue. But in the

great majority of cases the aneurysm commences in a region already diseased, and probably independently of any undue pressure: the passive and enfeebled wall slowly yielding before the normal dilating force to which it is subjected. The surface left by the erosion of an atheromatous patch, or the detachment of a calcareous plate, or the cavity produced by the discharge of a quantity of atheromatous detritus through a minute orifice, are all of them frequent sites of commencing aneurysm. But mere atheromatous change alone, apart from actual removal of tissue, especially if the middle coat be involved, will cause sufficient enfeeblement to allow of aneurysmal expansion.

Aneurysm is a far more common affection in males than in females, mainly on account of their different [illegible] Cons; and it belongs almost exclusively to adult life. It is a [illegible] ceed, chiefly of advanced years; still it not unfrequently occurs in men and in women, between the ages of 30 and 40, and even those who have led debauched or hard lives, and have suffered [illegible] hose conditions which produce endoarteritis.

Morbid anatomy.—Many needless refinements have been made in respect of the classification of aneurysms. We shall not waste time upon this subject, but will describe them with reference (1) to their form and size; (2) to the constitution of their walls; and (3) to the nature of their contents.

1. Aneurysms in some cases are mere globose or fusiform dilatations of some limited length of artery in its whole circumference. Much more frequently they are thimble-shaped or flask-like bulgings, which involve the vessel in a portion only of its periphery. In the latter case the orifices by which they communicate with the artery vary greatly in size relatively to the aneurysmal tumours; are round or oval, with the long diameter corresponding to the axis of the vessel; and present more or less tumid margins, which in large aneurysms, involving nearly the whole width of an artery, are distinctly developed above and below only. In other cases aneurysms present great irregularity of form. This may be due to the fact, either that several aneurysmal bulgings have taken place within a short distance of one another, and have coalesced during their progressive enlargement; or that the walls of the primary aneurysmal sac have yielded unequally; or that they have ruptured or been destroyed at certain points, and the blood has escaped into fresh cavities by laceration, which form diverticula from the original aneurysm, and remain henceforth portions of it. The configuration of aneurysms is also greatly determined by the nature, arrangement, and resisting power of the structures which surround them and oppose their extension. The size which aneurysms attain depends in some degree on that of the arteries from which they spring. Aneurysms of the cerebral arteries are rarely larger than a walnut, while those of the aorta may vary from the size of a pea to that of a cocoa-nut or a child's head.

2. Occasionally the walls of an aneurysm comprise all the arterial tunics in a fairly healthy condition; as may be seen in fusiform or globose aneurysms due to general dilatation of a certain length of artery. In most cases, however, the condition of things is different:—The lining membrane of the artery may be traced, often somewhat thickened and pulpy, over the lips of the aneurysmal orifice, and thence with more or less distinctness over the whole inner surface of the aneurysm. The external coat also of the artery may be traced from without over the whole extent of the aneurysmal tumour. And as regards the middle coat, while in small aneurysms this may often be recognised as a more or less attenuated layer throughout their whole periphery, in larger ones [illegible]ally incomplete, either stopping short around the orifice, or e[illegible] for a short distance into the walls, with traces of it still to b[illegible]cted here and there throughout the rest of the circumference. When an aneurysm commences with perforation of the internal [illegible]ane of the artery, an adventitious lining forms before long, and becomes continuous with that of the artery. And indeed it is obvious that in all large aneurysms the laminæ, which correspond to the inner and outer arterial tunics and are continuous with them, are mainly, if not entirely, of new formation. Further, these two coats become, in the course of time, identical in structure, and blend, enclosing within them any remnants there may still be of the middle coat. Not unfrequently also they become the seat of fatty or calcareous change. As an aneurysm extends, surrounding organs and tissues get involved in it, and take a share in the formation of its walls, the proper coats at the same time disappearing to a greater or less extent.

ANEURYSMS

3. An aneurysmal cavity sometimes remains perfectly free from clot; sometimes, on the other hand, becomes in a greater or lesser degree obliterated by its slow deposition. The local conditions which favour coagulation are roughness of surface and comparative stagnation of blood. Both are usually present in perfection in aneurysms which originate in circumscribed bulging of an artery, and in which the orifice of communication is comparatively small. In these the process begins with the deposition of a thin adherent film upon the surface of the lining membrane. To this other films are added in slow succession; and hence the resulting mass of coagulum gradually assumes a laminated or stratified character. This process may, in fortunate cases, go on until the cavity is obliterated, the last-formed laminæ forming a kind of irregular bar or septum across its mouth. But more frequently the aneurysm is obliterated in part only, the coagulum being often limited to some diverticulum. When the lining membrane is fairly uniform and smooth, and the orifice large in relation to the cavity, there is often no attempt whatever at coagulation. And fusiform aneurysms, or aneurysms due to general dilatation, always remain free, or at all events never present more than such patches of clot as may

be met with in an undilated aorta, of which the surface is studded with patches of atheroma or calcareous plates.

The origin of aneurysms in blood-pressure, which the arterial walls are incompetent to resist effectually, has already been considered. Their progressive enlargement is dependent on the continued operation of the same cause. In accordance with a well-known hydrostatic law, the force which the blood exerts on a given aneurysmal area is exactly equal to that which it exerts on an equal area of the artery in its neighbourhood; or in other words, the total pressure on the inner surface of an aneurysm is in exact proportion to the superficial extent of that surface, and has no relation whatever either to the size of the orifice or to the form of the aneurysm. Consequently the larger an aneurysm grows, the less capable its walls become of successfully opposing the blood-pressure within, unless they undergo some kind of compensative increase of strength. This, however, does not necessarily or even commonly occur.

The *effects of aneurysms* on the organs in their vicinity are in the main those of pressure, and necessarily vary therefore in importance and kind according to the situation in which the aneurysm is developed. When it occurs among easily-displaceable organs it may attain considerable size without causing any special mischief or uneasiness. In all cases, however, surrounding parts sooner or later get pressed upon; if they are rigid they are gradually destroyed; if yielding they first yield, and only at a comparatively late period are involved in the aneurysmal parietes, and undergo the same fate as that to which the unyielding tissues more readily succumb. Thus bones and cartilages get gradually eroded; and their eroded surfaces, first exposed in the walls of the aneurysm, presently stand out from them into the interior of the cavity. Muscular and other soft tissues are first displaced, then flattened and compressed or stretched, and finally incorporated in the advancing wall and lost. Nerves and veins are similarly affected—pressure on the former causing pain, spasm, or other functional disturbance, and then paralysis or anæsthesia; pressure on veins producing impediment to circulation, with subsequent congestion and dropsy. Similar effects of pressure may be exerted on the trachea, œsophagus, and intestines, and even on the brain, lungs, liver, and other solid organs, and in each case with the production of special symptoms, which we need not stop to discuss.

The *results of aneurysms*, unless a cure be effected by surgical procedure, are almost without exception unfavourable. In a small proportion of cases a cure takes place by the spontaneous filling of the cavity with laminated clot; but generally the tumour continues to enlarge, and after a time causes death, by implicating some important organ, or by perforation and consequent profuse discharge of blood. The latter event may take place into one of the serous cavities, in which case the actual opening is usually caused by laceration; or at the cutaneous

surface or into one of the mucous canals, when perforation is due either to ulceration or to the separation of an eschar. Rupture or perforation may also take place into the cerebral or spinal cavities, the veins, and even the heart itself.

Symptoms and progress.—The symptoms by which an aneurysm may be recognised are: first, those which are due to it as a simple tumour; and second, those which depend on its relations with other parts. An aneurysm is usually a pulsating tumour. If it be empty of clot its pulsation is expansile like that of the arteries, and if it can be grasped the fingers which enclose it will be sensibly separated at each expansion. If it be full of clot no such expansion occurs; and should pulsation be then felt it is merely such as may be presented by any other solid tumour lying upon an artery: the aneurysm simply follows the movements of the subjacent vessel. It is important to know that the mere imparted pulsation of a rounded tumour may easily be mistaken for expansile pulsation unless the tumour be grasped at its widest part; for if it be grasped in some narrower and more superficial zone, the alternate rise and retreat of the skin-covered wedge-like body between the fingers produce exactly the same periodical and measured separation of them which is so characteristic of true pulsation. The comparative hardness, however, of such a mass, and the probable fact that it may admit of removal from the influence of the subjacent artery, will generally correct any erroneous impression. The pulsation of an aneurysm is sometimes vibratile, especially if it be situated in the neighbourhood of the heart and associated with regurgitant aortic valve disease. It may be vibratile, however, owing to peculiarities of form and the condition of its walls and orifice.

Aneurysms are often attended with a murmur. This generally corresponds to the cardiac systole, and therefore to the tidal wave of the pulse, and is of a blowing character. It is probably created as a rule in the artery, and due either to contraction of its tube at the point of origin of the aneurysm, or to some irregularity at that part; but it may be more or less modified, or in some cases developed, by resonance in the aneurysmal cavity. Murmurs may equally be produced by the pressure of tumours or even of the stethoscope upon healthy arteries. Aneurysms of the aortic arch, like other aneurysms, are sometimes attended with a murmur synchronous with the heart's systole, and like them may be free from murmur. But here a double murmur is not unfrequent, especially if there be associated regurgitant aortic valve disease. In these aneurysms, again, it is not uncommon to hear the two cardiac sounds, or two sounds resembling them, even more distinctly than over the heart itself. They have been supposed to originate within the aneurysm, but are doubtless the normal cardiac sounds carried by the blood-stream, and perhaps increased by resonance.

The pulse is often distinctly affected in aneurysm. But its affection

is not so much due to the aneurysm itself (though this doubtless has some influence) as to the narrowing of the artery, from pressure or disease, which is so often associated with aneurysm. It is most obvious when the aneurysm involves either the innominate artery, the subclavian, the descending aorta, or one of the iliacs. In such cases the pulse in the implicated limb, as compared with that in the healthy limbs, is diminished in volume and strength, and appears to be retarded. The systolic rise is slow in attaining its maximum, and the diastolic fall presents a corresponding character.

The symptoms due to the direct influence of aneurysms on surrounding organs vary in different cases; but their general character may be gathered from the remarks which have already been made.

Treatment.—The treatment of internal aneurysms is far from satisfactory in its results. The chief object at which to aim is the gradual coagulation of blood within the cavity, and its consequent obliteration. This event occasionally takes place spontaneously in bed-ridden patients or those who are prostrated by lingering diseases—under conditions, therefore, in which the action of the heart and the circulation are unusually feeble. These facts furnish a clue to the general treatment which should be adopted. The patient should be kept at as perfect rest as it is possible to enforce. He should be exposed to no causes of mental excitement, and strictly debarred from all forms of muscular exertion, including that of straining at stool; if possible, therefore, he should be confined to his bed. His diet should be light and nutritious, and not more abundant than is necessary to maintain him in a condition of fair, but not robust, health. It is important, too, that the bowels should be kept moderately free, either by enemata or by mild laxatives, and at all events not permitted to get constipated; and that all bodily ailments which arise to complicate the aneurysm should if possible be obviated or cured. Various drugs have been recommended, with the object either of quieting the circulation or of promoting coagulation. Among those which have been employed with reputed success are acetate of lead, iodide of potassium, ergot, and digitalis. It may well be doubted, however, whether either of these can have any real influence for good, and whether indeed digitalis is not likely to be injurious. Reduction of the volume of the blood, and of strength, by repeated copious venesections, was formerly largely advocated; and it is not improbable that, at any rate in some cases, occasional bleedings may be really beneficial. To relieve pain or uneasiness opium is invaluable, and as local applications, with the same object, ice, belladonna and other sedatives.

A. *Thoracic Aneurysms.*

Morbid anatomy and symptoms.—These occur principally in the different parts of the aortic arch, the descending thoracic aorta, and the roots of the large arteries arising from the arch. They spring most

frequently from the ascending arch, and more commonly from the convexity than from the concavity of the arch. They usually form pulsating tumours which may be recognised as such if they abut on the surface of the chest, especially if also they be large, but which frequently escape recognition in consequence of being small or deep-seated. But whether they be positively recognised or not; they generally sooner or later induce characteristic phenomena by compressing the surrounding organs, and interfering with the due performance of their functions; and end fatally in one of several fully recognised modes. It is obvious that the situation of the tumour and the facility with which it may be recognised, the parts which are specially liable to compression, and the nature of the event, must be largely determined by the part of the aorta whence the aneurysm springs.

Aneurysms of that part of the aorta which is embraced by the pericardium are almost invariably of small size; and therefore liable to be confounded with simple aortic valvular disease, or degenerative arterial changes—with both of which they are commonly associated—or else altogether to escape recognition. They occasionally open into the pulmonary artery, right ventricle or auricle, or superior vena cava; sometimes lead to the production of loculated aneurysmal cavities, extending into the substance of the cardiac walls or along the auriculo-ventricular grooves; and are very apt to rupture at an early period into the pericardial cavity.

Aneurysms of the rest of the ascending arch often attain a very large size. In their growth they encroach, as a rule, on the upper part of the right side of the thorax, displacing the lung outwards, and coming in contact by their anterior surface with the anterior thoracic parietes. Sometimes they involve both sides of the chest. They not unfrequently also displace the heart downwards and to the left. According to the amount of displacement of the lung or lungs will be the extent of the dulness on percussion to which they give rise, and that of their visible pulsation. This may be heaving, vibratile, or purring, and if visible to the eye will probably be seen to correspond distinctly with that of the heart. As the tumour enlarges it causes bulging of the chest-wall over it; and soon (eroding the ribs and their cartilages, the sternum, and perhaps the clavicle, and at the same time involving the muscular tissue) forms a more or less hemispherical pulsating mass. In the interior of the chest it presses upon the right lung, which often becomes adherent to it and expanded in some degree over it; and it is apt to compress either the vena cava descendens or the left innominate vein, or both—impeding the passage of blood through them, or rendering them completely impermeable; and it may even involve the right pneumogastric nerve or the sympathetic trunk. Aneurysms in this situation are liable to open externally, into the pericardium or right pleura, or into the lung itself and thence into one of the bronchial tubes, or even into the right bronchus.

An aneurysm of the transverse arch, if it spring from its front or convexity, expands chiefly upwards and to the left, so that it presses upon and erodes the manubrium of the sternum and the adjoining portions of the left upper ribs and cartilages, and clavicle, and forms a tumour which occupies the situation here specified, and tends to rise from behind the sternum into the root of the neck. If it spring from the concavity or posterior aspect of the arch, it is often quite latent. If it grow mainly upwards and in front, forming a manifest pulsating tumour, it may, like aneurysm of the ascending arch, attain a large size and eventually burst externally; but much more frequently, owing to the confined limits of this portion of the chest and the many important organs which are contained therein, it causes death at a comparatively early period from the effects of pressure on one or other of those organs. Aneurysms of the transverse arch are especially liable to compress the trachea or left bronchus, and may also involve the œsophagus; and often prove fatal by opening into one or other of these tubes. They may also compress or destroy the left recurrent laryngeal nerve, or the left sympathetic or pneumogastric trunk; or obstruct the left innominate vein. Further, they may rupture into the pericardium, left pleura or lung.

Aneurysms of the descending arch or of the rest of the thoracic aorta are rarely to be detected until they have acquired considerable magnitude. They become superficial by destruction of ribs and vertebræ in the dorsal region to the left of the spine, and there in some cases form pulsating tumours of enormous size. But before they cause manifest tumour they may sometimes be recognised by the presence of dulness, pulsation, and murmur, and the absence of respiration, over a limited area. An important hint as to their presence is often furnished by the occurrence of more or less constant gnawing, aching, or burning pain in the situation of certain of the vertebræ, and of shooting or aching pains or uneasy sensations in the course of some of the nerves of the brachial plexus or of some of the intercostal nerves, more particularly on the left side. Aneurysms developed in these portions of the aorta not only tend to cause destruction of the bodies of the vertebræ and posterior parts of the corresponding left ribs, and to involve the dorsal spinal nerves and the sympathetic trunk of the same side, but are especially apt to compress the œsophagus and ultimately to open into it, or to rupture into the left pleura. They may indeed rupture into the right pleura. Those arising in the upper part of the chest may also compress the trachea, left bronchus or lung, and eventually open into one or other of them.

It may be convenient to pass in review the various pressure-symptoms to which aneurysms of the thoracic aorta give rise, and of which several are often present when as yet no tumour can be discovered by auscultation, percussion, palpitation, or inspection. They are as follows:—

1. *Impediment to the arterial circulation.*—This may depend either directly on the aneurysm or on the presence of atheromatous or other thickening of the vessels springing from the arch. Not unfrequently the artery of one arm alone suffers, and the radial pulse of that arm becomes comparatively feeble, or it may be entirely annulled; sometimes both carotid and subclavian of one side are thus affected; and occasionally all the arteries springing from the arch are implicated, so that all visible pulsation in them and their branches ceases. When, however, the impediment to the circulation is thus general, it has usually come on gradually, and there have been previous stages in which one or two arteries only have been involved. In consequence of impediment to the carotid circulation, we not uncommonly find patients with aneurysm of the arch liable to momentary attacks of vertigo, or loss of consciousness, sometimes attended with epileptiform convulsions.

2. *Impediment to the venous circulation.*—When the vena cava or both innominate veins are obstructed, the veins at the root of the neck form spongy masses immediately above the clavicles, and those of the head, neck, arms and upper part of the chest undergo great distension. The cutaneous surface gets congested, especially that of the face, the eyeballs injected and prominent, and before long the head, neck, and upper extremities swollen with œdema. The patient suffers also from drowsiness, coma, and other cerebral symptoms, and extreme dyspnœa. When one innominate vein only is obstructed, the venous distension and œdema are limited to one arm and one side of the head, neck, and chest. In this case, if the patient's life be prolonged, it is not unusual for remarkable clubbing of the fingers of the affected limb to supervene.

3. *Pressure on nerves.*—Pressure on the left recurrent laryngeal nerve is soon attended with paralysis of the intrinsic muscles of the larynx which it supplies. The left vocal cord becomes motionless midway between the position of closure and that which it should assume during ordinary calm respiration, and the voice loses its musical character and becomes hoarse or whispering. Pressure on the right recurrent, which may be produced by innominate or subclavian aneurysm, will have a corresponding effect on the right vocal cord. It has often been observed that in intrathoracic aneurysm one of the pupils (as compared with its fellow) is either abnormally dilated or abnormally contracted. Abnormal dilatation has been attributed to pressure upon the sympathetic trunk in the upper part of the chest, causing irritation; abnormal contraction to pressure on the same trunk, but sufficient to destroy it or annul its functions. The pneumogastric nerve is at least as liable as the sympathetic to suffer, and to its compression congestion and gangrene of the lungs have been attributed. The effects of pressure on the intercostal nerves and brachial plexus have already been considered. It may be added that pain is apt to shoot up the corresponding side of the neck. It is obvious that the phenomena of

nervous interference must be looked for chiefly in aneurysms situated to the left of the mesial line; but they occur also in aneurysms of the ascending arch, and of course in those of the larger branches.

4. *Pressure on trachea and bronchial tubes.*—The constantly increasing pressure of an aneurysm on the trachea, if exerted laterally, displaces it to a greater or less extent; but under any circumstances the pressure sooner or later drives that portion of the surface against which it is exerted inwards, first flattening it, and then causing it to bulge so as to reduce the tracheal channel at this part to a mere semilunar chink. This process is attended with the gradual involvement of the tracheal walls in those of the aneurysm, and their infiltration with inflammatory products, followed by their gradual disintegration and final perforation. While it is going on the patient suffers from more or less stridor of the breath sounds; which becomes especially audible when, from excitement, exertion, or the act of coughing, the respiratory acts are hurried or deepened, and is attended with more or less dyspnœa. Gradually these symptoms increase, and cough is superadded. The cough is at first occasional and dry, but soon gets paroxysmal, and each paroxysm is relieved by the discharge of a small quantity of mucus. The stridulous respiration, and the stridulous cough in prolonged paroxysms (threatening and sometimes ending in suffocation) are peculiarly suggestive of the presence of an aneurysm or other tumour in the thorax. The suffocative cough is due to the occasional closure by mucus of the narrow tracheal chink and the mechanical difficulty which there then is in effecting its dislodgment. Hoarseness or loss of the musical quality of the voice only exists when, associated with the tracheal pressure, there is involvement of the recurrent laryngeal nerve, or some distinct affection of the vocal cords or their muscles. Accumulation of mucus in the bronchial tubes, lobular pneumonia, congestion of lungs, and pneumonia, are all of them common sequelæ of tracheal obstruction. When only one of the bronchi is obstructed, feebleness of respiratory murmur and imperfect expansion may be observed on the affected side of the chest, on which presently supervene rhonchus, crepitation, and other signs of one or other of the lung affections just enumerated.

5. *Pressure on the œsophagus* causes the ordinary phenomena of œsophageal stricture.

Thoracic aneurysms are often exceedingly difficult of diagnosis, partly because the symptoms to which they give rise are obscure, partly because many affections simulate them in their general and local indications. Among such affections may be included: persistent violent palpitation of the heart, such as is met with in Graves's disease; and hypertrophy and dilatation of the heart, associated with regurgitant aortic valve disease. In both of these conditions there is often violent pulsation, attended with purring tremor of the arch of the aorta and large vessels; and in both, marked pulsation; and the cardiac sounds

may be propagated over a considerable portion of the right infraclavicular and mammary regions. There may even be, in the latter case especially, some retraction of the anterior edge of the right lung and consequent extension of aortic dulness to the right. Other conditions liable to be mistaken for aneurysm (especially if they be associated with palpitation or heart disease) are mediastinal tumours, consolidated portions of lung, and abscesses or growths involving the thoracic parietes.

In the foregoing account we have referred mainly to typical aortic aneurysms. But aneurysms of the intrathoracic portions of the large arteries which spring from the arch present much the same local and general symptoms as do aneurysms arising from the aorta itself in their immediate neighbourhood. They are to be distinguished mainly by their position and the special influence which they exert on the circulation through the arteries with which they are connected. We may add, that so-called 'dissecting aneurysms' are not unfrequent in the aortic arch. They are produced by the sudden laceration of the diseased or merely thinned internal coat of the artery, and the effusion of blood through the rent into the interval between the external and internal coats, and generally into the substance of the middle coat. The extent to which the dissection may take place, and the event, both vary. In some cases the dissection is limited to a small well-defined area; in other cases it circumscribes the vessel, and occupies an inch or two of its length; and in other cases, again, it involves the whole length of the aorta. As regards result, dissecting aneurysms occasionally undergo spontaneous cure by the coagulation of the extravasated blood; sometimes they prove fatal by causing complete obstruction of the aorta, in the thorax or abdomen; but more frequently they terminate in laceration of the external membrane, and the effusion of blood into some cavity, such as the pericardium, or into the connective tissue of the mediastinum or some other part.

Treatment.—In addition to the general plan of treatment which has been laid down for aneurysms, it is sometimes possible, from the fact that aneurysms of the ascending and transverse arch and of the vessels which spring from them come speedily into relation with the anterior walls of the chest, to employ mechanical or other means to cause coagulation within them. The methods which have been had recourse to, but unfortunately with very imperfect success, are galvano-puncture, the injection of perchloride of iron or other styptics, and the insertion of coils of thin iron wire or of needles. Ligature of the subclavian and carotid arteries, especially those of the right side in aneurysm of the ascending arch, has occasionally proved beneficial; it is less useful, however, here than in the treatment of aneurysms of the roots of these vessels.

B. *Abdominal Aneurysms.*

Morbid anatomy and symptoms.—Aneurysms may be developed in connection with any part of the abdominal aorta or of its branches within the abdomen. Those which chiefly concern the physician are connected with the aorta, cœliac axis, superior and inferior mesenterics, renals and common iliacs. The sources of abdominal aneurysms must be determined by their anatomical relations. They may generally, while still of medium size, be recognised as distinct pulsatile tumours, attended with more or less thrill and often with a murmur. It is easy, however, especially in thin persons, to mistake the pulsation of the abdominal aorta for that of an aneurysm, and especially so to mistake a carcinomatous or other tumour situated upon the aorta. Indeed, it is often impossible to distinguish accurately between an aneurysm and such a solid mass, unless by grasping the tumour we can distinctly satisfy ourselves that it does not expand, or by displacing it from its contiguity with the aorta we annul its pulsations. Abdominal aneurysms generally tend to attain a large size, to cause erosion of the vertebræ or other bones with which they come in contact, and to press upon the stomach, duodenum, or other viscera, veins, or nerves. They then cause: pain in the back, which is sometimes very agonising, and often shoots along the branches of the lumbar nerves; sickness from pressure on the stomach or obstruction of the duodenum; or compression and even obliteration of the inferior cava, or one of the common iliac or renal veins, with dilatation of the veins of the lower extremities and anasarca; or similar conditions in one lower limb only, or in a kidney. Abdominal aneurysms occasionally burst into the peritoneal cavity, or into one of the hollow viscera, or even into the spinal canal. More frequently they rupture primarily into the retro-peritoneal tissue; whence blood may be effused round the duodenum, or œsophageal opening of the stomach, or into the substance of the mesentery, mesocolon, or great omentum, and may thus before the supervention of death cause complete obstruction of the cardiac orifice, duodenum, or some other part of the bowel, and sometimes the most intense and long-continued agony of pain.

Treatment.—The most important of the special modes of treatment of abdominal aneurysms are: first, that of putting a ligature round the aorta; and, second, that of regulated pressure upon the aorta. The latter method may be carried out by the temporary application (say for eight or ten hours), under the influence of chloroform, of a specially adapted tourniquet to the aorta, if possible on the proximal side of the aneurysm. Pressure may be applied, with almost equal efficacy, on the distal side. It must not be forgotten, however, that the application of sufficiently forcible pressure completely to obstruct the aorta is attended with great risk of serious injury to the abdominal viscera; and hence it will generally be best to delay its employment until the effects of perfect rest have been fully tested.

Section III.—DISEASES OF THE VEINS.

I. PHLEBITIS.

Causation and morbid anatomy.—Inflammation of a vein is generally due: either to the formation of a clot within it, in which case the process commences at the inner surface and travels outwards; or to the involvement of the vein in inflammatory processes which are going on round about it, in which case its walls are invaded from without inwards. Phlebitis, indeed, is almost always secondary. Exceptions to this rule are furnished by inflammation of the uterine veins after parturition, and by the comparatively rare thickening of the inner coat of veins which co[illegible] the much more frequent thickening of the inner [illegible] of arteries issuing in atheromatous and calcareous degenera[illegible]. The presence of clots may be regarded as an essential accompaniment of all forms of phlebitis, with the exception of that chronic form last adverted to.

Inflammation of veins is characterised by thickening of their walls, connected with more or less active proliferation of the protoplasmic elements of their several laminæ. The latter process is generally especially active in the outer coat, which not unfrequently acquires considerable thickness and blends with the surrounding similarly affected connective tissue; and scattered abscesses are apt to appear here and there in its course. The inner coat tends to become rough, and even to give rise to granulations. The contained clot, whether it be formed primarily or secondarily, soon fills the channel of the vein and adheres more or less firmly to its inner surface. At the same time it tends to lengthen both above and below—above to the junction of the vein with the next branch or its communication with a trunk vein, below into the tributary branches. The further changes which such clots undergo will be considered under the head of thrombosis.

The *symptoms* of venous inflammation are, if the vein be within reach of direct observation, pain and tenderness in its course with more or less distinct cylindrical thickening and hardening, and sometimes superficial redness. Abscesses in the course of the vessel, communicating or not with its interior, are not unfrequent. There is necessarily more or less febrile disturbance. The remote effects of phlebitis are on the whole much more important than the local effects. They embrace on the one hand, those which are due to venous obstruction—dilatation of the distal veins, congestion, and anasarca; on the other, those dependent on the discharge of fragments of thrombus, or of inflammatory or other hurtful matters into the circulating blood. These will all be best considered hereafter.

II. VARIX. (*Dilatation of the Veins.*)

Causation.—Dilatation of veins is much more common than that of arteries, but its causes are a good deal more obscure. It occurs no doubt generally, in obstructive disease of the right side of the heart; and (when a vein is obstructed) throughout the venous system which is tributary to it, as well as in those collateral veins which take on, or divide between them, the duties of the defaulting vessel. But in a large number of cases veins get dilated and varicose independently of all obstruction, independently of overwork, and independently also of obvious degeneration or weakening of their walls.

Morbid anatomy.—When veins dilate they become at the same time elongated, and consequently more or less tortuous. The dilatation usually commences, and is always most marked, immediately above the valves; and the affected veins assume, therefore, an irregularly moniliform aspect. The walls, for the most part, thicken considerably, although presenting occasional attenuations, especially over the convexities of the dilated portions. The thickening is principally due to hypertrophy of the middle coat, the attenuation to its atrophy or disappearance. With the progress of dilatation the valves become inefficient, and often shrivel up; calcareous plates not unfrequently form in the middle coat; the connective tissue around gets thickened and indurated, and blended with the outer coat of the vein; phlebolites are often developed in the pouch-like protrusions; and the last occasionally become perforated either by extension of ulceration from without or by laceration.

Dilatation may occur either in veins of medium or large size, or in those which are ordinarily mere capillary tubes. The former occurrence is exemplified by the ordinary varicose veins of the lower extremities, and by varicocele, the latter by the tuft-like groups so common in the lower limbs of pregnant women. Dilatation and varicosity of veins rarely require treatment at the hands of the physician. For him they serve mainly as important aids to diagnosis. Varicose veins in the lower extremities, varicocele, and hemorrhoids, are surgical disorders. Dilated or varicose veins of internal organs no doubt occur, and aid in the production of functional disturbance; they may even rupture and cause death by hemorrhage. We have witnessed this accident in a case of varicose veins of the œsophagus. But their presence can rarely if ever be recognised during life. The importance of the dilatation of certain groups of superficial veins in enabling us to judge of the seat and character of internal diseases involving the obstruction of deep-seated veins is obvious

Section IV.—ARTERIAL AND VENOUS OBSTRUCTION.

THROMBOSIS AND EMBOLISM.

Definition.—There are few morbid processes of greater interest and at the same time of greater practical importance to the physician, than those which we are now about to consider. They are the frequent causes of many obscure complaints, as well as of some of the most clearly characterised maladies; they may involve any organ of the body, and present at least as many different groups of symptoms as there are organs; and they are intimately related to some of the gravest forms of disease which come under our notice, such as pyæmia and puerperal fever. The term '*thrombosis*' has been conveniently applied to the coagulation of blood during life in the heart, arteries, or veins, and includes within its meaning nearly all those cases which were formerly regarded as phlebitic. The term '*embolism*' has been introduced to designate those cases in which an artery or vein gets plugged by the impaction in it of a clot or other solid mass conveyed to it from a distance by the blood-stream. The morbid phenomena and symptoms which thrombosis and embolism induce are referrible partly to local inflammation, but principally to arterial or venous obstruction.

A. *Thrombosis.*

Causation.—The causes of thrombosis are mainly: stagnation or sluggish movement of the blood; the contact of the circulating fluid with inflamed or otherwise diseased surfaces; and special conditions of the blood which render it apt to coagulate.

Morbid anatomy.—1. In the *heart*, after death, the blood which was contained within its cavities at the moment of death is generally found coagulated, moulded to the form of the cavities, and continuous with cylindrical clots occupying the trunk veins, and often with similar clots extending into the trunk arteries. These clots are sometimes black-currant-jelly-like, sometimes partly decolourised; and the portions prolonged into the arteries are usually more or less purely fibrinous, while those seated in the veins are usually soft and black. But not unfrequently the clots contained in the heart's cavities, and more especially those occupying the ventricles, are almost entirely fibrinous, more or less opaque and buff-coloured, close in texture, and even indistinctly laminated. These have for many reasons obviously formed during life, probably during the agony; but are the consequence of dying and not the cause of death; and on the whole (except from the fact that their deposition helps, as it were, to confirm the fatal issue) have little clinical importance. Their presence, however,

throws light on the development of the peculiar bodies next to be considered. It is not uncommon to find after death in certain cases that rounded buff-coloured masses, varying perhaps from the size of a pea to that of a walnut, are situated either in the apical portions of the ventricles, or in the appendages of the auricles. These, which are sometimes termed softening clots, usually occur in groups, are moulded to the surface on which they lie, adhere to it, and are continuous with one another by processes which underlie the carneæ columnæ; so that, with careful dissection, they may generally be removed as a continuous whole. They are sometimes smooth, sometimes ribbed, upon the surface, and often variegated in colour. On section they may present a uniform character and consistence; but are more frequently broken down in their interior into a thick reddish or yellow pus-like fluid, containing products of disintegration only—namely, fat granules, degenerating red and white corpuscles, cholesterine, and sometimes hæmatoid crystals. The bodies are clots, in fact, which have formed in the heart's cavities sufficiently long before death to have undergone the degenerative changes which clots formed elsewhere also undergo. They may be found in any of the heart's cavities; in one alone, or in two or more at the same time; but are much more common in the left ventricle than elsewhere. The conditions under which they are found are various; but they are especially frequent in cases of advanced heart or renal disease in which the patient has lain for weeks with an extremely feeble circulation, and the balance trembling between life and death. During this period the enfeebled heart probably fails to empty its cavities completely; the blood remains stagnant or nearly so in those portions of them which are most remote from the direct current; and coagulation takes place either slowly, or more probably suddenly, on one of those occasions, which are so common in these cases, when the patient falls into a state of apparent death, from which he rallies. Other clots of old formation, which may be found in the heart, are laminated clots such as occur in aneurysms. They may be present in actual aneurysmal dilatations of the ventricles, and have been discovered behind a closely constricted mitral orifice, almost entirely occluding the left auricle.

2. In the *systemic veins* the coagulation of blood during life is common enough. When the venous circulation is simply enfeebled, as in the later stages of heart disease, and towards the close of phthisis, carcinoma, and other chronic wasting affections, venous thrombosis is of frequent occurrence. It then takes place more particularly in the trunk veins of the lower extremities, and in those of the pelvis or at its brim. So again when some impediment exists to the passage of blood along a vein, the distal portion of the vessel and in a greater or less degree its tributary branches fill with clot. When veins are involved in inflammation which is taking place round them, this, as has

been pointed out, tends soon to pervade the entire thickness of the walls, and then to induce coagulation of the blood within them and their complete obstruction; and occasionally, indeed, by perforation of the vein or some other process pus or other inflammatory products find their way into the interior of the vein or into the substance of the thrombus. Thrombosis, secondary to inflammation, is common in erysipelas, diffuse cellular inflammation, carbuncle, and the like; in puerperal pelvic cellulitis; in inflammation involving the cancellous structure of bones, or the walls of the parturient uterus; and in the venous sinuses of the interior of the skull in connection with disease of the internal ear.

The different characters which venous thrombi display depend largely upon their age, and correspond with those presented by cardiac clots. When fresh either they have a uniform consistence and colour, or there may be a central black cylinder, enclosed in a more or less complete fibrinous capsule. They do not necessarily at once fill the vessels in which they are seated, even if they be in a greater or less degree adherent to them, and hence fresh blood tends to insinuate itself between them and the venous parietes, and presently to coagulate there. The clots which finally occlude vessels thus get more or less distinctly laminated. In their further progress venous thrombi undergo various changes. In some cases they blend with the venous walls, and, becoming converted into connective tissue, cause the obliteration of the vessels; in some they undergo softening in their interior, and conversion into loculated cavities full of fatty detritus and caseous remnants of white corpuscles; and occasionally they suppurate and form abscesses.

3. *Arterial thrombosis* is due in a large number of cases to simple stagnation of blood. Thus the arteries leading to a district, in which (owing to morbid processes going on in it) the blood has ceased to circulate, get filled secondarily with coagulum. And in precisely the same way, if an artery be ligatured, or obliterated at any point by the pressure of a tumour or tourniquet, the proximal portion of the vessel up to the nearest branch becomes the seat of thrombosis. Not unfrequently also, when the circulation is simply feeble, obliteration of an artery by coagulation of its contents takes place. This occurrence in the smaller branches of the pulmonary artery is a common cause of pulmonary apoplexy. It is occasionally also observed in the arteries of the extremities and even in the aorta itself. Diseases of the inner coat of arteries—atheroma, calcification, arteritis, and syphilis—are all of them liable to induce thrombosis and consequent obliteration. Among the arteries which are especially liable to suffer thus are those of the base of the brain and of the extremities. The varieties of arterial clots and the changes which take place in them are identical with those which have been described in connection with veins.

B. *Embolism.*

Causation and morbid anatomy.—The sources of emboli are mainly venous thrombi, cardiac vegetations, and disintegrating calcareous, atheromatous, or inflamed surfaces. Additional sources are softening clots in the interior of the heart, and morbid growths or other adventitious bodies.

The detached solid mass, whatever its nature, is carried along more or less rapidly by the blood-stream until it reaches a vessel which is too small to allow of its further progress. The point at which it becomes finally arrested usually corresponds to the bifurcation of a vessel or to the giving off of a comparatively large branch. Here it gets wedged, sometimes blocking up the channel completely, but more frequently forming at first a partial impediment only. In the latter case the constant pressure from behind tends to drive it farther and farther onwards, in consequence of which, or of the gradual coagulation of blood around it, the vessel becomes at length, as in the former case, completely occluded. Subsequently thrombosis takes place on both sides of the embolus; the artery and its distal branches get filled with clot which, gradually undergoing changes, blends on the one hand with the arterial parietes, and on the other with the embolus. So that although the embolus may, at first, be readily recognised as an independent body, it often becomes undistinguishable from the thrombus to which its presence has given rise.

Emboli taking their origin in the systemic venous system, or right side of the heart, necessarily become fixed in the pulmonary arteries. Those which originate in the pulmonary veins, left side of the heart, or larger systemic arteries are conveyed to the periphery of the systemic arterial circulation. And those, lastly, which are yielded by the veins of the chylo-poietic viscera find their resting-place in the branches of the vena portæ.

Owing to the infrequency of disease of the valves of the right side of the heart, embolism involving the lungs is almost invariably due to the detachment of venous clots or fragments of them. In some cases entire systems of thrombi become free, and a complete cast, some inches long, of a venous tree may be carried into the pulmonary artery and impacted in a more or less convoluted form within it. More frequently shorter lengths get successively separated and successively lodged in different branches of that vessel. It is much more common, however, for venous clots to crumble as it were gradually away; and for minute fragments to get impacted from time to time in the pulmonic arterioles.

It is rare for thrombosis to take place in the pulmonary veins; and hence embolism is seldom due to this cause. The most common source of embolism of the systemic arteries is undoubtedly the detachment of

granulations from the diseased aortic or mitral valve; but another frequent cause is the separation of atheromatous or calcareous particles, or other detritus, either from the valves or inner surface of the heart, or from the large arteries. It is obvious, therefore, that embolism of the systemic arteries must in a very large proportion of cases depend on valvular disease, and is to be regarded as one of the common risks of that affection. Emboli from the various sources just indicated are carried along the aorta and thence into some of the smaller branches of the systemic arteries—whither is in some degree a matter of accident; but there are certain parts, namely the brain, liver, spleen, and kidneys, and, it may be added, the lower extremities, which are specially prone to suffer. It is probable, however, that their arteries are not so much specially liable to obstruction, as that their obstruction produces particularly serious and obvious ill-effects. The cerebral arteries chiefly liable to obstruction are the middle cerebral branches of the internal carotids; and it is curious that the obstruction generally occurs in the middle cerebral of the left side.

C. *Consequences and Symptoms of Thrombosis and Embolism.*

It is certain that, whenever a thrombus forms or an embolus becomes fixed, inflammation of the implicated vascular walls, if it did not previously exist, speedily ensues; and that hence pain and tenderness will mark the course of the vessel if it be within reach of investigation, and more or less febrile disturbance generally be present. It is further certain that in both cases complete obstruction to the passage of blood through the affected vessel takes place very soon if not quite suddenly. It is this fact, indeed, which gives to thrombosis and embolism in common their characteristic features, and which renders it difficult, if not impossible, to make any practical distinction between them. In aid, however, of correctness of diagnosis it may be pointed out: first, that obstruction of the pulmonary and systemic veins by clots can depend on thrombosis only; second, that obstruction of arteries or of the portal veins may be due either to thrombosis or to embolism; third, that the pre-existence of systemic venous thrombosis renders it probable that any obstruction occurring in the pulmonary arteries is due to embolism; and lastly, that the presence of valvular disease on the left side of the heart, or the fact of previous rheumatism, is presumptive evidence that supervening obstructive disease of any of the smaller systemic arteries is of embolic origin.

The results of venous thrombosis are stagnation of blood in the tributary veins with dilatation, soon followed by compensatory dilatation of the anastomotic veins, and œdema. These conditions are not secondary to thrombosis only, but attend all cases in which veins from whatever cause are obstructed. The consequences of arterial thrombosis or embolism, on the other hand, are impairment of nutrition

of the region which the artery supplies, and, following on this, congestion, hemorrhage, inflammation, degeneration, or gangrene, together with special symptoms due to the organ or part whose integrity is compromised. Similar phenomena necessarily ensue upon all forms of arterial obstruction, no matter how they are produced. The special effects of thrombosis and embolism will for the most part be best discussed in connection with the other morbid conditions of the several organs in which they occur. There are two or three cases, however, which may be most conveniently considered now. They are phlegmasia alba dolens, thrombosis and embolism of the heart and pulmonary artery, obstruction of the larger arteries of the limbs, and multiple embolism of the smaller systemic arteries.

1. *Phlegmasia alba dolens.*—This term is generally applied to the painful and œdematous condition of leg which often follows upon parturition. An almost exactly similar condition may, however, occur independently of parturition, and even in males, and is not unfrequently developed in the course of phthisis and carcinoma. The arms also may be affected in like manner as the lower extremities. Phlegmasia alba dolens is due to thrombosis of the trunk veins of the limb, or of the larger veins to which these converge, which become converted into painful rigid cords. When it follows parturition it generally begins from a week to a month after that event, and almost invariably in the left lower limb. And even if the right become affected it is usually affected in company with the left but at a later period. The commencement of this disease is generally sudden and indicated by the concurrence of diffused pain throughout the affected member, and œdema. The pain varies in character and intensity, and is generally attended with soreness or tenderness, sometimes with distinct hyperæsthesia, sometimes with loss of sensation; and not unfrequently the patient is unable, either from pain or from loss of power, to move the limb or any of its parts. The œdema gradually increases until the member gets large and smooth, and of a peculiar pale waxy aspect: it does not generally pit distinctly on pressure, and often presents remarkable elasticity and tension. The superficial veins usually become dilated and unnaturally visible; and the skin often presents a mottled, retiform character, owing to the rupture, as in pregnancy, of the deeper layers of the cutis. There is not as a rule any manifest change of temperature in the affected limb; but more or less general febrile disturbance is usually present. If there be no serious complication, the patient probably recovers at the end of three or four weeks. For the most part, however, the veins primarily obstructed remain impervious; and sometimes there is more or less permanence of œdema.

Treatment.—Little can be done in the way of special treatment for phlegmasia dolens or other œdematous conditions arising from obstructed veins. It is generally desirable that the patient be kept at rest, and the affected limb elevated or in the horizontal position. If

there be distinct inflammatory mischief in the course of a large vein, a few leeches may be serviceable; and when œdema and tenderness are present, it is generally of benefit to envelop the limb in wadding or flannel, in order to keep it warm and promote perspiration. Hot fomentations and baths may also be employed. The internal treatment must be determined by the general condition of the patient; but for the most part tonics are chiefly indicated.

2. *Cardiac thrombosis.*—It is not easy to specify symptoms by which clots formed in the heart during life may be recognised. It is possible of course that, from their position, they may occasionally interfere with the due action of the valves, and so induce endocardial murmurs; but it is certain that in the great majority of cases they have no such effect. It may be taken for granted that their presence must in almost all cases be a source of embarrassment to the heart's action, and that they must therefore tend to aggravate the feebleness of circulation out of which they arose, and to increase the severity of the cardiac symptoms which the patient had previously suffered from. It is important, however, to know that when such clots form in the heart, the feebleness of circulation which determines their presence there very commonly also determines their formation in arteries and veins; and that hence the condition of the lungs and kidneys, connective tissue, and skin, may be of some assistance in the formation of a diagnosis. The detachment of such a clot and its entanglement in one of the valvular orifices of the heart have been assigned as a cause of sudden death.

3. *Embolism and thrombosis of the pulmonary artery.*—We do not intend here to discuss the results of that blocking up of the smaller branches of the artery which is so commonly associated with, and so often the cause of, pulmonary apoplexy, lobular pneumonia, circumscribed abscesses, patches of gangrene, and the like. Our object is to consider those embolic or thrombotic obstructions of the arterial trunk, or of its chief divisions, which are occasionally the cause of sudden death.

It is now well established that the chief danger of thrombosis of the larger systemic veins lies in the separation of the whole or a large portion of the clot and its impaction in the trunk of the pulmonary artery. This accident is especially apt to occur in cases of phlegmasia dolens, and where, after parturition, the uterine veins have become plugged. The patient, probably in the midst of apparently fair health, is suddenly seized with severe pain in the region of the heart, attended with intense distress and gasping for breath, pallor or lividity of face, and extreme feebleness or even suppression of pulse, and dies collapsed. It has been disputed whether death is due to asphyxia or syncope. It is certain, however, that the sudden obstruction of the pulmonary artery causes 'shock,' or collapse, and that the patient sometimes dies of this shock within a few seconds; and it is further certain that the symptoms of sudden obstruction are often undistinguishable from those of

angina, or rupture of an aneurysm, or of the heart itself, into the pericardial cavity. Indeed, the symptoms of pulmonic obstruction are by no means typical; and its diagnosis must depend mainly on the association of the symptoms above described with those conditions of the venous circulation which are known to be productive of embolism.

There are two or three points, however, in relation to this subject which demand a word or two of comment:—First, sudden obstruction of the pulmonary artery by an embolus, even if attended with symptoms of great severity, does not necessarily end in immediate death. The clot may be driven onwards into a branch, the symptoms of impending death subside, and the phenomena due to the obstruction of a branch only presently ensue. Second, it is important to bear in mind that many of the recorded cases of sudden death from pulmonary embolism are cases in which the only foundations for this diagnosis were: suddenness of death, possibly from syncope; and the discovery after death of an ordinary fibrinous clot in the right ventricle, prolonged thence into the pulmonary artery and its branches—a clot originating in the spot in which it was found, and the consequence of dying, not the cause of death. Third, thrombi sometimes form in the larger branches of the pulmonary artery. Occasionally, indeed, the trunk and the greater number of its ramifications are almost entirely occluded by them. It is a fact that these may form without pain, and cause little or no distress, until by some little displacement of them, or by the sudden coagulation of the blood still circulating between them and the walls of the tubes in which they lie, they suddenly bring the pulmonary circulation, and with this life itself, to a stop.

4. *Embolism and thrombosis of the larger systemic arteries.*—It sometimes happens that, from either embolism or thrombosis, one or more of the arteries of the legs, the femorals, the iliacs, or the abdominal aorta itself, become obstructed. And the same thing may occur in respect of the arteries of the upper extremities. The immediate result is serious impediment to the circulation through the implicated limb or limbs, characterised by cessation, or at any rate diminution, of pulsation in the vessels beyond, and more or less pallor and coldness. In some cases collateral arteries gradually enlarge, and the general condition of the limb after a time becomes normal. In other cases the circulation comes generally, or in certain areæ, to a permanent standstill, the affected parts gradually lose their temperature, the surface gets pallid but mottled with purplish spots, and the tissues assume a doughy consistence, bullæ, filled with sanious fluid, soon rise upon the discoloured patches, and gangrene becomes established. Arterial embolism is generally attended with severe pain at the point of impaction, and much pain and tenderness are generally present in the course of plugged arteries. It usually happens, moreover, that pain and tenderness are, for a time at least, present in a greater or less degree in the parts which are in process of sphacelation.

Treatment.—For the local treatment of gangrene little can be done beyond keeping the parts warm. For this purpose they may be greased and covered with cotton-wool or wadding. For general treatment, it is chiefly important to maintain the patient's strength by the administration of food and stimulants, aided by tonics; and to relieve pain and distress by opiates.

5. *Multiple embolism of the smaller systemic arteries.*—This is usually the consequence of *ulcerative endocarditis*, or of the gradual erosion and disintegration of parts of the endocardium—mainly the aortic and mitral valves—which have been the seat of inflammatory thickening and overgrowth. It may come on in the course of acute rheumatism as a complication, or it may supervene accidentally, so to speak, in persons suffering from chronic heart-disease.

It depends on the constant or repeated discharge into the blood-stream of minute fragments of detritus, or emboli; which distributed throughout the system obstruct the smaller arteries, and mainly those of the kidneys, spleen and liver—causing infarcts with more or less attendant inflammation, which is apt to spread from the solid organs to the serous membranes. The exact nature of the embolic changes occurring under the above circumstances in different parts of the organism has been described in the article on pyæmia, and will be further considered in connection with the diseases of particular organs.

The symptoms of this condition have a close resemblance to those of pyæmia, but on the whole are less intense, and less rapidly terminate in death. The patient generally has rigors, with elevation of temperature, up, it may be, to 105 or more, and perspirations. The febrile symptoms intermit, sometimes several times a day. The respirations increase in frequency; the pulse becomes rapid and feeble or irregular; the tongue gets coated; anorexia, thirst, nausea or sickness, and oftentimes diarrhœa ensue; the spleen generally enlarges and gets distinctly tender; occasionally more or less jaundice supervenes; the urine frequently contains albumen; the patient becomes restless, delirious, or drowsy, and sometimes at length comatose. In addition, inflammations of the joints or serous membranes, and roseolous, petechial or pustular rashes are occasionally developed. The collective symptoms nevertheless are very often vague and misleading, and are liable to be mistaken, not only for those of pyæmia, but for those of enteric fever or ague. Most frequently, no doubt, the disease chiefly simulates chronic pyæmia; and if its source in cardiac disease were overlooked, would be taken for pyæmia. Its resemblance to ague is occasionally very remarkable. In a case recently under our care, which lasted altogether several months, the main symptoms were typical ague-like paroxysms, coming on almost without exception twice in the twenty-four hours, and separated by intermissions of complete apyrexia, associated with gradual failure of strength, and drowsiness finally passing into coma.

Slight attacks of this affection doubtless occur not unfrequently, are recovered from, and overlooked; but where it is present in an aggravated form it is probably always fatal sooner or later, and generally perhaps in the course of two or three weeks.

For *treatment* little can be done, beyond relieving symptoms, reducing fever, and maintaining the patient's strength.

Section V.—DISEASES OF THE DUCTLESS GLANDS, BLOOD, &c.

I. DISEASES OF THE THYROID BODY.

To diseases of this organ the term 'goitre' or 'bronchocele' is commonly applied. It is more convenient, however, to restrict these names to a certain group of hypertrophic affections than to include under them every variety of lesion to which the thyroid body is liable.

The chief affections which would on these grounds be excluded are inflammation and carcinoma.

Idiopathic inflammation of the thyroid body is certainly of unfrequent occurrence; it may, however, follow secondarily upon goitre, or result from operation, or injury inflicted upon the gland. It is probable also that some of the overgrowth of the hypertrophic organ may be due to chronic inflammation.

Carcinoma of the thyroid body is extremely rare. Undoubted examples of this affection have however been recorded, in some of which the morbid growths were primary, in others due to extension from neighbouring organs, in others secondary in the usual sense of that term.

It is needless to discuss particularly the symptoms to which these conditions give rise, or the special treatment they may require.

A. *Goitre.* (*Bronchocele.*)

Causation.—The circumstances under which goitre arises are various and not very clearly understood. There is no doubt that it is far more common in females than in males; and indeed, as regards women, it has long been known that there is not unfrequently a tendency to some temporary enlargement of the thyroid body both during pregnancy and at the catamenial period. It is occasionally observed in the fœtus, and is then commonly associated with some peculiarity in the form and situation of the gland.

Goitre appears to originate with special frequency between the ages of eight and puberty; but rarely, if ever, after forty. It occurs in a sporadic form in probably all parts of the globe; that is, isolated cases,

for which no cause can be assigned, are nearly everywhere occasionally met with. It is remarkable, on the other hand, that goitre is endemic in many limited areæ scattered nearly all over the world. Such places are, in England, met with in Derbyshire, Hampshire, Nottinghamshire, Sussex, and Yorkshire. Goitrous districts are as a rule of peculiar geological formation; they are mostly valleys; and usually their soil, or that of the adjoining mountain ranges, is formed largely of lime or magnesian limestone; and the water of the wells or watercourses which traverse them is largely impregnated with carbonate or sulphate of lime, with which in a considerable number of cases magnesia is associated. Various reasons have been assigned for the prevalence of goitre in these localities. All evidence, however, seems to point to the drinking water as the efficient agent in its production; and it is generally held that the poisonous ingredient is either sulphate or carbonate of lime, or both, in combination probably with magnesia. The main objection to this view—and it is a serious one—is, that hard waters containing such ingredients in excess occur and are used in non-goitrous localities by persons who never become goitrous. And hence it is probable, as is suggested by Virchow, that these salts do not act directly, but that associated with them there is some other principle of a malarious character to which the goitrous tendency is essentially due. It may be added: that endemic goitre is endemic in the strict sense of the term; that it belongs, as it were, to the locality; that new-comers are liable to suffer equally with those who have been born and bred in it (allowance being of course made for the relative length of their exposure to the goitrous influence); and that, although the children of goitrous parents become in large proportion goitrous in such localities, the tendency is not hereditary, and ceases in them when they are removed from the influence of the poisonous principle. In goitrous districts the disease is not limited to man; but dogs, mules, and horses are all liable to suffer.

Morbid anatomy.—Goitre consists in a kind of hypertrophy of the normal constituents of the gland—namely the blood-vessels, connective tissue, and groups of intercommunicating vesicles forming the ultimate lobules which the connective tissue circumscribes. In some cases all of these become increased in equal proportion, and the goitre then differs little if at all, except in bulk, from the healthy organ. More frequently, however, one of these constituents undergoes disproportionate development, and hence the texture of the tumour becomes characteristically modified. Thus, sometimes the connective tissue alone undergoes hypertrophy, and the tumour gets hard and dense; sometimes the vascular tissue especially becomes preternaturally developed—the veins and arteries, or more frequently the veins alone, attaining comparatively enormous dimensions; sometimes the vesicles are the chief seat of change—they become dilated and filled with an albuminous fluid, or a solid albuminoid or gelatinous substance. Such

cysts, partly by simple dilatation, partly by coalescence, may attain the size of a pigeon's or hen's egg, or even a larger bulk. In a case recorded by Mr. Spencer Watson, a cyst of this kind yielded on puncture a pint and a half of blood-coloured fluid. It must be added: that cysts of considerable size may be developed in glands which are in all other respects healthy; that a goitrous tumour may become, in whole or in part, the seat of inflammation, and that consequently blood may be poured out into the cysts which it contains, or suppuration and ulceration may take place in it; and that degenerative changes may ensue after a time, the cells within the cysts undergoing fatty disintegration, and the contents of the cysts consequently acquiring a milky character, or the fibroid stroma becoming the seat of earthy deposition —a change which is often attended with diminution in size and induration of the tumour.

A bronchocele varies in consistence according to the nature of the processes which have been going on in it; so that in some cases it is hard and resisting, in others it is soft and yielding or elastic, and in others, again, presents in certain situations distinct fluctuation; and when the enlargement of its arteries constitutes a special feature of the tumour, there may be pulsation resembling that of an aneurysm.

A goitre sometimes retains accurately the form of the healthy gland; but more frequently it becomes unsymmetrical in the progress of its growth (the right lobe being especially liable to disproportionate development), and then by growing in certain directions, or throwing out lobules, may press inconveniently or dangerously on important organs in its vicinity. Occasionally small supplemental thyroid bodies may be detected in the neighbourhood of organs thus hypertrophied; and it is especially by the development of such masses at the posterior part of the lateral lobes that compression of the œsophagus is sometimes effected.

The size of a goitrous tumour varies from that which produces a mere fulness (by some persons regarded as ornamental) in the lower part of the front of the neck, to a mass (usually more or less irregular in form) as large as a cocoa-nut, or in rare cases of such enormous dimensions that it hangs pendulous from the neck, concealing the chest, and abdomen, or even extending to the middle of the thigh.

Symptoms and progress.—A goitre may generally be readily recognised by its relations with the various structures occupying the lower and anterior part of the neck, and especially by its situation in front of the trachea, and by its following the movements of that tube. Its development is seldom attended with pain, and not usually with uneasiness; nevertheless various injurious consequences are apt to ensue. In the first place the tumour may prove seriously inconvenient by its mere bulk and weight. In the second place, it may exert pressure on the large veins in its neighbourhood, or on the trunk of the sympathetic nerve, or on the pneumogastric or recurrent laryngeal, or on the

rachial plexus. And in the third place it may displace and compress the œsophagus or trachea. Pressure on the œsophagus is induced mainly by enlargement of the posterior parts of the lateral lobes, or of the supplemental bodies occasionally found in this situation. Pressure on the trachea is by far the most important of the consequences which goitre entails. In some instances it acts unilaterally, the trachea being displaced towards one side of the neck; in some instances this tube is compressed between the two enlarged lateral lobes; in some the pressure is exerted in the antero-posterior direction, the trachea then becoming more or less flattened against the spine. The effect of pressure in either of these cases is often remarkable: the implicated portion of tube being sometimes flattened, sometimes made to form a convex bulging, so that consequently the passage becomes on transverse section semilunar or concavo-convex, or (if pressure be exerted equally on opposite sides) a rectilinear or biconcave chink, and sometimes actually obliterated. A slight amount of compression is not unfrequent, the patient breathing naturally when quiet, but with some degree of stridor and difficulty under exertion or excitement, and yet not with sufficiently pronounced difficulty to excite alarm in himself or others. In all such cases, however, there is danger of the supervention of fatal obstruction. In some this takes place gradually from the slow encroachment of the tumour; but in many it comes on more or less suddenly either from the rapid development of some cyst, or from inflammatory tumefaction, or from congestion and œdema of the mucous membrane of the already compressed trachea. The danger of suffocation depends less on the size of the goitre than on its form and situation; the most serious cases, indeed, are: first, those of *sub-sternal goitre*, in which the lower portion of the gland, or some process of it, sinks, in the course of its enlargement, behind the sternum, and compresses the trachea there, while there is yet little obvious sign of thyroid gland enlargement; and, second, those of *sub-maxillary goitre* (a congenital defect described by Virchow) in which the gland is situated at a higher level than natural, and the lateral lobes extend backwards behind the angles of the jaws, and sometimes as far as the mastoid processes. The contraction of the sterno-thyroid muscles may materially aggravate the compression of deep-seated organs. To the list of dangers just enumerated may be added that due to the rupture of cysts or abscesses either externally or into the trachea or œsophagus.

Treatment.—In the treatment of goitre we have to consider: first, the medicinal and other means by which the tumour may be either reduced in size or prevented from increasing; and, second, the measures which may be requisite to obviate the effects of its pressure upon important parts. Whenever a goitrous patient lives in a goitrous district the obvious remedy is his removal to some more salubrious locality; or, if this be impossible, a careful investigation of the available drinking water of the neighbourhood, and the selection for use of

that which is least contaminated with earthy salts, or the adoption of measures, such as boiling, distillation, or Clark's process, for the precipitation of these ingredients previous to use. Such measures, and especially emigration, are often efficacious in the complete removal of goitrous tumours which are of small size, or have been but a short time in existence, and are generally beneficial even in advanced cases. Burnt sponge was formerly largely employed, and with reputed benefit, in the treatment of goitre; but Dr. Coindet, of Geneva, after the discovery of iodine in sea-water and marine productions, was led to suspect that the efficacy of the burnt sponge was due to the iodine which it contained, and to make trial of iodine itself as an anti-goitrous remedy. Since that time iodine and its various preparations have replaced almost all other internal remedial agents and have enjoyed a singular reputation as specifics against this disease. The testimony, indeed, in favour of the curative influence of iodine is almost overwhelming. On the other hand, it must be remarked: that, altogether apart from the influence of iodine, goitre is liable to considerable fluctuations of size, and when small and recent often disappears entirely; that there is little or no evidence that the drug is efficacious in the treatment of exophthalmic goitre, which is structurally identical with the endemic form of the disease; that, notwithstanding the supposed curative action of iodine, there is no proof that goitre is now less prevalent or less severe in goitrous localities than it formerly was; and that, mixed up with the evidence in favour of the specific virtues of iodine, is evidence equally striking in regard to the production of a remarkable concurrence of symptoms known by the name of iodism, which now seems never to attend the use of iodine, however largely it is administered. We must confess that, in our own limited experience of the treatment of goitre, iodine has signally failed. But we need not limit ourselves to the employment of internal remedies. By many persons counter-irritants applied to the surface of the tumour are strongly advocated. Among such applications may be enumerated, iodine paint and other iodic preparations, strong mercurial ointment, and blisters or other forms of blistering agents. In some cases (generally, however, when the tumour has been of large size or has given indications of compressing vital organs), operative measures have been resorted to. The tumour has been excised—an operation of no inconsiderable difficulty and danger, owing to the relations of the thyroid body and its enormous vascular supply; it has been treated by passing a seton through its substance and so exciting and maintaining inflammation or suppuration in it; and, again, one or more of the arteries supplying it have been tied. Each of these operations has proved more or less successful in certain cases; but none of them sufficiently successful on the whole to encourage its frequent performance. It must be observed, however, that cysts of the thyroid body admit in most cases of ready and successful treatment, either by simple puncture

with the discharge of their contents, or by puncture and injection of some stimulating fluid, or by the employment of the seton.

When goitrous tumours are threatening to obstruct the trachea, we must be alive to the possibility of the supervention at any moment of sudden and fatal asphyxia. What can be done under these circumstances? Unfortunately very little. If the enlargement be mainly cystic, relief no doubt can be afforded by the puncture of the cyst and the discharge of its contents. If, however, it be solid, as in the main it commonly is, it is difficult to see what other resource than tracheotomy is left us; and tracheotomy in these cases is both difficult and unsatisfactory; for it can rarely be performed below the seat of obstruction; it is a formidable operation if effected through the substance of the enlarged gland; and if done above the gland it is necessarily useless unless it be completed by the passage of a sufficiently long tube through and beyond the constricted portion of the trachea.

B. *Cretinism.*

Cretins are persons in whom feebleness of intellect or idiocy is combined with certain peculiarities of bodily conformation. They are for the most part stunted in growth, with tumid bellies and coarse skins. In a large proportion of cases they are more or less obviously goitrous, though occasionally the goitre is of the latent or submaxillary kind, to which reference has already been made. The head is usually large and misshapen—expanded at the sides and flattened at the top; the cheek-bones high and prominent; the nose flattened or sunken at the bridge, broad at the root, and upturned; the interval between the eyes increased; the lips thick; the mouth wide and open; and the tongue large. There is generally more or less muscular weakness, deficiency of cutaneous sensibility, and impairment or annulment of the sexual functions; and not unfrequently deaf-mutism is conjoined with the other corporeal defects. The degree of mental impairment varies between complete dementia and mere dulness or slowness of intelligence. Cretins are usually quiet and harmless, not given to mischief, but liable to occasional outbursts of ungovernable violence.

True cretinism appears, according to Virchow's researches, to originate during fœtal life in an unnatural tendency which the basilar portion of the occipital bone, and the post-sphenoidal and præ-sphenoidal bones, have to coalesce with one another by ossification of the discs of cartilage by which they should at that time be separated. The consequences are: that the base of the skull ceases prematurely to elongate, and thus becomes modified in form; and that this arrest of development leads, on the one hand, to defective development of the corresponding portion of the brain, and, on the other, to wide-spread changes in the osseous framework of the skull and face. The form of the skull gets modified, partly by the need which its contraction in one direction involves of

compensatory expansion on the part of those regions whose bones have not yet coalesced, and partly by the opposing tendency which also exists in these cases to precocious union of the bones of the cranial vault along the lines of suture. The peculiar form which the face assumes is due in some measure to imperfect development of the nasal septum, in some measure to displacement of the cheek-bones and bones of the orbits. Further, in many of these cases the cranial bones acquire remarkable thickness, and the foramina at the base of the skull become much diminished in size. The same tendency which is presented by the cranial bones is presented by those of the extremities, which soon unite with their epiphyses. And, indeed, it is probably due, in part at least, to this cause that these bones remain incompletely developed.

According to the above account of the pathology of cretinism, this condition must be regarded as of congenital origin. Children are born cretins; that is, they are born, either with the peculiar features of cretinism more or less obviously developed, or with that coalescence of the bones at the base of the skull which necessitates the gradual development of cretinism during the period of childhood.

Like goitre, cretinism may occur either sporadically or endemically. The causes of sporadic cretinism and those of sporadic goitre are alike obscure. Endemic cretinism, however, and endemic goitre are always associated, and obviously originate in a common cause. Wherever goitre prevails largely there cretinism is also prevalent; the goitrous tendency, however, occupies a wider area, and goitrous persons always largely outnumber their idiotic compatriots. It would seem, indeed, that for the production of cretinism some special intensity of the poison which also causes goitre is requisite. Cretins are not only in large proportion goitrous, but also in large proportion the offspring of goitrous parents. Yet there is no sufficient reason to believe that cretinism, any more than goitre or ague, is hereditary; for goitrous parents do not beget cretinous children when once they have removed from those regions in which these affections prevail, and under similar circumstances the children of cretins are themselves free from both goitre and taint of cretinism. It seems clear indeed, that the morbific matter which, taken into the mother's system, renders her goitrous, acts also on her fœtus, causing in it, may be, not only goitre, but also those special developmental changes which ultimately lead to malformation and mental deficiency. In reference to the association in cretinous infants of arrested development of the base of the skull and goitre, it is interesting to bear in mind the fact, pointed out by Virchow, of the close proximity in the fœtus of the base of the skull to the thyroid body. Assuming the common cause of goitre and cretinism to be, or to have some close relation with, the existence of a superabundance of earthy salts in drinking water, it is natural to speculate on the influence which these salts may have in causing the too early completion of the process of ossification.

We have referred to the great obscurity which involves the causa-

ɹtre and sporadic cretinism. There is no
ɹcts of sporadic goitre ever beget either goitrous
ɔn, or that sporadic cretins are ever the offspring of
ɹecile parents. At the same time sporadic cretins seem
esent some abnormal condition of the thyroid body. In
ɔorded cases such cretins have been distinctly goitrous; but
ɹarge proportion of them there is an apparent absence of the
ɹroid body. In Dr. Fagge's[1] cases, and in two previously recorded
y Mr. Curling, there were soft elastic lumps occupying the angles between the sterno-mastoids and clavicles, which lumps, in Mr. Curling's cases, were found post mortem to consist of fat only. It may, however, be questioned whether these are not to be regarded as examples of that latent form of bronchocele which Virchow speaks of, and to which attention has already been directed. These facts evidently ally the cases of sporadic cretinism to those of the endemic form of the malady, and suggest the dependence of both on a common cause; the poison (if it be a poison) being introduced, in the one case constantly and indifferently into the systems of a more or less ɹnsive population, in the other case accidentally, so to speak, into the blood of casual units.

Treatment.—The mental condition of cretins, like that of other idiots, admits in many cases of amelioration by proper training; for which purpose a well-ordered asylum with skilled officials is essential. The improvement, far more the cure, of the structural lesions which underlie cretinism is, however, entirely beyond the resources of our art. The prevention of cretinism depends, so far as we know, neither on the prevention of marriage between those who are goitrous or in a condition of semi-cretinism, nor on prophylactic measures adopted with reference to the young children in whom its presence is obvious or merely suspected; but solely on the observance by the parents of those special hygienic measures which are efficacious in the prevention of goitre.

II. MYXŒDEMA.

Definition and history.—This is a peculiar disease, hitherto recognised only in adult females, and characterised mainly by the general development of a kind of solid œdema, in connection with a tottering feeble gait, slow and monotonous utterance, and general slowness of thought and movement.

It was first described by Sir W. Gull[2] as a 'cretinoid state supervening in adult life in women;' and has since been investigated by

[1] 'Med. Chi. Trans.' vol. liv.

[2] 'Clinical Society's Transactions,' vol. vii.

Dr. Ord,[1] who has also had the opportunity of ex[illegible]ir increase in number into the morbid anatomy of the disease. We have [illegible]changes which ensue, cases and specimens, and are satisfied that myxœ[illegible]te destruction. In disease; but, while admitting that it has some resem[illegible]glands is usually nism, we are by no means satisfied that it has any essenti[illegible]ually enlarged, with that disease, and we place it after cretinism only provis[illegible]adherent to

Causation.—So far as is known myxœdema occurs only in [illegible]re found and never attacks them prior to adult age. No cause for it has been discovered. sub- are

Symptoms and progress.—It begins insidiously, and only after some years attains its full development. At that time the condition of the patient is very remarkable. She is probably well-nourished and even fat. There is general œdema, which is more marked in the face and hands than elsewhere; and the skin is for the most part dry and harsh. The œdematous parts do not pit on pressure. The features become thick, the alæ nasi tumid, the lips large and pendulous, and the connective tissue round the eyes swollen, translucent and colourless. The skin of these parts and of the rest of the face, though dry, is smooth, delicate-looking and slightly translucent; and although the face has generally a pale and waxy aspect, the lips are more or less rosy, and there is a persistent circumscribed blush upon the cheeks. The hands are large, thick and clumsy, and as Sir W. Gull describes them 'spade-like'—an appearance which is due to the fact that the fingers are thickened, have lost their natural markings and contour, and are pressed and flattened against one another. Her utterance is slow, monotonous and thick in tone, something like that which characterises tonsillitis. She speaks as if her tongue were too large for her mouth, and as if also there were some impediment to the free use of her organs of articulation. On looking into the mouth the tongue probably appears to be actually larger than natural, and the interior of the cheeks and the soft palate, like the superficial parts of the body, œdematous. The muscles generally appear to be well-developed; but there is manifest feebleness, especially in the lower extremities; she walks with a tottering uncertain gait, and occasionally her legs give way under her while she is walking, and she falls down. The patient's expression is placid and foolish; and, just as she is slow and deliberate in her speech and walk, so she is slow and deliberate in all her movements and mental processes. It takes her probably twice as long to get through her work, of whatever kind, whether mental or bodily, as it did when she was in health; and her interest in what is going on about her is manifestly diminished. Yet notwithstanding this slowness of thought and action, there is little or no actual mental incapacity—she retains her memory and understanding, and probably expresses herself as well as ever she did in speech and writing; and

[1] 'Med. Chi. Trans.' for 1877–78.

lysis of any muscle, no numbness or tingling; she touch unimpaired, and has the full use of her eyes, gans of sense. Further there is not necessarily dis- ortant internal viscus; the heart and lungs are healthy, ıo normal in quantity and quality. Nevertheless in Dr. ı fatal cases, which had been long under observation, albu- ıa supervened before death, and apparently caused it. The ase appears to be incurable.

Morbid anatomy.—The chief condition discovered by Dr. Ord after death was general œdema of the connective tissue, including that of the kidneys, liver and heart. The œdematous tissue of the skin was examined chemically by Drs. Ord and Charles and found to contain mucine in comparatively large quantities—ordinary œdematous skin presenting mere traces or several hundred times less. It is from this peculiarity that the name of the disease has been derived. The myx-œdematous condition of the kidney closely simulated subacute interstitial nephritis. The brain was healthy. It may be added that Dr. Ord believes that the thyroid body in these cases undergoes atrophy.

Treatment can only be palliative.

III. DISEASES OF THE SUPRA-RENAL CAPSULES.

The supra-renal bodies are doubtless liable to most of the organic and other lesions to which other organs are liable; but there are only two such lesions of them which have any clinical interest, namely tubercle (Addison's disease) and malignant disease.

A. *Addison's Disease.* (*Melasma Addisonii.*)

Definition.—Tubercular infiltration of the supra-renal bodies, together with the remarkable group of symptoms which seem always to be associated with this lesion, constitutes the malady to which the name of 'Addison's disease' is now universally applied. When present in its typical completeness it comprises, in association, tubercular destruction of the supra-renal bodies, general pigmentary deposition in the rete mucosum, and a remarkable form of progressive asthenia which sooner or later ends in death.

Causation.—Addison's disease occurs much more frequently in males than in females, and is rarely if ever met with under ten or over fifty. Its first symptoms have often been attributed to local injury; and it is certain that it occasionally appears to supervene on caries of the neighbouring vertebræ.

Morbid anatomy and pathology.—Miliary tubercles appear in the

supra-renal bodies, as in other organs, and by their increase in number and size, their coalescence, and the degenerative changes which ensue, lead after a while to their more or less complete destruction. In fatal cases of this disease, the disorganisation of both glands is usually complete. They may be diminished in size; but are usually enlarged, forming nodulated, rounded, or irregular masses which are adherent to surrounding structures by cicatricial tissue. On section they are found to consist of dense, greyish, translucent, fibroid material, in the substance of which opaque, yellow, cheesy nodules of various sizes are imbedded in greater or less abundance. In some cases these have undergone earthy infiltration, in some have softened into tubercular abscesses. There are no lesions of internal organs or tissues which are constantly associated with the supra-renal affection. In a large proportion of cases there is absolutely no trace of any such complication; in about half the total number (or rather less) miliary tubercles have been met with in the lungs, peritoneum, mesenteric glands, and other parts; and in a small, but yet significant, proportion of them caries of the vertebræ has been present. The condition of the skin has a close resemblance to that of a mulatto; it is variously described as yellowish-brown, dark-brown, greenish-brown, or bronze-like. This discolouration, which is more or less general, affects especially those parts of the body which are most exposed, and those which are normally the seat of pigment. Thus, while it tints the face, neck, and hands on the whole more intensely than the chest, belly, and legs, it is usually especially dark in the axillæ, areolæ of the nipples, umbilical region, external genital organs and groins. The extensor aspects of the joints are generally more deeply tinged than the flexor, and the knuckles, therefore, and backs of the hands are darker than the palmar surfaces. The discolouration never presents an abrupt margin, but is occasionally spotty, especially on the face and neck; and it is for the most part especially deep upon surfaces which have been blistered or superficially destroyed. Deep cicatrices, on the other hand, tend to remain pallid. Similar brown discolourations may generally be observed along the lines of junction of the lips, and spots and patches of the same kind may often be discovered on the mucous surface of the cheeks, gums, and tongue. The change of colour is due, as is that of common freckles, or of the negro's skin, to the accumulation of molecular pigment in the cells of the rete mucosum. The hair is said occasionally to share in the general pigmentation.

The relation which exists between the tubercular disease of the supra-renal capsules, the discolouration of the skin, and the remarkable group of symptoms which attend these lesions, is as yet a matter of impenetrable obscurity. It has been suggested that the explanation of the phenomenon lies in the intimate connection which exists between these bodies and the great sympathetic in the abdomen. It has also been suggested that the supra-renal bodies, like other ductless glands, exert

some important influence over the condition of the blood, and that it is in the abolition of this influence that the source of the special symptoms of the disease is to be sought. But these are, at all events at present, mere barren speculations. It has never been shown that disease of the abdominal sympathetic induces symptoms resembling those of suprarenal disease; nor that the blood or the excretions in Addison's disease present any constant departure from the healthy state. It seems probable, however, that the morbid condition of the supra-renal bodies is directly or indirectly the cause of all the other phenomena of the disease.

Symptoms and progress.—The chief symptomatic phenomenon of Addison's disease is the gradual development of extreme debility, without commensurate, it may be without appreciable, loss of flesh. The patient observes: that he is less capable than he formerly was of sustained muscular exertion and less disposed for it; that he cannot walk far without suffering from shortness of breath and palpitation; and that if he persist in his efforts he falls into a state of prostration, which may continue on him for many hours or for days. Together with these symptoms he suffers from general lassitude and chilliness, and frequent sighing and yawning; he probably loses his appetite, and has occasional attacks of nausea and vomiting. He perhaps also complains of pains across the loins or sacrum, or in the epigastrium and hypochondriac regions. There may possibly be some giddiness and dimness of vision. The heart's action becomes extremely feeble, its sounds perhaps scarcely audible, and the pulse at the wrist small, weak, and sometimes imperceptible. As to rate, it may be normal, or quickened, but is often below the average. In the great majority of cases some obvious darkening of the skin goes along with the above symptoms; sometimes it precedes them in point of time, sometimes follows them, sometimes makes its appearance concurrently with them. It is often first observed by the patient's friends, who probably think that jaundice is coming on, or accuse him of want of cleanliness; but before long it gets quite obvious to the patient himself as well as to those about him. It is first recognised in the face, neck, and hands; and generally manifests itself on the upper half of the body earlier than on the lower half. The tint gradually increases in intensity, especially in those situations which usually tend to get darkest; but the degree which it ultimately attains differs greatly in different cases. In some, though obvious, it is slight up to the close of life; in others the skin acquires the depth of hue of that of a mulatto or negro. In a small proportion of cases no change of colour whatever ever takes place. The conjunctivæ in all cases maintain their normal pearly lustre throughout. The phenomena above detailed are associated with many negative features of significant importance. The skin remains cool, pliable and normal in texture; there is no rise of temperature; the tongue is clean and moist; and, beyond nausea and sickness, there are no indications whatever of inflammation

or organic disease of the chylo-poietic viscera; the bowels are regular; and the urine is scanty but normal in appearance and constitution, excepting that urea is for the most part largely reduced in quantity.

With the progress of the case the debility increases. This is not always obvious as the patient lies quiet in bed (to which he is probably before long confined) but especially manifests itself in the supervention of alarming prostration after any unwonted effort. The nausea and sickness increase, but are liable to variation, and may even disappear for a while; they are not unfrequently associated with good appetite. The patient suffers occasionally from headache in addition to his other pains, and complains at times of chilliness—his hands, feet, and nose probably becoming cold and livid from imperfect circulation; the temperature in the axilla not unfrequently falls a degree or more; sometimes, on the other hand, it rises one, two, or three degrees; and although no actual paralysis may be present, he is apt to complain of numbness in his lower extremities and to believe that he has lost the use of them. Towards the close of the disease the breath and skin often yield an offensive cadaveric odour; the skin occasionally becomes furfuraceous; the patient grows apathetic, and disinclined to make any unnecessary movement, or even to reply to questions; and, although now and then becoming delirious, usually remains conscious to the last. Death results from asthenia, and is sometimes brought on by a sudden attack of faintness, which may be referrible to some apparently trivial exertion.

It has been assumed in the foregoing account that the patient is free from tubercle of other organs or from vertebral caries. The presence of such complications tends more or less to mask the phenomena due to the supra-renal disease. It is important, however, to note that, even in complicated cases, the complications are rarely so extensive or serious as of themselves to cause death, or so engrossing by the phenomena to which they give rise as materially to obscure the diagnosis of the supra-renal lesion. It might, indeed, almost be said that the presence of tubercles in the lungs and elsewhere, or of caries of the spine, should bring with it a thought as to the possible presence of supra-renal complication.

There is unfortunately no reason to doubt that Addison's disease is always ultimately fatal. The duration of the malady is, however, subject to considerable variation. It is probably not possible in any case to ascertain the exact date of the commencement of the disease; there are good reasons, indeed, for believing that the process of supra-renal degeneration is always far advanced before the clinical signs of the affection reveal themselves. Counting, then, from this latter date, the malady is sometimes remarkably rapid in its progress—proving fatal in the course of two or three weeks—while sometimes it is prolonged for several years. More commonly it terminates fatally within a year. It is important, however, to observe: that the progress of patients with this disease is not always uniformly from bad to

worse; but that they are liable to attacks of nausea and prostration, so severe as to threaten life, alternating with periods of greater or less duration in which they gain flesh, and seem to be fairly comfortable and hopeful; that many subjects of it doubtless fight against advancing weakness, not admitting themselves to be out of health, until possibly one of those sudden failures of the vital power to which they are liable compels them to yield; and, lastly, that such sudden seizures may often be warded off by scrupulous avoidance of mental or bodily exertion, exposure to the influence of cold, and errors of diet, and thus the patient's life be greatly prolonged. The debility induced by supra-renal disease is in this respect very much like that which attends saccharine diabetes.

Treatment.—The cure of Addison's disease is beyond our power; and all, therefore, that we have to do is to endeavour, by counteracting the various secondary phenomena of the disease, to prolong life and render it endurable. It is of the utmost importance to maintain the patient at rest, as regards both mind and body, and to keep him warmly clad and in an apartment of agreeable and moderate temperature. Sickness and irritability of the stomach should be relieved by appropriate remedies; tonics (the nature of which must be determined by the condition of the patient's digestive organs) should be administered; and he should be nourished and supported by wholesome and nutritious food, with such a proportion of alcoholic stimulants as may seem to be needed.

B. *Tumours of the Supra-renal Capsules.*

The various forms of malignant disease are all apt to attack the supra-renal bodies secondarily; and in rare cases these organs are the seat of their primary development. When the disease is secondary, the supra-renal growths rarely attain a large size, and probably nothing occurs during the whole course of the case to direct attention to them. When, however, the disease is primary in them, they may form tumours as large as a cocoa-nut, which from their size and situation may be easily recognised during life. It would be difficult, if not impossible, to distinguish such tumours from renal tumours; they occupy, in fact, exactly those situations which tumours originating in the upper part of the kidneys would occupy. They form rounded or lobulated immovable masses, springing from the posterior part of the abdomen, and are usually crossed by the ascending or descending colon which they push forwards in their growth. Their development is sometimes attended with frequent paroxysms of agonising pain, and always with the emaciation, debility, cachexia, and other phenomena which are associated with the progress of visceral malignant disease; but never, so far as is known, with the specific symptoms of Addison's disease.

IV. DISEASES OF THE SPLEEN.

A. *Congestion.*

Causation.—Congestion of the spleen is a condition of common occurrence under a large number of circumstances. It habitually takes place during the progress of digestion. Pathologically it is mainly observed: first, in dependence on lesions involving mechanical impediment to the escape of blood from the spleen, such as obstructive cardiac and pulmonary affections, and especially those diseases of the liver in which the portal vessels are implicated; and, second, in connection with numerous acute febrile disorders, of which typhus, enteric fever, pyæmia, and malarious affections may be taken as the types.

Morbid anatomy.—In congestion the blood accumulates in the small vessels and intervascular blood-passages, and the organ becomes proportionately enlarged. The rapidity with which this enlargement takes place and subsides is quite remarkable. The congested organ may attain five or six times its original bulk, while retaining its normal form; and usually becomes, in proportion to the amount of blood which it contains, pulpy, lacerable, and even diffluent. When the congestion is frequently repeated, as in ague, or long continued, as in portal obstruction, the enlargement tends not only to increase, but to become permanent.

Symptoms and progress.—Simple congestion of the spleen rarely, if ever, reveals itself by symptoms, and equally rarely calls for special medical treatment. It can, however, often be recognised during life (if sought for in those cases in which it is specially liable to occur) by the presence of a manifest tumour in the splenic region. The normal spleen is situated upon the cardiac extremity of the stomach, its convex surface being in contact with the diaphragm, and no part descending below the ribs. Its lowest point is then in close proximity with the anterior extremity of the eleventh rib, from which point upwards a limited area of dulness, due to its presence, may sometimes be detected on the left side of the thorax. The enlarged organ, however, while partly rising into the chest and increasing the area of splenic dulness in that situation, mainly spreads farther and farther into the abdominal cavity, taking a course downwards and inwards. In cases of extreme enlargement it may occupy nearly the whole of the left half of the abdomen—extending from the ribs above to the inguinal region below, and from the lumbar region behind to beyond the umbilicus, and causing distinct protrusion of the abdominal parietes. A splenic tumour is usually readily movable, sinking and rising with the respiratory movements, and capable of obvious displacement under manual pressure; its anterior edge can generally be readily felt, and found to present the characteristic splenic notches. If symptoms be present

they are mainly a sense of weight or tension in the side and more or less tenderness on pressure. It has occasionally happened that rupture of the greatly congested spleen has taken place; in which case death has occurred with some rapidity, either from the escape of blood into the peritoneal cavity, or from peritonitis.

Treatment.—The treatment of hyperæmia consists mainly in the treatment of the morbid condition which gives rise to it.

B. *Hypertrophy.*

Causation.—True hypertrophy is for the most part the consequence of long-continued or repeated congestion. It is therefore frequently found associated with cirrhosis and other chronic affections of the liver, and is a common consequence of repeated attacks of malarious fever. It is, moreover, a usual complication of rickets. But some of the most remarkable examples of this affection are furnished by persons who have never suffered from any of the above disorders, and in whom there is no history pointing to the operation of any specific cause.

Morbid anatomy.—In true hypertrophy, the organ enlarges without undergoing any obvious change in texture; there is a general increase of all its elements in pretty nearly equal proportion, and it acquires for the most part a more or less firm fleshy consistence. It is in this condition that the spleen attains its greatest volume, sometimes filling the left side of the abdomen, from the ribs above to the pelvis below, and from the lumbar region behind to some inch or two, or more, beyond the umbilicus. It may then measure as much as sixteen inches in length, ten in breadth, and five or six in thickness, and weighs ten, twelve, or even twenty pounds. It retains its normal shape.

Symptoms and progress.—The symptoms due to simple hypertrophy are, for the most part, very vague, and difficult to disentangle from those of other lesions with which they are frequently associated. Persons thus affected often suffer from anæmia, discharges of blood (especially from the gastro-intestinal mucous membrane), and abdominal dropsy; but it is uncertain how far these phenomena depend on the hepatic lesion which so commonly goes along with splenic enlargement, how far on the splenic disease. But, putting such symptoms aside, there is nothing left to indicate the presence of splenic hypertrophy beyond the local phenomena to which it gives rise. The chief of these is the manifest existence of a tumour, which presents the characters (before described) of enlarged spleen, is tough and unyielding in consistence, gives to the patient a sense of weight and fulness, especially if he lie upon his right side, and is unattended with pain or tenderness on pressure. A venous hum, of more or less musical character, may occasionally be recognised on the application of the stethoscope over the tumour. The duration of these cases is always uncertain, and often much prolonged. In some instances amelioration

or cure takes place under suitable treatment; in some the organ remains stationary, and yet with little manifest deterioration of the patient's health; in many death ensues sooner or later, either from simple anæmia and debility, or from these conditions associated with hemorrhage, dropsy, or some other intercurrent affection.

The *treatment* of hypertrophy must depend largely on the constitutional malady which has given rise to it. If it be a sequel of ague, quinine or arsenic is indicated; if the patient be suffering from rickets the remedies suitable for that condition must be employed; if there be heart, pulmonary, or renal disease, our efforts must be regulated accordingly. In many cases, however, no such clue is furnished; and we must then have recourse to those remedies which the general condition of the patient seems to suggest; among the more important of which may be enumerated iodine, iodide and bromide of potassium, iron, quinine, and other tonics. The bowels should be kept freely open—if necessary, by the use of mild laxatives.

C. *Inflammation.*

Causation.—Inflammation of the spleen, at least in an acute form, is exceedingly rare, excepting in those cases in which it is due to injury, embolism, pyæmia, or the presence of morbid growths or foreign bodies.

Morbid anatomy.—Splenic embolism is most frequently a consequence of valvular disease of the heart. It leads to the formation of wedge-shaped blocks, or masses, which vary in size from a cubic inch or two downwards, are often multiple, and usually abut on the surface of the organ. In the first instance they are mainly hemorrhagic, and distinguishable from the splenic tissue by their darker colour and greater solidity; but soon the colouring matter gets absorbed, and the masses pass through various stages of reddish-brown, yellowish-brown and buff colour, until they become almost pure white. Sometimes they soften into a puriform pulp, sometimes undergo actual suppuration, and sometimes (especially if small) get absorbed, leaving depressed cicatrices behind, in which earthy particles may remain imbedded. The presence of these infarctions generally gives rise to more or less inflammation in the peritoneal surface over them.

Pyæmic formations present much the same characters; but they are usually more numerous and smaller, and their tendency to soften, suppurate, and involve the peritoneum covering them, is much more marked.

Splenic abscesses may result from the above and various other causes, and, like other abdominal abscesses, may acquire large dimensions, and are liable to various terminations. They may open externally through the abdominal walls, or rupture into the peritoneum, or discharge their contents into the stomach, colon, left lung, or pleura.

Adhesive inflammation is not uncommon at the surface of the spleen, and occasionally circumscribed suppuration occurs between this organ and some neighbouring part, such as the stomach, diaphragm, colon, or abdominal walls.

Symptoms.—In most of the affections now under consideration there is little or nothing special excepting locality to direct attention during life to the spleen as the seat of disease. There may be, and indeed probably always is, manifest increase of size of the organ, together with uneasiness, pain and tenderness. The pain, when severe, is mainly due to circumscribed peritonitis, and, from the position and relations of the organ, is liable to augmentation during the respiratory movements. The recognition of an abscess will depend on its attainment of such a size as to form an appreciable fluctuating tumour in the splenic region, and on the phenomena which attend and follow the process of pointing and the discharge of its contents. In all these cases, sympathetic vomiting and febrile symptoms of more or less intensity will almost certainly manifest themselves, and rigors are not unlikely to supervene. But it is rare for the splenic affection to be so free from complication as to justify us in attributing them to it.

Special treatment will only be called for when pain is complained of or when an abscess becomes manifest. In the former case, poultices, fomentations and leeches are the most useful applications; in the latter the case must be treated as one of hepatic or other internal abscess.

D. *Tubercle.*

Tubercles are very common, especially in young children, and in connection with tuberculosis of other organs. The spleen thus affected is usually somewhat enlarged and studded more or less thickly with them. They are frequently miliary and grey, in which case they may be readily mistaken for the Malpighian bodies; usually, however, some of larger size may also be detected which have already undergone caseation, and thus furnish a clue to the nature of the others. Yellow tubercular masses, irregular in form, and varying from the size of a horse-bean to that of a tare, are also not unfrequently discovered, in greater or less abundance. Occasionally they soften into cavities or form abscesses. Filamentous processes of false membrane, themselves studded with tubercles, are often attached to the surface of tuberculous spleens.

Tuberculosis of the spleen can scarcely be recognised during life. If symptoms attend it, they will be such as to suggest either congestion, abscess, hypertrophy, or some other than tubercular lesion.

E. *Tumours.*

The various forms of malignant disease affect the spleen with different degrees of frequency and in different modes. First, the peri-

toneal aspect, or the connective tissue about the hilum, may get involved by continuity, in the course of malignant disease of the peritoneum, stomach, or glands in the neighbourhood of the stomach; and then the morbid growth either invades the organ from different parts of its surface, or runs into its substance along the vessels which enter at the hilum. Second, the spleen may have isolated secondary growths developed here and there in its substance. Or, third, it may be the seat of the primary manifestation of the disease. The last alternative, however, is rare.

Most of the different forms of malignant disease fail to cause any great enlargement of the spleen, or to indicate their presence by special symptoms; and consequently the splenic affection is usually overlooked during life. Still such growths may attain considerable size in that organ, and convert it into an irregular and more or less indurated mass, readily recognisable during life by palpation, and even (in connection with other phenomena) as a malignant growth of splenic origin. It must not be forgotten, however, that tumours of the great omentum, or other parts of the peritoneum in the neighbourhood of the spleen, are very apt to simulate splenic tumours and to be mistaken for them. These remarks do not apply to lymphadenoma. In this, as in simple hypertrophy, the spleen undergoes a nearly uniform enlargement; sometimes acquiring gigantic proportions, but still retaining its natural form, and the characteristic features by which an enlarged spleen may usually be recognised.

The *symptoms* of splenic malignant disease are not usually of much interest or importance. Those which attend lymphadenoma of the spleen will be most conveniently discussed hereafter in connection with those due to the same affection of the lymphatic glands.

F. *Cysts and hydatids.*

Simple serous cysts are rare in the spleen, and, so far as we know, unimportant. They are occasionally multiple, and associated with the development of numerous similar cysts in the liver and kidneys.

Hydatids are more common and far more important. But their course and the symptoms to which they give rise are identical (excepting in one or two obvious particulars) with those of hydatids of the liver or peritoneum, and need not be particularly considered now.

G. *Atrophy.*

Atrophy is exceedingly common, and traceable to various causes. In some cases it appears, like cirrhosis of the liver, to be consequent on an interstitial overgrowth of connective tissue; in some, as also occurs in the liver, to the investment of the organ in a dense and slowly contracting fibrous capsule. But, however produced, it is a

lesion which, so far as we know, causes little or no inconvenience and no symptoms by which its existence may be diagnosed.

H. *Lardaceous Degeneration.*

Morbid anatomy.—The spleen is perhaps more frequently the seat of the lardaceous change than any other part of the body; but it is generally thus affected in association with one or more of the several other organs which are liable to the same change. Lardaceous degeneration first affects the minute arterial twigs and the cells external to them with which they are in relation. It is especially apt to commence in the Malpighian bodies and vessels which are connected with them. The lardaceous spleen undergoes gradual and uniform enlargement, and may attain dimensions nearly as extreme as those reached by the simply hypertrophied organ. Its capsule is usually smooth and glistening; and on section the organ presents different appearances according to the degree to which the degeneration has advanced. In the earlier stages it exhibits those characters which have gained for it the name of the 'sago' spleen. It is thickly studded with greyish translucent rounded masses, which have a close resemblance to boiled sago-grains, and which are separated from one another by a network of still healthy tissue. In the later stages these rounded bodies have coalesced, and the spleen is involved uniformly in its whole extent. In this condition the spleen presents on section a nearly uniform greyish, translucent, glistening aspect, yields little or no fluid on pressure, and takes the impress of the finger like a piece of wax or stiff dough. It is abnormally heavy, and readily lacerable—breaking however with a somewhat vitreous fracture.

Symptoms.—Lardaceous spleen is always associated with more or less anæmia or cachexia, and often with dropsy, tendency to hemorrhage, and other symptoms, for the most part indicative of debility. It is never possible, however, to decide to what extent these various symptoms depend on the splenic disease—which is always secondary to grave chronic lesion of other organs, and generally associated with similar degenerative changes elsewhere; to what extent they are referrible to these several antecedent or concurrent affections.

Treatment.—Lardaceous spleen probably never calls for independent treatment. Our first efforts must be directed to the cure of the lesion out of which the tendency to lardaceous change has arisen; our next to the improvement of the patient's general health by the exhibition of iron and other tonics, the administration of abundant nutritious food, and attention to all those hygienic measures which are generally beneficial in cachectic conditions.

V. DISEASES OF THE LYMPHATICS.

There are probably no organs or tissues of the body the pathological relations of which are more important than those of the lymphatic vessels and glands; no organs which are more frequently involved in the course of diseases originating in other parts; none, the proper diseases of which more profoundly affect the general organism. Their affections are, for the most part, however, so intimately connected with those of other organs, or with so-called 'general' diseases, that the discussion of the latter necessarily involves that of their lymphatic complications. It is needless, therefore, notwithstanding its surpassing interest and importance, to enter at any length upon the subject of the diseases of the lymphatic system.

A. *Inflammation.*

Causation.—Inflammation of the lymphatics is, no doubt, sometimes primary, in the sense in which idiopathic pneumonia is primary, and sometimes the consequence of blows or other forms of direct mechanical violence; in the great majority of cases, however, it arises secondarily to some local inflammation, or is the consequence of some irritant acting through the blood.

Morbid anatomy.—If the glands be secondarily affected, those only suffer which lie next above the inflamed area, in the line of the lymphatic vessels In this case irritating matters, probably the products of inflammation, are taken up by the lymphatics, and conveyed along them until they get arrested in their progress by the glands. During the passage of these matters the vessels sometimes inflame, their parietes get thickened and vascular, and the connective tissue around them congested and infiltrated; and thus their course becomes indicated by red tumid bands. Sometimes, indeed, abscesses form along them. On the other hand, lymphatic vessels frequently convey, without injury to themselves, matters which excite violent inflammation in the glands, and ulterior mischief of the gravest character. Inflammation of the lymphatic glands is marked by hyperæmia, succulence, softening and swelling, and an excessive development of cells resembling those natural to the healthy organs. Suppuration sometimes ensues, and occasionally (especially among lax tissues such as that of the axilla) enormous abscesses result. In some instances the inflammation assumes a chronic character, and ends in the induration, contraction, and atrophy of the glands. The nature of the inflammation, and its tendency in respect of result, differ in accordance with the characters of the local inflammation, and of the specific disorder to which it owes its origin.

Symptoms and progress.—The symptoms due to lymphatic inflam-

mation are principally swelling, heat, pain and tenderness in the course of the affected vessels and in the affected glands, with visible hyperæmia in the situation of such as occupy a superficial position, and febrile symptoms of more or less severity. Indeed the fever is generally severe—apparently out of all proportion to the extent and importance of the inflamed tract—and not unfrequently attended with rigors. Its severity is doubtless due in no small degree to the fact that the inflamed lymphatics are in direct communication with the blood, and are constantly pouring the products of their inflammation into it.

Treatment.—For the general treatment of inflamed glands (supposing them to need any apart from the affection to which their inflammation is secondary) no rules need be laid down beyond such as should guide us in the treatment of tonsillitis and other such disorders. For local treatment, leeches, fomentations, poultices, and in some cases cold applications, are chiefly important. When the inflammation is chronic, counter-irritants, iodine paint, strong mercurial ointment and blisters will probably be more efficacious.

B. *Tubercle. Scrofula.*

Morbid anatomy.—It is not easy to draw a distinct line between tubercle of the lymphatic glands and that enlargement of them which so commonly occurs in so-called 'scrofulous' children.

But however different these affections may appear to be from one another in their early stages, it is certain that in both there is an equal tendency for the affected glands to undergo speedy caseous degeneration, and to be converted into opaque yellowish, friable, fattily-degenerated masses, which, according to their situation and other attendant circumstances, either soften or suppurate, or become converted into encysted mortary or cretaceous masses. Softening with ulcerative destruction takes place especially in connection with mucous surfaces; softening with formation of abscesses, in the case of the glands which are superficially placed; cretaceous changes, in the glands of the mediastinum and mesentery, and others which lie deep in the interior of the body.

Symptoms and progress.—The symptoms of tubercular or scrofulous disease of the glands are rarely characteristic except when the affected glands are so situated as to admit of ready examination. They are then as a rule scarcely painful or even tender, and are usually indolent in their progress; suppuration is long delayed and slow to reach the surface; and even after the contents have been evacuated the abscess continues to discharge for an indefinite time; and when at length the cavity heals, the scar which remains is ragged and unsightly. The general symptoms are those of debility and constitutional weakness.

Treatment.—The general treatment of scrofulous disease of the glands consists in the use of tonics, cod-liver oil, and good, nourishing

diet, change of air, and generally careful attention to hygienic measures. The local treatment belongs mainly to the surgeon. So long as the glands are neither painful nor suppurating, it is probably best to trust wholly to constitutional treatment; but when pain or suppuration arises, poultices or fomentations are demanded, and, in the latter case, sooner or later the surgeon's knife.

C. *Morbid Growths.*

Morbid anatomy.—Malignant disease, commencing elsewhere, invariably soon attacks the lymphatics, and in the first instance those glands which lie nearest to the primary spot of disease, between it and the thoracic duct. These glands indeed generally become rapidly and extensively involved, forming large tumours, which sooner or later coalesce with one another, and implicate in the progress of their growth the surrounding tissues. Thus, in malignant disease of the tongue or mouth, the glands at the angle of the jaw first suffer; when the breast is the source of infection, the axillary glands; when the lungs, the bronchial glands; when the stomach or bowels, the mesenteric or retroperitoneal glands; when the penis, the glands of the groin; when the testicle, those lying in the lumbar region. In some cases involvement of the lymphatics forms a still more obvious factor of the disease; and it may be primary. The most remarkable example of this kind is furnished by lymphadenoma, which (as has been before pointed out) affects primarily not only the lymphatic glands but the lymphatic tissues throughout the system, and though not necessarily limited to these in its ulterior development, commits its ravages mainly upon them.

Symptoms and progress.—The constitutional symptoms caused by malignant disease of the lymphatics are mainly those of malignant disease generally; when, however, these organs are implicated, the morbid process has already begun to exert a specific influence over the system, and the so-called 'cancerous cachexia,' if not previously manifest, becomes for the most part rapidly developed. The local symptoms are those of a more or less painful rapidly growing tumour, the direct results of which depend upon its situation.

Treatment.—Palliative measures only are as a rule available in malignant disease of the lymphatic glands. Accessible glands occasionally admit of removal with temporary benefit.

D. *Mediastinal Tumours.*

Morbid anatomy.—Malignant tumours are of common occurrence in the mediastina, and are often primary in this situation. It is not always easy to determine in what tissue they have originated. It is certain, however, that they often appear to start from the lymphatic

glands in the posterior mediastinum, and from that part of the anterior mediastinum in which are situated the remains of the thymus gland. It is not improbable that they arise also in the substance of the connective tissue. The nature of the disease varies in different cases; sometimes it is cancer, but probably much more frequently sarcoma or lymphadenoma. The growth gradually increases in bulk, and, even if it did not originate in the lymphatic glands, very soon involves them, and gradually implicates all the surrounding parts. Thus it may invade all the tissues of the anterior and posterior mediastina, surrounding and involving the fibrous pericardium and the adjoining parts of the parietal pleuræ; or it may involve the roots of the lungs, extending along the bronchial tubes and vessels into the substance of the lungs, or implicating the neighbouring parts of these organs by continuity, and probably constituting large tumours in them; or it may extend into the cardiac walls, either infiltrating their substance or forming distinct growths. Further, it is apt, sooner or later, to implicate the trachea, bronchi, or œsophagus, the innominate veins or cava, or the recurrent laryngeal nerves; or to involve the lymphatic glands above one or other clavicle; or to lead to the development of tumours in the ribs or soft tissues of the thoracic walls. The dimensions which mediastinal tumours attain are sometimes enormous; they may become as large as an orange, cocoa-nut, or child's head; moreover in their growth they tend to cause much compression and displacement of parts. The heart, for example, may be carried into the left axilla, or even into the right.

Symptoms and progress.—The symptoms to which mediastinal tumours may give rise are necessarily very various, and depend mainly on their seat and bulk and the particular intrathoracic organs which they implicate. They are almost identical, indeed, with those caused by intrathoracic aneurysms. The early symptoms are vague, but not unfrequently include more or less progressive anæmia, debility, and shortness of breath. The more characteristic phenomena slowly supervene—the order of their sequence varying, however, in different cases. Sometimes the veins get obstructed; those of one half of the head and neck and face and of the corresponding shoulder, arm, and side of the chest, or those of both sides equally, become dilated, tortuous, and full; and the implicated regions acquire a ghastly, livid, or congested aspect, and get more or less puffy or œdematous. This limited congestion and œdema are very striking phenomena; especially when, as generally happens, the rest of the body is getting pallid and wasted. Sometimes the respiratory organs suffer, and the patient has difficulty of breathing, with cough, and probably expectoration. The symptoms then are either much like those of slowly advancing bronchitis; or, owing to implication of the trachea or recurrent laryngeal nerves, like those of laryngeal disease, and attended with hoarseness or aphonia, and attacks of suffocative cough; or, in consequence of the formation

of tumours in the lungs or of the supervention of pneumonia or pleurisy, like those ascribed to these several affections. Sometimes the symptoms are mainly cardiac, and simulate those due to valvular disease. Sometimes the patient has difficulty or pain in swallowing. And often in connection with cardiac, pulmonary, or laryngeal symptoms, or those of venous obstruction, he complains of vertigo, headache, and even of occasional attacks of momentary unconsciousness or slight convulsion. It is not uncommon to have blood in the expectoration; and late in the disease the sputa are apt to be abundant, muco-purulent, and fetid.

The diagnosis of mediastinal tumours is often largely aided by physical examination; by the gradual extension of the area of præcordial dulness, by the increase of resistance experienced on percussion, by the displacement of the heart or lungs, or by the supervention of pulmonary consolidation or pleural effusion, and the modification in the auscultatory phenomena which these several affections entail. It is further aided by the presence of localised dilatation of veins in the thoracic parietes. But the most important indications are those furnished by the development of tumours in the thoracic parietes or above the clavicles.

It must not be forgotten that in the course of mediastinal disease secondary tumours are apt to arise in other parts of the body; and that these occasionally cause more striking symptoms than the primary disease, which may then be overlooked. Thus it is not uncommon in these cases for secondary tumours to develop in the brain, and for the patient to die of the cerebral complication.

It is obvious that the symptoms of mediastinal growths are made up mainly of those due to implication of the various important organs which occupy the mediastina or abut upon them; and in order that the reader may have a clear conception of their variety and importance, and a thorough picture of the disease, we must refer him to the descriptions elsewhere given of the phenomena referrible to lesions of the several organs here adverted to.

It need scarcely be added that mediastinal tumours are progressive in their course, and always sooner or later prove fatal. The causes of death are various.

Treatment.—There are no special indications for the treatment of mediastinal tumours—symptoms must be dealt with as they arise.

E. *Obstruction and Dilatation of the Lymphatic Vessels.*

Morbid anatomy and symptoms.—Obstruction of the thoracic duct may be caused by the pressure of tumours, by disease of its walls, or by a morbid condition of its contents; but is of rare occurrence. It might be supposed that it would lead to very rapid innutrition, and at the same time to general dilatation of all the lymphatics, excepting

those of the right upper extremity and corresponding side of the head, neck and thorax. But experience and experiment alike seem to show: that whilst *sudden* obstruction usually results rather quickly in great over-distension of the lower part of the duct and especially of the receptaculum chyli, which presently ruptures with extravasation of its contents into the retroperitoneal tissue; *slowly induced* obstruction may be compensated for by the enlargement of existing communications between the obstructed left and the still previous right duct.

Obstruction occurring in a group of lymphatic glands in consequence of disease going on in them, or in a group of lymphatic vessels as a result of pressure upon them or of their involvement in disease, always leads in the first instance to stasis and accumulation of lymph within the tributary vessels, which consequently dilate, and subsequently to similar accumulation within the lymphatic spaces and to their disproportionately large expansion. The lymph-channels, indeed, and the tissues generally, become surcharged with lymph—a clear or milky yellowish alkaline fluid of a sickly odour, which contains albumen, fibrinogen, and lymph-corpuscles, and among other occasional constituents sugar and molecular fatty matter, and which, like the plasma of the blood, coagulates more or less perfectly on removal from the body. The result is the development of what is often termed solid œdema or leucophlegmasia of the implicated portion of the body; which becomes swollen and tense, and of a pale waxy aspect, but does not pit on pressure as in ordinary venous dropsy. And, further, if the condition be of long duration, and especially if it originated in infancy when the organism was undergoing rapid growth, the tissues of the affected region—not only connective tissue, but muscles, bones, and skin—all become distinctly hypertrophic. Obstruction and dilatation of the lymphatics is the essential feature or an important factor of several well-recognised pathological conditions. A particular form of enlargement of tongue, usually congenital, in which the organ tends to grow, to protrude from the mouth, and to interfere by its bulk with the growth of the jaws, has been shown by Virchow to be due to lymphatic obstruction. The tongue is honeycombed with dilated lymph-channels, and the seat of consequent overgrowth of all the tissues of the organ inclusive of the muscular substance and of the papillary surface. The upper extremity has occasionally become, from accidental circumstances, similarly affected. But the most frequent, and, on the whole, the most interesting example of such obstruction and its consequences is afforded by the lower extremity and the adjoining portions of the abdomen and genital organs, in the condition we have already described under the name of elephantiasis lymphangiectodes. The last morbid condition characterised by dilatation of the lymphatics to which we shall refer is elephantiasis Arabum, a disease which, like the last, is more fully discussed in another part of this volume.

Treatment.—It is obvious that no medicines are competent to relieve the various consequences of obstruction of the lymphatics; recourse can only be had to mechanical or operative measures. In enlargement of the tongue, portions of the organ have been excised with benefit; as also have portions of the prepuce when that structure has got hypertrophied.

VI. LEUCOCYTHÆMIA. (*Leukæmia.*)

Definition.—By the above term is meant a disease characterised by a combination of enlarged spleen, enlarged lymphatic glands, or both of these conditions, with an excess of white corpuscles in the blood.

Causation.—The cause of leucocythæmia is very obscure. Dr. Gowers[1] has recently shown that twenty-five per cent. of cases of the splenic variety of the disease presented a history of ague or of exposure to malaria. But certainly that origin cannot be suggested for the great majority of cases. All forms of the disease appear to be more common in men than women.

Morbid anatomy.—The anatomical substratum of leucocythæmia has already been discussed at some length under the head of lymphadenoma in an earlier portion of this work. We there pointed out that lymphadenoma is a form of disease especially apt to attack the lymphatic glands and spleen—sometimes the one or the other exclusively, but more frequently both, and then many other organs and tissues at the same time or consecutively. We also adverted to its influence on the condition of the blood. In a considerable number of cases (to which Dr. Wilks has given the name of anæmia lymphatica, Trousseau that of adenia) the disease is attended in its progress with gradually increasing but simple anæmia, the blood becomes progressively more and more watery, and the blood-corpuscles (red and white in equal ratio) gradually disappear. In other cases, which are undistinguishable anatomically from these, and in which the general symptoms and progress of the disease are as nearly as possible identical, progressive anæmia also takes place; but it is an anæmia distinguishable from the former by the fact that, while the red corpuscles disappear, the white multiply until, in advanced cases, they nearly equal their red companions in number, and after death are not unfrequently found aggregated in pale clots or thick creamy masses in the terminal branches of the pulmonary artery, the cavities of the heart, and the systemic vessels. We also pointed out in our account of lymphadenoma that two varieties of leucocythæmia had been distinguished by Virchow:

[1] 'Transactions of Pathological Society,' vol. xxix.

one in which the spleen was involved, and where the superabundant white corpuscles were of the normal size of these bodies; one in which the glands were implicated, and where the abnormal leucocytes were smaller than natural. It is an important fact, however, which has been clearly established by the discussion[1] on lymphadenoma at the Pathological Society: that leucocythæmia is much more frequently associated with lymphadenoma originating in, and limited to, the spleen, than with similar disease of the lymphatic glands; and that lymphadenoma exhibiting distinctly malignant characters seldom or never causes special changes in the blood.

Symptoms and progress.—Splenic leucocythæmia comes on insidiously. In some cases it is the painless enlargement of the abdomen which first attracts attention. In some cases gradually increasing asthenia, pallor, and shortness of breath are complained of for some time before the condition of the abdomen is observed. And occasionally all the other phenomena are preceded by irregularly recurring slight febrile paroxysms. But under any circumstances the patient gradually gets anæmic, loses flesh and strength, becomes incapable of exertion, short-breathed and liable to palpitation, and the abdomen gets large, solid and heavy; and then careful examination reveals the fact that the spleen is enlarged, perhaps enormously, extending not only upwards into the chest, but probably downwards to the groin, and across the mesial line of the abdomen. The progress of the case is slow, but as it goes on: the patient's languor and debility gradually increase; his pulse becomes frequent—up to 90 or 100; his breath continues short, especially on exertion or under excitement, and is from time to time deep-drawn or sighing, and often attended with yawning; his tongue remains fairly clean; his appetite is variable, but on the whole probably pretty good; there may be some clamminess of mouth, if not actual thirst; and diarrhœa is liable to ensue; the urine is generally fairly abundant, acid, and loaded with urates, and often contains albumen in small quantity, with hyaline or granular casts; hemorrhages are apt to take place, either into the subcutaneous or subserous tissues, or from the mucous surfaces, more especially that of the nose; and occasionally anasarca, mainly of the lower extremities, and even accumulations of fluid in the serous cavities, supervene. Febrile symptoms are sometimes absent from first to last; sometimes the patient is liable to paroxysms, coming on at long and irregular intervals; and occasionally he suffers, either during his whole illness or towards its close, from well marked hectic fever—the temperature rising during the exacerbations to 101°, 102°, or even 103°. With this are necessarily associated night-sweats and other characteristic features of hectic. Besides hemorrhages and dropsical effusions, other complications are apt to supervene, especially during the later periods of the disease; among which may be enumerated splenic peritonitis, pulmonary

[1] 'Transactions of Pathological Society,' vol. xxix.

or pleural disorders, and the development of subcutaneous abscesses. It should be added that the spleen does not necessarily enlarge progressively during the whole duration of the patient's illness; but that it often becomes stationary after a while, or even liable to slight variations of bulk. Further, the blood always presents a large excess of white corpuscles. So far as is known splenic leucocythæmia is invariably fatal (probably within six months or two years of its first appearance) either by simple asthenia, or by this in conjunction with the effects of some intercurrent malady.

Lymphadenoma, affecting the lymphatic glands primarily, generally begins in a group of these bodies; and then either remains limited to this group, or, as more commonly happens, gradually involves the lymphatic glands and tissues of other parts of the body. The progress of the disease is generally attended with more or less anæmia, and symptoms not unlike those of splenic leucocythæmia; but the development of tumours forms an essential element in the case; and death is likely to ensue ultimately, as in other forms of malignant disease, not merely from gradually increasing debility, or intercurrent disorders, but from the involvement in the specific growth of vital or important organs, such as the larynx and trachea, heart, lungs, or abdominal viscera.

Treatment.—The successful treatment of splenic leucocythæmia, as also of lymphadenoma, appears to be altogether beyond the resources of our art. We can do little if anything beyond treating symptoms as they aris . and promoting the health of the patient by attention to diet, hygienic management, and the exhibition of iron or other tonics.

VII. IDIOPATHIC ANÆMIA. CHLOROSIS.

Definition.—Anæmia is the name applied to a condition in which there is diminution of the solid constituents of the blood and in particular of the red and white corpuscles, attended with pallor of the general surface and of the mucous membranes, palpitation, feebleness and rapidity of pulse, panting respiration, sighing and yawning, headache, restlessness, functional disturbance of the organs of sight and hearing, tendency to faint, and general debility. Idiopathic anæmia is a form of anæmia coming on independently of any organic lesion or dyscrasia, and chiefly in young women. In the last case it is usually termed chlorosis.

Causation.—Anæmia is a frequent complication or result of many morbid conditions: of the dyscrasiæ, for example, connected with tuberculosis, malignant disease, syphilis, and malarious affections, and of the more or less frequent and copious hemorrhages which take

place under various circumstances from one or other of the mucous tracts.

Idiopathic anæmia is occasionally met with in men; it also affects women of mature age, and those in whom menstruation is disappearing. But it is especially a disease of young females, from the period of commencing puberty to about twenty-five. Many causes have been assigned for it, such as deficient and unsuitable diet, unwholesome habitations, sedentary habits and want of fresh air, late hours, emotional affections, masturbation, and especially functional uterine or ovarian disturbances. It may be readily admitted that some of these conditions may be predisposing causes of chlorosis, it is certain that some of them may be consequences of it, but it is very doubtful if any of them can lay claim to being the exciting cause. The nature of this cause, indeed, is still veiled in mystery.

Symptoms and progress.—Chlorosis generally first reveals itself by gradually increasing paleness of the surface, palpitation, breathlessness on exertion, loss of muscular power, and more or less gastrodynia and impairment of the digestive functions, without loss of flesh. To these phenomena, however, many others sooner or later are superadded. The pallor usually becomes extreme—the general surface assuming a white or sallow wax-like appearance; the face, indeed, may present a greenish tinge—whence the name chlorosis. But the loss of colour takes place in the mucous membranes as well as the skin, and is for the most part strikingly obvious in the palpebral conjunctivæ, and in the lips and gums, which become in some cases scarcely distinguishable in tint from the skin itself. It may be pointed out, however, that, even in advanced cases, a fallacious bloom may appear in the cheeks under the slightest emotional excitement. Palpitation is a prominent symptom, and painfully apparent to the patient herself; it is rarely absent, and is always aggravated either by mental excitement or by bodily exercise; the rapidity with which the heart's contractions succeed one another is sometimes extraordinary, and not unfrequently their rhythm becomes remarkably irregular. The development of abnormal sounds in the heart and blood-vessels, independent of organic lesions, is of common occurrence and highly characteristic: a soft systolic murmur is frequently to be heard over the situation of the aortic or pulmonic valve, and along the course of the ascending arch and innominate artery; murmurs, coincident with the cardiac systole, may be developed more readily than natural by pressure on the subclavian, carotid, and other large arteries; and, lastly, continuous murmurs, more or less musical and varing from a feeble hissing to a deep droning (*bruit de diable*), may readily be evoked by the pressure of the stethoscope on the veins of the neck, more especially on the right side. The respirations are usually more rapid and shallow than in health, and occasionally become extraordinarily frequent, particularly under the influence of bodily exertion or emotional disturbance: and the patient

consequently complains of shortness of breath and inability to exert herself. There is usually some impairment of the digestive functions, with uneasiness or weight after food, flatulence, loss of appetite, and pain more or less severe and varying in character, either in the epigastric region or between the shoulders, in the left hypochondrium, or some neighbouring part. It is apparently in chlorotic girls that perforating ulcer of the stomach is most common, on which account their dyspeptic symptoms must always be regarded with suspicion and treated with care. The bowels are usually constipated. The urine, for the most part, is abundant, pale, and of low specific gravity. There is not unfrequently leucorrhœa; and although the menstrual function in some cases continues to be normally performed, it is usually at fault: the flow is sometimes regular, but scanty; sometimes profuse or too frequent, or attended with severe pain; most commonly there is amenorrhœa. Trousseau points out, and probably with truth, that the sexual appetite is diminished rather than (as is often asserted) increased. The muscular system becomes generally enfeebled; but the subcutaneous fat undergoes little or no diminution—sometimes, indeed, becomes increased—so that the patient, as a rule, presents more or less *embonpoint*. Some degree of anasarca, especially in the lower extremities, occasionally supervenes in the course of the disease. The nervous phenomena which are apt to attend chlorosis are many and various: there is usually more or less listlessness, inability of application to any pursuit or even train of thought, lowness of spirits, and irritability of temper; usually, also, chlorotic girls complain of neuralgic pains, sometimes in the face and head, sometimes in the intercostal muscles, sometimes in the internal organs or extremities. Again, they are not unfrequently hysterical, have depraved appetites, or suffer from paralysis or convulsions, or even become maniacal. It is rare for the chlorosis of young women to terminate fatally, or even to lead to the development of tuberculosis or any other organic disease, excepting, perhaps, ulcer of the stomach. Under proper treatment the patient generally recovers in the course of a few weeks or a month, but is liable to have relapses.

But anæmia coming on without obvious cause, especially in men, and in women above the age at which chlorosis is common, is occasionally altogether unamenable to treatment, and terminates sooner or later in death. During life such cases are liable to be mistaken, at any rate for a time, for cases of visceral cancer, undetected hemorrhages from the bowels, or Addison's disease without melasma. To this affection, the symptoms of which are in the main identical with those of the last-named disease, the designation of 'pernicious anæmia' is sometimes given.

Pathology.—The pathology of idiopathic anæmia is not at all understood. Trousseau regards chlorosis as a neurosis, looking upon the morbid condition of the blood as secondary to the nervous affection.

Some consider the reproductive organs, others the chylo-poietic viscera, as being primarily at fault. It is natural to refer the diminution of the corpuscular elements of the blood to some functional disturbance or organic lesion of the lymphatic tissues; but unfortunately nothing has yet been detected in their condition to justify this view. It is attempted to make a distinction between ordinary forms of anæmia and chlorosis by reference to the composition of the blood. Ordinary anæmia, it is said, is characterised by the diminution in equal proportion of all the solid constituents of that fluid, whereas in chlorosis it is the corpuscular elements which alone are deficient. It is clear, however, that this distinction can be of little value: for it is well known that when anæmia is caused by abstraction of blood, the corpuscles and other organic principles being removed in equal proportion, the albuminous and other such matters are far more speedily restored to that fluid than the corpuscles, and that hence (whatever may have been the patient's condition at first) a time speedily arrives in which the blood presents the assumed typical characters of chlorotic blood.

Treatment.—It is no doubt important in the treatment of chlorosis to obviate all possible sources of ill-health, and especially to secure for the patient change of scene, good air, moderate exercise, early hours, innocent amusement, and wholesome diet. But of far greater importance than these is the administration of iron. This metal, indeed, appears to be almost a specific remedy in this disease. Different authorities recommend different preparations; but they are probably all (if given in equivalent doses) equally efficacious. They are generally best administered in combination with some vegetable bitter or stomachic, such as quinine, cinchona, or calumba; and in association with occasional purgatives, such as aloes and myrrh pills, to obviate the obstinate constipation which is so often present. The form in which iron should be given must be determined by the special circumstances of the case. If dyspeptic symptoms are predominant, the tartrate of iron, in combination with an alkali and calumba or quassia, may be most suitable. It may even, under such circumstances, be well to delay the use of iron until some amendment in the condition of the stomach has been obtained by other measures. If menorrhagia be present, the perchloride of iron or the sulphate, in combination with mineral acids, may prove especially serviceable. Zinc is believed by some to have similar virtues to those of iron. In a large number of cases the ferruginous treatment cures not only the chlorosis, but the various complications—dyspeptic and uterine—which accompany the chlorosis: but that is not always the case, and just as it is frequently necessary to deal with the dyspepsia directly, so it may be essential to direct our treatment to the cure of the uterine derangement. In so-called 'pernicious anæmia' all the usual remedies appear to fail.

VIII. PURPURA.

Definition.—Extravasations of blood, in the form of points, petechiæ, vibices, or ecchymoses, are not uncommonly observed beneath the surface of the skin in various diseases, and under many other conditions, and are then often termed purpuric. Not unfrequently these subcutaneous extravasations (especially if due to constitutional disorders) are associated with similar extravasations into the solid organs, and beneath the serous and mucous membranes, and with more or less abundant escape of blood from these surfaces. Such extravasations are especially common in typhus, small-pox, measles, scurvy, obstructive heart affections, and liver disease, and are also met with in scarlet fever, diphtheria, pyæmia and embolism. They further occasionally complicate certain skin diseases, more especially some forms of erythema and urticaria, and may even be induced by mere exposure to atmospheric influences. But to none of these affections, however severe they may be, can the term purpura be properly applied.

Purpura, in the strict sense of the term (the *morbus maculosus Werlhofii* of the Germans) is the name given to a disorder characterised by such hemorrhages as have been above specified, but unconnected, so far as we know, with any local mischief or general specific disease.

Causation.—The causes of purpura or the conditions under which it arises are exceedingly obscure. It occurs at all ages, but mostly in young children of both sexes. It is frequently observed amongst those who are sickly, underfed, or surrounded by unwholesome sanitary conditions; but it is also met with amongst the robust and healthy-looking, and those whose hygienic and other circumstances appear to be unexceptionable. It is certainly not due to insufficiency of vegetable food, nor has it been traced to any special dietetic default. It is apt to recur; and consequently it is not uncommon to find a child (and apparently a healthy one) having periodical relapses, at intervals of three, six, or even twelve months.

Symptoms and progress.—Purpura is sometimes ushered in with vague premonitory symptoms, such as lassitude, loss of appetite, headache, and aching in the limbs, lasting from one to perhaps three or four weeks. In many cases, on the other hand, the characteristic lesions suddenly manifest themselves in the midst of apparently good health. The skin becomes more or less thickly studded with circular, deep red, almost black spots, varying from about a quarter of an inch in diameter downwards, which are unattended with any abnormal sensation, are not elevated above the level of the skin, and do not fade on pressure. They are usually most abundant on the lower part of the trunk and the lower extremities, but are by no means confined to these situations; and not unfrequently extravasations take place into

the eyelids, and beneath the conjunctivæ and the mucous surface of the tongue, lips, gums, and other parts within the cavity of the mouth. These spots go through the ordinary changes of colour which characterise bruises, and, thus fading away, usually disappear completely in the course of a few days. Successive crops of petechiæ, however, commonly appear from time to time, and thus the disease may be continued for two, three, or four weeks, and sometimes for a still longer period. Larger extravasations—vibices and ecchymoses—are usually associated in a greater or less degree with the eruption above described. But these are generally deeper seated, present less abrupt margins, are attended with swelling, and not unfrequently first reveal their existence as deep-seated bruises do, by the gradual diffusion and coming to the surface of their more or less modified colouring matter. They are not unfrequently the result of mechanical violence. There is always a tendency in these cases (more pronounced in proportion to their severity) for hemorrhages to take place from the mucous surfaces. Thus, there may be epistaxis, bleeding from the gums or other parts within the mouth, hæmoptysis, or bleeding from the stomach or bowels, kidneys or other parts of the urinary tract, uterus or vagina. In many cases the hemorrhage is small in quantity and of little importance; but occasionally it is profuse and frequently repeated.

When the affection is slight, the patient may seem during its continuance to be in good general health; more frequently, perhaps, he suffers from a continuance of such symptoms as may have ushered in the attack; sometimes the progress of the case is attended with febrile symptoms of a remittent type; but when profuse hemorrhages take place, the symptoms due to loss of blood get developed. Not only does the patient then become excessively pallid, but his pulse increases in frequency and gets more or less jerking; he has noises in his ears, dilated pupils, indistinctness of vision, with muscæ and headache; he yawns, becomes uneasy and restless, and sometimes falls into delirium, mania, or convulsions. Death is usually due to asthenia or syncope. His temperature is sometimes lowered, sometimes, on the other hand, considerably elevated. The milder form of purpura is sometimes termed *p. simplex*; the more severe, *p. hæmorrhagica.*

Morbid anatomy throws little light on this disease. Hemorrhages similar to those beneath the skin are sometimes discovered in the subserous and submucous tissues, and less frequently in the parenchyma of various organs, more especially the lungs, heart, and kidneys. Extreme fatty degeneration of the muscular fibres of the heart has been detected in cases fatal from repeated hemorrhage after long continuance of the disease. The blood seems to present no constant departure from the normal condition. It is curious, however, that Dr. Parkes has, in two cases which he has examined, detected in this fluid an excess of iron together with a general diminution of the solid constituents. It seems more probable, however, that the primary morbid condition is in the

capillary and other small vessels than in the blood, and that the latter escapes into the tissues in consequence of their rupture.

Treatment.—The principles of treatment of purpura are as little understood as its pathology. The majority of patients get well in the course of a week or two without treatment. The severer cases are unfortunately apt to go on from bad to worse, whatever treatment be adopted. A certain *primâ facie* resemblance which purpura presents to scurvy has induced a common belief that antiscorbutic remedies—fresh vegetables, citric acid, and potash—are indicated here also. Experience, however, does not confirm the truth of this opinion. Among the remedies which have been chiefly recommended are perchloride of iron, acetate of lead, arsenic, digitalis, turpentine, and gallic and sulphuric acids. If the discharge of blood be profuse, one or other of these drugs may be prescribed; and at the same time the patient should be kept quiet and cool, and should have ice or ice-cold drinks given to him. Hemorrhages taking place from accessible parts may, of course, be treated by local measures. If asthenia be extreme, it may be absolutely necessary to give alcoholic stimulants. On the whole, tonic treatment is indicated in those persons who have a tendency to purpura and in those who are convalescent from it.

IX. SCURVY. (*Scorbutus.*)

Definition.—Scurvy may be regarded as a peculiar form of anæmia arising from deficiency of vegetable diet, and attended with a tendency to the occurrence of hemorrhages, profound impairment of nutrition, and great mental and bodily prostration.

Causation.—Scurvy formerly occurred largely among sailors during long voyages. It has often broken out in armies on active service and among populations suffering from famine. It still occurs from time to time under these various conditions; and is occasionally met with as a sporadic affection among persons who are ill-fed, or whose diet has been, from some cause or other, too exclusively animal. It is needless to go into a history of scurvy, or to discuss the various hypotheses which have been propounded in reference to its causation. It will be sufficient to state that its origin has been clearly traced to insufficiency or total want of fresh vegetables; but among these must not be included corn and the other graminaceæ, or peas. It is still uncertain, however, to what constituent or constituents, common to vegetables, their virtue is due. Dr. Garrod believes it to reside in the salts of potash; others maintain that it dwells in the citric and other vegetable acids which they so often contain. There are objections, however, to both of these views; for the antiscorbutic powers of vegetables do not

appear to be proportionate to the potash salts they contain, and potash salts alone are probably inefficacious; and potatoes, which are powerfully antiscorbutic, are devoid, or nearly so, of vegetable acids. The constant use of salt meat, and long-continued exposure to privation and other such causes of ill-health, can only be regarded as indirectly favouring the production of scurvy.

Symptoms and progress.—The early symptoms of scurvy may be easily misunderstood when presented by sporadic cases; they cannot, however, fail to attract attention when they arise simultaneously or in rapid succession among a number of persons equally exposed to the conditions which are liable to give origin to the disease. They are: rapidly progressive anæmia, indicated by a dirty-looking, pallid, sallow, or earthy aspect; growing indisposition for bodily exertion; pains of a rheumatic character in the back and limbs; and more or less mental apathy or depression; while probably the tongue continues clean, though becoming large, flabby, and indented by the teeth, the appetite remains good, and the bowels are constipated. But soon other phenomena arise: petechial spots appear, first on the lower extremities, and then on other parts of the surface; and to these presently succeed large subcutaneous extravasations, and sooner or later, colourless puffy swellings, which seem to be due to deeper-seated and more copious hemorrhages, and the nature of which gets revealed ere long by the occurrence of bruise-like staining of the tissues superficial to them. These puffy swellings affect mainly the popliteal spaces, the corresponding parts of the elbows, the anterior aspect of the lower part of each leg, and the regions behind the angles of the jaw—interfering with the movements of these parts, and causing more or less pain and tenderness. Similar extravasations take place especially into the loose connective tissue in and about the eyelids, leading to considerable puffiness and bruise-like discolouration of these parts, and to sanguineous accumulation in the ocular sub-conjunctival tissue. Concurrently with the appearance of these hemorrhages the gums swell at their edges, and rapidly increase in bulk until they form lobulated masses, which rise up around the teeth, and sometimes hide them altogether from view. These masses are spongy, deep red or livid, and insensitive, but apt to bleed; they readily ulcerate or slough, and impart a fetid odour to the breath. The teeth get loose, and frequently drop out. The same tendency to ulcerate or slough is manifested in a greater or less degree by all parts of the surface of the body, but especially by those which are the seats of the puffy swellings above adverted to, and by those which present the cicatrices of former injuries. The slightest scratch, pressure, or blow is often sufficient to induce these destructive processes. Along with these phenomena the patient's anæmia increases; his face gets puffy; more or less anasarca takes place in his lower extremities; he becomes breathless; his heart acts rapidly and feebly; and even though retaining, as he probably

does, a good deal of muscular strength, he is liable on the slightest exertion, even that of rising in bed, to attacks of sudden syncope, which are attended with the utmost danger to life. During the later periods of the disease the appetite often fails; the patient suffers from looseness of bowels, the motions frequently being highly offensive, and containing more or less blood; he has disturbance of vision (hemeralopia, nyctalopia), singing in the ears, vertigo, want of sleep, and occasionally delirium. His intellect, however, remains for the most part unaffected. In many cases during the progress of the disease thoracic complications arise, especially effusion into the pleuræ, congestion of the lungs with extravasation of blood into their tissue, congestion of the bronchial tubes, cough, and sanguinolent expectoration, not unfrequently attended with a marked gangrenous odour. The duration of scurvy is uncertain, but it may extend over many weeks or even months. Death is usually due to sudden syncope or gradual asthenia, and may at any time be hastened by the occurrence of hemorrhage, ulceration, thoracic affections, or other complications. Recovery is generally rapid under suitable treatment. But the patient is liable to remain in enfeebled health, and ultimately perhaps to fall a victim to pulmonary phthisis or some other chronic visceral disorder.

Morbid anatomy.—The morbid anatomy of scurvy accords with the symptoms of the disease; there is tendency to rapid decomposition; extravasations of blood in various stages of transformation may be found, not only in the superficial regions already specified, but in the substance of the lungs, beneath the pleuræ, in the walls of the heart, in the sub-pericardial tissue, in the intestinal parietes, and beneath the peritoneal membrane. Sanguinolent serum also may be found in the various serous cavities. In other respects the condition of the viscera is very variable. The lungs, liver, and spleen may or may not be congested; the heart may be contracted and empty, or distended with black blood. The brain generally is healthy. The blood contains an excess of fibrine, but presents a diminution in the number of the red corpuscles, and an abnormally low specific gravity.

Treatment.—The only effectual treatment of scurvy is the restoration to the dietary of those articles of food to the want of which the disease has been traced—namely vegetables, and especially those, or those substances extracted from them, which contain citric acid and potash. Among the ordinary articles of diet which are efficacious in this respect must be enumerated potatoes, yams, onions, carrots, turnips, green vegetables of all kinds, inclusive of mustard and cress and scurvy grass; lemons, oranges, limes, grapes, and apples; and, among their derivatives, lemon- and lime-juice and sauerkraut. The provision enforced in emigrant ships, and which has been found effectual in preventing the occurrence of scurvy, is, that each person must have weekly at least eight ounces of preserved potatoes and three ounces of other preserved vegetables (carrots, onions, turnips, celery, or

mint), besides pickles, and three ounces of lime-juice. And among the suggestions issued by the Board of Trade to shipowners is the following:—namely, that each man should have at least two ounces of lime- or lemon-juice twice a week, to be increased to an ounce daily if any symptoms of scurvy manifest themselves. The importance of additionally supplying scorbutic patients with good nourishing diet, of taking precautions against sudden syncope, and of relieving by local applications the bleeding ulcerated gums, and ulcers which may exist in other parts, is of course obvious.

X. CHRONIC ALCOHOLIC POISONING. (*Alcoholism.*) DELIRIUM TREMENS.

Persons who are in the habit of drinking freely fall after a while into ill-health. They lose their appetite, suffer from nausea and sickness, have a furred tongue and offensive breath; the limbs become tremulous and enfeebled, the face dull and expressionless, the conjunctivæ congested and watery; an eruption of acne rosacea or acne tuberculata not uncommonly appears upon the nose and cheeks; they cannot sleep, become low-spirited and vacillating, and lose in some degree both memory and readiness or quickness of apprehension. They are apt to become, also, cowardly, cunning, and untruthful. Further results of drink are: cirrhosis of the liver, which may be followed by ascites, jaundice, or hæmatemesis; affections of the nervous centres, including delirium tremens, epilepsy, mania, dementia, and general paralysis; and probably also gout and its various consequences. Drinkers (especially, it is said, those who take beer) very often grow exceedingly fat; on the other hand they not unfrequently get much emaciated. Innumerable material lesions and functional disturbances are, and have been, rightly attributed to the abuse of alcohol; but there is no doubt that, in a very large proportion of cases, the mistake is made of attributing every ailment from which a drinker suffers to the influence of his drink, forgetful of the fact that habits of intemperance, long continued, expose their subject to many dangers, and to be attacked by many diseases, from which he would otherwise probably have escaped.

The parts which principally suffer are the alimentary canal, liver, and nervous centres; but it is to the affections of the last-named organs only that we now propose to direct attention.

Nervous Disorders. Delirium Tremens.

Causation.—Of affections of the nervous centres the most frequent, and on that account, if on no other, the most important, is that com-

monly known by the name of 'delirium tremens.' That delirium tremens, or as it is sometimes called delirium e potu, is a direct consequence of the abuse of alcohol is beyond dispute. But different views have been held in respect of the mode in which alcohol influences its production. It was long believed to occur only in persons who, after drinking heavily, were suddenly deprived of their accustomed stimulus. More recent enquiries, however, show that it is more commonly the immediate consequence of excessive drinking, and that it usually comes on in the course of long-continued intemperance or of those occasional outbreaks of intemperance (lasting it may be for a few weeks at a time), to which some persons are liable. It may no doubt supervene at the time when such persons are commencing to abstain; but not simply in consequence of their abstinence.

Symptoms.—The symptoms of delirium tremens creep on gradually. The patient loses his appetite, becomes restless and wakeful at night, his sleep being disturbed by frightful dreams; he grows suspicious, inclined to quarrel, agitated, restless, disposed to busy himself about various matters, and often (as Trousseau observes) to pack up his clothes and prepare for a journey. Generally by the time his disease has become fully established he has had no rest whatever for many nights, and has taken little or no solid food for many days.

The symptoms of the declared affection comprise delirium with hallucinations, and tremulousness of the muscles, together with various more or less characteristic disturbances of the other corporeal functions.

The face is either congested or pale. The pupils usually are dilated, the conjunctivæ injected, the skin bathed in more or less profuse sweat. The tongue varies in character, but in most cases is covered with a thick creamy fur. There is more or less thirst, but the appetite is in complete abeyance. Muscular tremors are almost invariably present; they may be general, or limited mainly to certain parts, such as the head and neck and upper extremities; and they manifest themselves especially when the patient exercises his muscles, but are not necessarily absent at other times:—thus, the arms tremble when he holds them out, the legs when he stands, the lips when he speaks, and the tongue when it is protruded. But, besides the ordinary tremblings, there are often constant fibrillar twitchings of the muscles, which scarcely reveal themselves by causing obvious movement, but may be distinctly felt when the patient's limbs are grasped; and there are often also (but more especially towards the later stage of the disease) involuntary startings of the limbs. The pulse varies: in most cases it does not, in the beginning, exceed the normal, and is then probably large, soft, and dicrotous; at a later period, however, and especially if the disease has taken an unfavourable turn, it increases in rapidity—rising it may be to 120 or 140, or more—and becomes at the same time small and extremely feeble. The tempera-

ture usually does not exceed 101°, and often never rises to that height; but occasionally it runs up more or less rapidly to 105°, or even 108° or 109°. There is no relation between the frequency of the pulse and the elevation of temperature. The mental phenomena are peculiar:—The patient's sleeplessness and tendency to dream are soon attended with hallucinations; he hears noises; he sees black spots, or sparks, or figures; he perceives flavours, or smells smells. His mind begins to wander; he looks suspicious or frightened; he searches behind the bed-curtains, under the bed, or in corners, to satisfy himself that there is nothing there; he becomes garrulous—talking for the most part of business and of projects which he has in hand, but interrupting himself from time to time under the influence of some passing dread, suspicion, or angry feeling. At this time he can be readily recalled to himself, and will answer questions rationally and coherently. The incoherence and delirium, however, soon increase upon him. He now probably is incessantly chattering, talking more or less incoherently of things absent and things present, but still with a marked tendency, as a rule, to dwell upon matters of business, to give orders to his servants or workpeople, to talk with customers. He suffers, also, from manifest illusions; he not only has singing and other noises in his ears, but he hears voices, and it may be enters into conversation with them; he not only sees muscæ, but he takes them (according to their characters) to be insects, or sparks, or coins, and he may be seen consequently endeavouring either to catch the animals which infest him, or to pick up the silver which is strewed around him; or he fancies that he sees larger objects, dogs or cats, strange persons or devils, and watches them as they slip behind some article of furniture, or peep at him from some obscure corner. In many cases his illusions are wholly of a nature to inspire horror or terror; policemen are after him for some murder he has committed; he is haunted by bad spirits; foul reptiles are crawling about him; great disasters threaten or have already involved his dearest friends. In some cases they are pleasing or funny; he is surrounded by beautiful scenery, he hears sweet music, he sees dancing girls or acrobats performing the most extraordinary feats. In some cases again he becomes wildly maniacal; in some sullen, morose, and stupid. He is apt also to mistake those about him for persons who are absent, or to confound them with the grotesque or horrible creations of his mind. His actions are no doubt in relation with the thoughts or fancies which are passing through his brain; he will often, as above pointed out, be seen busily picking up insects, flowers, or coins which are crawling or falling about him; or he will sit up and look suspiciously around; or he will endeavour to rise from his bed and will hunt everywhere for imaginary objects; or he will strive to avoid some danger or some foe, or will attack his attendant in the belief that he is contemplating or perpetrating some injury against him or his friends; or he will perform various grotesque acts, such as climb-

ing up the bed-post, standing on his head, or turning head over heels, or will applaud by shouts or laughter some imaginary performance. But in all cases, even though he has well-marked dominant illusions or frames of mind, there is a remarkable changeableness in his illusions and moods; he passes momentarily from one thing to another, and is suspicious, cowardly, violent, and merry in rapid succession; and in all cases, or nearly all, he can be recalled momentarily to himself, and restrained, by the voice of authority. Epileptiform attacks occasionally come on in the course of the disease.

In most cases, delirium tremens terminates favourably; and at the end of three or four days, or it may be a week, from the commencement of his malady, the patient falls into a gentle sleep and awakes refreshed and convalescent. But occasionally (and in those persons whose habits insure frequent recurrences, necessarily at length) the attack ends fatally by coma or asthenia. The circumstances which, according to M. Magnan, foretell a fatal issue, are elevation of temperature, persistent muscular agitation, and muscular debility or paresis. If the temperature rises to 102° or 103° (even though other symptoms appear favourable) there is ground for alarm; if, after continuing at this elevation for a day or two, it suddenly rises above 104°, the danger becomes very great and in some degree proportionate to the amount of the rise. As regards muscular tremors, it is not so much their intensity as their general prevalence and persistence which should excite alarm. They are especially of ill omen when they continue during sleep, and when to the general muscular vibration is superadded subsultus tendinum. Great rapidity and extreme feebleness of pulse, epileptic convulsions, coma, and the formation of bed-sores point also to a fatal termination.

The subject of delirium tremens must not be dismissed without drawing attention to the fact that in persons who are habitual drinkers, it not unfrequently happens that other illnesses (acute or chronic) which attack them become complicated with some of the symptoms of delirium tremens. Thus it is with serious accidents, pneumonia, and other inflammatory and febrile disorders; and thus, also, it not uncommonly is with hysteria. Nor must it be forgotten that delirium tremens is apt to be closely simulated by various affections, and more especially by meningitis and acute inflammations.

Other consequences of drink are epilepsy, insanity in its various forms, general paralysis, and dementia. These, however, are not special to alcoholism, and need not now detain us. Dr. Wilks has specially drawn attention to a form of incomplete paraplegia—attended sometimes with inco-ordination, sometimes with anæsthesia, and often with more or less pain in the limbs, and involving sometimes the legs only, sometimes the legs and arms—due to alcoholism, but immediately dependent, he thinks, on chronic spinal meningitis. It appears generally to be curable by abstinence.

Pathology and morbid anatomy.—Alcohol taken into the stomach is rapidly absorbed. It is eliminated, but apparently in very minute quantity, by the kidneys, lungs, and skin; yet it disappears quickly from the system. Generally, even if large quantities have been imbibed, none can be detected by chemical analysis after the third or fourth day; but Dr. Dupré believes that ten days may be taken as the period needed for its entire discharge. It is obvious, therefore, that the great bulk must undergo chemical decomposition in the interior of the body. Alcohol may be found post mortem in various organs; it has been discovered in the liver, but is much more readily detected in the brain, for which it seems to have a special affinity. The conditions of the stomach, intestines, and liver which supervene upon chronic alcoholic poisoning, are elsewhere described. The post mortem appearances presented by the central nervous organs are not very striking; in those who have been long given to drink, the brain is often found to be somewhat shrunken, the subarachnoid tissue opaque, and its proper fluid in excess; but in those who die of delirium tremens, there is usually more or less marked congestion of the cortex and medulla of the brain and of the upper part of the spinal cord. Moreover, there is not unfrequently discovered a deposit of refractive granules, and even of hæmatoidine crystals in the walls of the small vessels. There is no reason to believe that other tissues or organs suffer in any important degree. Dr. Dickinson has shown that there are no sufficient grounds for referring chronic renal disease to the effects of alcohol.

Treatment.—Our remarks under this head will have reference solely to delirium tremens. It is impossible to reconcile the different views that are held with respect to the treatment of this disorder. Formerly it was held, and by physicians of high eminence and large practical experience, that the one thing needful was to give the patient sleep. But now Drs. Laycock, Gairdner, Wilks, Anstie, and many others, urge that the disease is one of low mortality, which tends to get well of itself within a limited time, and that not only is opium not needed, but that its use is attended with no inconsiderable danger. The patient has been without food, or almost without food, for a considerable length of time; and they strongly urge that he should be fed with such nourishment as he can be made to take, and that it is by nourishment mainly that he is to be successfully treated. We do not deny that many of these cases do tend to recovery, and that feeding is an essential point in their treatment; but we cannot help thinking that more power for harm, and less power for good, than it deserves, have latterly been attributed to opium. We think, too, that opiates may be given with more benefit and less danger in inflammatory and other lesions of the brain than is generally suspected. A person suffering from delirium tremens should be separated from other patients; the room in which he is placed should be kept absolutely quiet, and the bright light of day excluded. Everything, indeed, around him

should tend to quietude and to solicit sleep. He should be constantly watched by a trustworthy and competent attendant. Under these circumstances it is not generally requisite to employ mechanical restraint; yet sometimes it becomes absolutely necessary to tie him down with a sheet or band, or to fasten his hands and feet to the bed with gauntlets. Nutriment should be administered with careful attention in small quantities and frequently. The most appropriate articles of food are milk, arrowroot, beef-tea, broths, and eggs. The bowels should be regulated. Those who consider sleep indispensable would now administer either chloral or some preparation of opium. The chloral is sometimes given with advantage in doses of from ten to twenty grains every half-hour until sleep is induced. Opium or morphia may also be given in comparatively small doses at short intervals. It is better, however, we believe, to administer it from the beginning in large doses, and to repeat it or not according to its effect; to give, for example, from half a grain to a grain of morphia, or from half a drachm to a drachm of laudanum at once, and to repeat the medicine in smaller doses at intervals of an hour or two, if sleep be not induced. So also with respect to chloral, we believe it better to give a large dose at once, say sixty or eighty grains, and to supplement this with subsequent smaller doses, if needful. If, as is doubtless best, the morphia be given by subcutaneous injection, the dose must be reduced to one-sixth or one-third of a grain. It may be well to add that patients with delirium tremens are difficult to bring under the influence of narcotics. Other remedies which have had, or have, strong advocates, are: digitalis in large doses (half an ounce to an ounce of the tincture); cayenne pepper; and bromide of potassium in doses of from ten to thirty grains. It is sometimes advisable to give the patient some of the alcoholic stimulus to which he has been addicted. When he is convalescent, quinine or other tonics are indicated, and he should, as far as possible, be debarred from drink. The probability, however, is that he will, so soon as opportunity offers, resume his evil habits.

XI. CHRONIC LEAD-POISONING. (*Plumbism.*) COLIC. DROPPED HAND.

Causation.—When lead, in even minute quantities, is habitually introduced into the system, characteristic and more or less serious consequences are pretty certain to ensue sooner or later. In most such cases the poisoning is very insidious, and not unfrequently its source is only discovered after patient research or by accident. Plumbism was formerly largely prevalent: in Poictou, in consequence of the habitual addition of lead to inferior qualities of wine; in the West

Indies, owing to the fact that leaden worms were used in the stills employed in the manufacture of rum; in Devonshire, as a result of the general employment of lead in the construction of the vessels used in making cider; and both in our own country and elsewhere, from the storage of drinking-water in leaden cisterns, or its conveyance through leaden pipes. In all these cases, the fluid, acting chemically upon the lead, and rendering it soluble, became in a greater or less degree impregnated with it. It is worth while to draw attention to the fact that rain-water and other soft waters become much more readily impregnated with lead than hard waters, provided these latter contain sulphate and carbonate of lime, and not too large a proportion of alkaline chlorides or nitrates. At the present day the contamination of drinking-water with lead is comparatively rare; and the chief source of lead-poisoning is the employment of this metal in manufacture and trade. To quote the words of Dr. Taylor, 'The carbonate' (to which salt chronic poisoning is usually attributable) 'finds its way into the system, among white-lead manufacturers, either through the skin or through the lungs, or both together; it is diffused through the air as a fine dust, and is not only respired, but taken into the mouth and swallowed with the saliva. It has been remarked that in factories where the powder was ground in a dry state, not only have the labourers suffered, but horses, dogs, and even rats have died from its effects. Since the practice has arisen of grinding the white lead in water, cases of colica pictonum have not been so numerous. They are still, however, not unfrequent among painters, plumbers, pewterers, the manufacturers of some kinds of glazed cards, the bleachers of Brussels lace, and among those engaged in the glazing of pottery, where oxide of lead is employed in the glaze.' 'The workers in metals—plumbers who handle metallic lead—are but little subject to the disease.' Amongst rare but well-ascertained causes of lead-poisoning are the employment of lead medicinally, its application to ulcerated surfaces (Althaus), the use of snuff impregnated with lead (Hassall and Garrod), and sleeping in a newly-painted-room. Some persons present the symptoms of plumbism who have been exposed in a very slight degree to the poison, who have taken, it may be, only a dose or two of lead medicinally; while others (painters for example) may go on with their work for twenty years or more, and yet escape.

Symptoms and progress.—Those who are under the influence of the chronic operation of lead often suffer more or less in their general health; their complexion is said to get sallow and earthy-looking, their skin dry and harsh; they become thirsty, lose appetite, and have a sweetish or metallic taste in the mouth. Dr. Garrod points out a remarkable connection between gout and plumbism, shown by the circumstance that a very large proportion (one-fourth) of his hospital gouty patients had suffered from lead-poisoning. And, indeed, whether that connection be accidental, or due to the fact that lead-poisoning

predisposes to gout, or that constitutions liable to gout are also peculiarly susceptible of the influence of lead, general experience seems to confirm the accuracy of Dr. Garrod's observation. Chronic albuminuria is also not unfrequently associated with lead-poisoning. A curious effect of lead was discovered some years ago by Dr. Burton, which is of great importance from a diagnostic point of view: namely the formation of a blue line along the edges of the gums immediately adjoining the teeth. This is situated in the substance of the gum, but appears to be largely determined by the amount of tartar present, and is supposed to be due to the precipitation of the lead, in the form of the sulphide, by the sulphuretted hydrogen emitted by the decomposing matters which are mingled with the tartar. This blue line is not, however, an infallible sign of lead-poisoning; for it occasionally gets developed in the course of a few hours after the use of two or three medicinal doses of lead; it is generally present in lead-workers who are free from all other symptoms; it often remains long after all possibility of poisoning has passed away; it is sometimes absent from cases of undoubted plumbism; and further, it may be caused by cuprons and other varieties of metallic impregnation. It is said that a similar blue line may be detected at the verge of the anus, and at the margins of ulcers. By far the most important consequences of lead-poisoning, however, are colic, and certain affections (mainly paralytic) of the nervous system. Of these colic is the more common, and, when the two conditions co-exist or alternate, is usually the earlier in making its appearan

A. *Lead colic* is characterised by the more or less gradual supervention of severe griping pains, attended with obstinate constipation and vomiting. The pains differ in no respect from those which follow upon impermeable intestinal stricture, and apparently are due to the same cause—namely, the powerful contraction, frequently repeated, of certain lengths of bowel above, in order to overcome some impediment to the passage of their contents into and through the length of bowel immediately following. They are referred mainly, as such pains usually are, to the umbilical region, come on at intervals with extreme severity, and, when the disease is fully established, are associated with more or less intense inter-paroxysmal uneasiness or pain. The pain is not generally aggravated by pressure, and indeed is often relieved both by that means and by friction. The paroxysms are attended with more or less obvious peristaltic movement of the bowels, and borborygmi. Vomiting may arise early from sympathy or late from the direct influence of obstruction. The abdominal walls are usually retracted, and the muscles hard and tense. Colic rarely proves fatal; but might readily become fatal if the cause to which it is referrible should continue in operation. Its duration varies; it may last for a day or two only, or be continued for a week, or, with remissions, for a still longer period. Moreover, when once there has been an attack

there is great liability to recurrence. It is rarely if ever attended with fever or intestinal inflammation.

B. *Nervous disorders. Dropped hand.*—Of nervous disorders, dropped hand, from paralysis of the extensors of the forearm, is by far the most frequent; but sometimes the paralysis is of much more general distribution. Dropped hand generally comes on consecutively to colic, sometimes gradually, sometimes more or less suddenly. In some cases one hand only is affected, and this is usually the right; but more frequently both hands are implicated, though in unequal degrees. The more obvious symptoms of the affection are loss of power over the extensor muscles of the forearm, in consequence of which the patient is unable to extend the hand upon the arm, or the first phalanges of the fingers upon the metacarpal bones, to adduct or abduct the hand, or to abduct the thumb. The hand consequently drops when the arm is held out prone, and both the hand and the first joints of the fingers are more or less powerfully flexed in consequence of the predominant action of the flexor muscles. The paralysed muscles waste rapidly, so that a distinct hollow is apt ere long to manifest itself between the bones at the back of the forearm; and moreover, while retaining their electro-sensibility, they lose more or less completely their Faradic contractility. The remaining muscles of the forearm, and even those of the upper arm, are apt to get enfeebled, though not otherwise affected. There is no impairment of cutaneous sensibility; but it not unfrequently happens that more or less severe pain and tenderness in the situation of the affected muscles attend the acute stage of the affection.

It is an important fact that the paralysis is limited, as a rule, to muscles supplied by the musculo-spiral nerve, and mainly to those supplied by its posterior interosseous branch. Those which usually suffer are the following, enumerated in the order in which (according to Duchenne) they are liable to be attacked:—extensor communis digitorum, extensor indicis, extensor minimi digiti, extensor secundi internodii pollicis, extensor carpi radialis brevior, extensor carpi radialis longior, extensor carpi ulnaris, extensor ossis metacarpi pollicis, and extensor primi internodii pollicis. Occasionally also the muscles of the ball of the thumb suffer. But the supinators, the muscles of the front of the forearm, and those of the hand (with the exceptions which have been pointed out), although they may get enfeebled, seem never to become distinctly paralysed, to lose their Faradic contractility, or to waste. It must be borne in mind, however, that the muscles are not necessarily involved in the order above named, and that they do not necessarily all suffer in every case. If the extensor communis be alone affected, the middle and ring fingers alone drop, the index and little finger retaining the power of extension, though somewhat enfeebled; if the extensores indicis and minimi digiti also suffer, all four fingers are implicated; if the radial and ulnar extensors of the carpus be paralysed, the wrist falls; if the radial or ulnar alone, then the

patient in endeavouring to extend the hand tilts it towards the opposite side; if the muscles of the thumb be implicated, the thumb lies in front of the hand in the position of opposition. The power of supination and that of pronation remain intact; and provided the first phalanges be supported in the extended posture, the second and third phalanges may always be voluntarily extended, a fact confirming the integrity of the interossei muscles.

In some instances the paralysis, loss of Faradic contractility, and wasting involve other muscles of the upper extremity besides those of the forearm. Those which are then chiefly liable to suffer are the deltoid and triceps. In some cases the paralysis is limited to the deltoid. Occasionally, again, lead-palsy involves the muscles of lower extremities, selecting especially the extensors of the foot upon the leg, and of the leg upon the thigh; or the intercostal muscles; or the diaphragm. And in some very rare cases, of which Duchenne quotes a striking example, paralysis attacks with more or less suddenness nearly all the voluntary muscles.

The duration of saturnine paralysis is very various; it may be weeks, months, or years. Moreover, the paralysis, like the colic, is apt to recur. The longer it has been in existence, the less, as a rule, is the prospect of ultimate recovery; and further, extreme wasting of the muscles, and persistent abeyance of Faradic contractility, are also of bad augury. Yet Duchenne draws attention to the interesting fact that in these cases voluntary power may occasionally be recovered, even though th muscles remain irresponsive to Faradic excitation.

Epileptic attacks sometimes come on in the course of lead-poisoning, and other cerebral phenomena, including coma.

Pathology and morbid anatomy.—After lead has been received into the organism, it is deposited in various parts, and discharged by various emunctories. It has been found post mortem in the spleen, liver, lungs, kidneys, heart and intestinal walls, and also in the substance of the brain and in the muscles. It passes off mainly with the urine; but, according to Dr. Pereira and others, there is some elimination by the skin; and Dr. Taylor states that it has been found in the milk. It is apt to remain in the tissues for some time, and has been detected in them by M. L. Orfila as long as eight months after its reception has been discontinued. The appearances found after death from chronic poisoning are for the most part very indecisive. After death from colic, or in cases in which colic has been present, the bowels (especially the large intestines) are said to be generally contracted and empty, or to present alternate contractions and dilatations, or intussusceptions; occasionally, also, spots of congestion have been seen in the mucous membrane. These are changes, however, which may be observed in many cases besides those of lead-poisoning. The paralysed muscles, as has been already stated, shrink rapidly and to an extreme degree; and post mortem are often observed to be remark-

ably pale and yellowish. But, on microscopic examination, their tissue is usually found to present a perfectly normal appearance. It is only after paralysis has existed for many years that degenerative changes get added to simple atrophy. The fibres sometimes become fatty. Whether the intestines are affected through the nervous system, or by the presence of lead in their walls, may be a subject of doubt. But, as regards the paralysis of the voluntary muscles, there is no doubt that Duchenne is right in regarding it as a consequence of nervous disorder. For if it were muscular, not only should we find the muscular fibres degenerated in proportion to their loss of power, but we should find Faradic contractility surviving as long as any healthy muscle was left. On the other hand, the rapid shrinking of the muscles, without degeneration, and their speedy loss of Faradic contractility, obviously point to lesion either of the nerve-trunks or of their nuclei of origin.

Treatment.—Whenever a case of lead-poisoning comes under treatment a careful enquiry should be made into the probable source of contamination, with the object of removing or counteracting it, or of putting the patient upon his guard. It is obvious that it would be well for patients whose occupation exposes them to the danger of continued lead-poisoning, to seek some other employment. But this they will rarely consent to; and, indeed, it is often impossible for them to do it. Apart from the question of the improvement of processes of manufacture in order to minimise the risks of those employed (a subject upon which we do not presume to enter) it may be mentioned: that extreme personal cleanliness is important for all those who are exposed to danger; and that there are good grounds for believing that the habitual use of lemonade made with sulphuric acid is to a considerable extent protective, by converting the carbonate or other salts of lead in the stomach into the insoluble and inert sulphate.

Various methods of treatment have been suggested with the object of removing lead from the system—the more important of them being the employment of baths containing some soluble sulphide, and the internal use of iodide of potassium. Dr. Pereira recommends baths medicated with sulphide of potassium, in the proportion of two ounces to fifteen gallons, in the belief that the lead escaping from the surface of the skin would thereby be converted into the insoluble sulphide. This result does in fact happen; but there is no reason whatever for suspecting that the baths promote the escape of lead in any important degree. M. Melsens suggested the employment of iodide of potassium, on the ground that the iodide makes, with the insoluble salts of lead deposited in the tissues, a soluble double salt capable of removal by the kidneys. This practice is commonly followed.

In the treatment of lead-colic it is best, we believe, to relieve pain and discomfort by opiates and fomentations, and to leave the bowels to act of themselves, as they will usually do at the end of

a few days. If it be thought right to remove the contents of the lower bowel, this may be effected by means of copious enemata of warm water or warm gruel. Many, however, prefer the course, which Sir Thomas Watson advocates: namely, the exhibition of a full dose of calomel and opium—ten grains of the former with two of the latter—which he says usually soothes the vomiting, restlessness, and pain, and may be followed up successfully by a dose of neutral salts or castor oil. Alum, in doses of a scruple or half a drachm three times a day, has been highly recommended.

For the restoration of the paralysed and wasting muscles, galvanism is the only effectual remedy. Faradisation is employed by M. Duchenne, who recommends that a powerful current should be used three times a week for as long a period as may be necessary—it may be as much as two or three months. Each sitting may last for ten or fifteen minutes. He recommends also, that each muscle should be separately galvanised. The slowly interrupted constant current, similarly employed, is even more efficacious.

XII. CHRONIC MERCURIAL POISONING. (*Mercurialism.*)

Causation.— Chronic mercurial poisoning may result from the long-continued medicinal use of any of the preparations of mercury; but it is most frequently due to habitual exposure to the vapour or dust of mercury, or its salts, which certain manufactures or trades involve. Those, therefore, who chiefly suffer are the workmen engaged in quicksilver mines; water-gilders; the manufacturers of looking-glasses, barometers, and thermometers; furriers, and those engaged in the packing of furs which have been brushed over with solution of nitrate of mercury.

Symptoms and progress.—The symptoms of chronic mercurial poisoning have reference mainly to the nervous and muscular systems, and are commonly included under the term 'metallic tremor.' The first indications of this condition are: a general tremulousness of the hands and arms, coming on for the most part gradually; slight numbness or tingling in the hands or feet; and occasional pains in certain joints, more especially those of the thumbs, elbows, feet, and knees. Tremors are common amongst workpeople exposed to the vapour of mercury, and may continue for years without materially interfering with their capacity for work or their general health. But sooner or later they tend to get aggravated; they not only become more violent, but gradually extend to all parts of the muscular system; so that they involve at length the hands, arms, and legs, the head and neck, including the muscles of expression, speech, and deglutition, and the

trunk with the muscles of respiration. Then the violent trembling of the hands and arms renders the patient more or less incapable of using them for any purpose, especially for delicate operations—he probably cannot lift a glass of water to his lips, or feed or dress himself; the agitation of his legs gives to his attempts to walk or stand a peculiar jerkiness or choreic character, and, indeed, before long he is probably unable to stand or walk without support; the convulsive action of the muscles of his head and neck causes constant tremulous movements of these parts, while that of the muscles of expression reveals itself in grimaces, and that of the lips, tongue and muscles of mastication causes tremulous, indistinct and divided utterance, and difficulty of mastication; the involvement of the respiratory muscles induces more or less difficulty of breathing. All these convulsive movements are usually in abeyance when the patient is lying down and making no muscular effort; but they reveal themselves whenever he attempts voluntary movement, and become especially aggravated whenever he is under observation. Further, the patient is liable to occasional, apparently causeless, exacerbations of more or less severity. At a very advanced period of the disease, the convulsions do not wholly cease when the patient is in bed; and then occasionally continue also during sleep. Moreover, they are now not unfrequently associated with sharp pains in the limbs, and occasional attacks of tonic contraction. It would seem, and the point is an important one, that the muscles of the eyeballs do not share in the convulsive movements, and that there is an absence of nystagmus. There is no real loss of sensation.

The symptoms above enumerated are not necessarily associated with any other indications of mercurial poisoning or signs of ill-health. But in a considerable number of cases the patients either have previously suffered from salivation and ulceration of the gums, fetid breath, nausea, sickness, colicky pains, disturbance of the bowels, and fever; or present these phenomena in a more or less marked form at the time when the nervous symptoms supervene; or begin to suffer from them during the course of the tremors, even if they have never thus suffered previously. And generally, after the tremors have attained a high degree of severity, cachectic symptoms come on; the patient gets sallow, emaciated, and weak; he loses his appetite; and there is more or less general failure of his circulatory and other functions. Sometimes, also, various cerebral complications are developed, such as vertigo, headache, loss of memory, delirium, epilepsy, paralysis, or coma.

Chronic mercurialism is not generally a fatal or even dangerous disease; mainly, however, for the reason that those who are affected with it are usually compelled to give up their employment, and thus escape further risk. But for such as continue to expose themselves to the vapour of mercury, the prospect of early death is by no means uncertain—death under such circumstances being caused, either by

extreme debility, or by some of the ordinary effects of mercury on the gums and mouth, or intestinal canal, or by some cerebral complication, or by the supervention of intercurrent disorders.

The affections with which mercurial tremors are most likely to be confounded are disseminated sclerosis, and paralysis agitans. But careful attention to the history and details of symptoms will generally enable an accurate differential diagnosis to be made between them. It may, however, be worth while to point out, as practical hints, that paralysis agitans does not commonly affect the muscles of the head and neck, but imparts to the patient a tendency to run forwards, and that generally in disseminated sclerosis there is well-marked nystagmus.

Morbid anatomy.—No characteristic lesions have been discovered in the internal organs of patients who have died of chronic mercurialism. But mercury has been detected chemically in various parts of the body, more especially the brain, liver, and kidneys.

Treatment. — The preventive treatment of mercurial poisoning includes the taking of measures to guard against the entry of mercury into the system, either by adopting such modifications of the processes of manufacture as minimise the diffusion of the poison through the atmosphere, or by compelling the workers to wear respirators or other protective coverings to the face, to wash their hands before eating, and to change their clothes and wash after leaving work. The direct treatment of chronic mercurialism by drugs is of little use. It may on the whole be judicious to act on the bowels, kidneys, and skin, for the purpose of promoting the discharge of the poison. It may even be well to adopt the treatment already suggested for chronic lead-poisoning, namely, the administration of iodide of potassium, with the object of combining the mercury in the system therewith into a soluble double salt. But the essential part of the treatment, and that which is alone of real efficacy, is the removal of the patient from the influence of mercury. Excepting in cases of extreme severity or long duration, restoration to health is thus effected in the course of a few weeks or months. Tonics may often be given with advantage to the patient's general health; and galvanism may be applied with benefit to the enfeebled muscles.

CHAP. V.—DISEASES OF THE DIGESTIVE ORGANS.

SECTION I.—DISEASES OF THE MOUTH, FAUCES, AND ADJACENT PARTS.

I. CATARRH.

Causation.—The most common, and on the whole perhaps the most important, variety of inflammation affecting the mouth, fauces, and parts in relation with them is that which results from exposure to cold, and gives rise to the phenomena which collectively constitute what is commonly known as a 'cold' or catarrh.

Morbid anatomy.—Catarrhal inflammation commences with hyperæmia, infiltration, and tumefaction of the affected mucous tissue, diminution of the secretions from its surface, and from the glands which open upon it, and consequent abnormal dryness. Before long, however, the inflamed parts begin to pour out a thin, watery, somewhat acrid discharge, in considerable abundance; and at the same time the tumefaction usually undergoes some diminution. Subsequently the secretion gets thick, opaque, and yellowish or greenish, and assumes the characters of muco-pus or pus. This change generally indicates the commencement of the end; for now, if nothing occurs to interfere with the normal progress of the case, the tumefaction and secretion both gradually subside, and the mucous membrane returns to its healthy state. Catarrhal inflammation does not, as a rule, seize at once on any extensive tract, but rather, like erysipelas, begins in a comparatively small area, whence it spreads. Nor does it, even in the case of anyone who is liable to it, always commence in the same spot. Thus it often, perhaps most frequently, begins in the mucous membrane of the nose, whence it spreads by continuity to the fauces, and thence to the larynx and probably to the trachea and bronchial tubes; or it first manifests itself in the larynx, whence it extends upwards into the nose, and downwards into the chest; or it first attacks the fauces, soft palate, or it may be the gums. The regions which are liable to be involved in the course of catarrh, and in any one of which probably it may commence—thence extending to the others—are the following:—the nose and sinuses in relation with it, with the lachrymal ducts and conjunctivæ; the fauces and pharynx, together with the

Eustachian tubes and tympanic cavities, and the œsophagus; the oral cavity, including the palate, gums, sockets of the teeth, and tongue; the periosteum of the facial bones, and branches of the fifth pair; and, lastly, the larynx and subordinate respiratory passages.

Symptoms and progress.—The symptoms of a cold necessarily differ according to the regions which mainly suffer. The special symptoms, however, are always associated with the ordinary phenomena of febrile disturbance. The latter vary in severity, but are generally mild—sometimes scarcely noticeable—and always most severe during the first day or two of the attack. They comprise elevation of temperature, heat and dryness of skin alternating with perspirations which come on mainly at night-time, increased frequency of pulse, thirst, constipation, scanty urine with abundant uratic deposit, muscular pains, and frequently drowsiness. The febrile symptoms are sometimes alarming in the case of young children.

The symptoms of catarrh affecting the cavity of the nose are in the first instance dryness, obstruction and irritability of the nasal passages, associated with frequent paroxysms of sneezing, the performance of respiration mainly through the open mouth, and inability to pronounce the nasal consonants, *m*, *n*, and *ng*. To these succeeds more or less copious defluxion of thin watery mucus, which frets the margins of the nostrils and the upper lip. There is probably still great irritability of the mucous surface, with paroxysmal sneezing; but with the continuance of the discharge the nasal passages become more pervious, and the symptoms due to obstruction to some extent subside. Finally, the discharge gets thick, and at the same time less abundant, the tumefaction and irritability of the mucous membrane diminish, and convalescence ensues. Associated with nasal catarrh there is always more or less complete loss of smell, especially during the earlier stages; and, probably owing to implication of the frontal sinuses, there is often severe headache, limited to the situation which the sinuses occupy, and not unfrequently attended with drowsiness. The extension of inflammation to the conjunctivæ is shown partly by obstruction of the lachrymal ducts, in consequence of which the tears are compelled to flow over the face, partly by the supervention of ophthalmia.

The indications of catarrhal inflammation of the fauces are, unnatural redness of the soft palate and pillars of the fauces, and, in a greater or less degree, of the contiguous mucous surfaces; together with tumefaction of the same parts, but more particularly perhaps of the lax tissue of the uvula, which is apt to become œdematous and enlarged in all its dimensions. The first symptoms of which the patient complains are dryness, stiffness, and itching or tingling, commencing at one side, or in some defined area, but soon becoming more or less general throughout the fauces and soft palate, and frequent tendency to swallow in order to relieve the uncomfortable feeling in the throat, to clear the throat, and to cough a slight hacking cough. The act of

deglutition is more or less painful. With the supervention of the stage of secretion, the efforts to swallow and clear the throat get more effective and less painful, the patient becomes comparatively comfortable, and convalescence soon follows. Involvement of the Eustachian tube and ear is indicated, first by itching or shooting pains in the course of the tube and in the ear, then by deafness, and the usual signs of aural inflammation. Extension of catarrh along the œsophagus to the stomach is rarely if ever manifested by prominent symptoms. Those usually observable are a sensation of warmth along the œsophagus and in the stomach, and slight dyspeptic symptoms, more especially frequent eructations and craving for food.

Catarrhal inflammation of the mouth more frequently and seriously affects those who suffer from bad teeth than those whose teeth are sound, and reveals itself mainly by pain, tenderness, and swelling of the gums, and particularly of the periosteum of the sockets of the teeth. The teeth consequently become loose and tender; and neuralgic pains, often most severe at night-time, flicker about the gums, and sometimes extend to the periosteum of the jaws, and along the superficial branches of the fifth pair.

Catarrhal inflammation of the larynx is elsewhere described under the name of laryngitis, and that of the bronchial tubes under the name of bronchitis.

It remains to say that catarrh, in the sense in which the word is employed in the present article, is an affection of very various importance. In the majority of cases it must be regarded as a trivial disorder, which reaches its full development in the course of a day or two, and lasts at the outside not more than a week or ten days. Yet, without attaining any special severity, it may be kept up for an almost indefinite period if the patient continue to expose himself to its exciting cause. Nor can it be regarded as entirely devoid of danger, especially if it involve the larynx or bronchial tubes; for, although in many cases the laryngeal or bronchial affection is really slight, it differs only in degree from the severest forms of primary laryngitis or bronchitis, and may readily pass into one or other of them. Further, although the pain and discomfort of catarrh are commonly neither severe nor of long duration, there are exceptions to both of these rules. The chief exceptions are furnished by those cases in which the inflammation spreads to the teeth, periosteum, and branches of the fifth pair, and those in which it attacks the ear—in both of which cases the pain is often intense, and continues maybe, with little intermission, for weeks or months.

Treatment.—Trivial as a common cold may seem to be, it is yet of such frequent occurrence, and a source of so much discomfort, especially to those who are liable to its attacks, that its treatment cannot be regarded as unimportant. As a general rule patients suffering from cold should confine themselves to a warm and well-ventilated but

not draughty room; and should, if not in bed, be warmly clad. A hot bath—water, vapour or air—should be taken before going to bed, together with some warm drink, and a little Dover's powder—measures which are serviceable in relieving pain and discomfort, in promoting sleep, and in exciting perspiration. During the day the occasional inhalation of steam is often very useful, as also are frequently repeated small doses of ipecacuanha and opium, either in the form of Dover's powder, or associated with some febrifuge mixture, or with ether or ammonia. Sir T. Watson notices with especial approval the treatment of a commencing catarrh with (in the adult) about twenty minims of laudanum at one dose, or with about half that quantity of laudanum combined with seven or eight minims of vinum antimoniale, repeated every third or fourth hour for three or four times; as also Sir Henry Halford's practice (which accords pretty nearly with the usual domestic routine) of giving at bed-time a beaker of hot wine negus with a tablespoonful of the syrup of poppies. He also observes that there is 'a period in catarrh which has gone on unchecked when you may accelerate its departure by a good dinner and an extra glass or two of wine.' Counter-irritation is sometimes serviceable; and if the fauces or larynx be dry and uncomfortable, the frequent sipping of warm milk, barley-water, gruel, or 'treacle posset,' or the use of black-currant jelly, or such-like things, is often a source of considerable comfort. Sucking ice in many cases answers the same purpose. In the latter stages of faucial catarrh, or when the affection has become chronic, astringent applications, either in the form of gargles, or of spray by means of the atomiser, may be useful. Occasionally, but for the most part as the result of repeated catarrhal attacks, the uvula gets elongated, and is believed to irritate the larynx, with which it comes in contact. Under such circumstances the tip may be readily and safely snipped off with scissors.

It is very desirable to obviate, if possible, the liability to catarrh which so many persons labour under. There is no doubt that active exercise in the open air, and all other habits which tend to promote good health, tend also to diminish this liability; and many a person will in his autumnal holiday expose himself with impunity to conditions which at home would certainly have brought on a severe attack. So far as possible, therefore, exercise and other health-conducive practices should be enjoined. It is not, however, the exposure which attends active exercise that as a rule induces cold, unless, indeed, the patient has undergone great fatigue and consequently fails to keep himself warm; but it is rather the exposure when one is still, especially when one is still after previous violent exertion, and exposed to a cool breeze, to a cool draught of air, or to the coldness induced by wet clothes or the evaporation of sensible sweat. The means of obviating such dangers are too obvious to need enumeration. It is generally held, and we believe with reason, that a matutinal cold bath followed by

friction with a rough towel, and then by walking or other exercise, is a good preventive of colds. The shower-bath has been especially recommended for this purpose. It is, nevertheless, a fact that the continued use of the shower-bath will in some persons, so far from obviating the liability to cold, induce it, and keep up a permanent catarrhal state.

II. THRUSH. (*Aphthæ.*)

Causation and morbid anatomy.—Inflammatory affections of the mouth and fauces frequently arise in connection with stomach and bowel disturbance—sometimes simultaneously with it, sometimes secondarily to it, and more rarely, perhaps, as the first step in the order of events. Such inflammations are sometimes catarrhal in the anatomical sense of the word, and hence not readily distinguishable in all cases from the effects of ordinary cold. They do not, however, so far as we know, tend, as the latter variety does, to involve the nasal cavity and air-passages, or to extend to the eye, ear, sockets of the teeth, or branches of the fifth pair; while, on the other hand, they involve the mucous membrane of the mouth much more prominently.

Thrush is characterised for the most part by the appearance in greater or less abundance, on the tongue, gums, and palate, inside the lips and cheeks, on the soft palate, and pillars of the fauces, and even on the surface of the pharynx, of small, elevated, opaque, whitish spots, which are round or irregular in form, pretty firmly adherent, and not unfrequently appear like attached flakes of curdled milk. These can be easily separated, leaving more or less distinctly excoriated areæ or ulcers behind; and appear to be due mainly to inflammatory overgrowth of the epithelium with tendency to its detachment.

In some instances thrush presents a different character. It begins with the formation of minute white rounded elevations, which gradually increase in size until individually they attain perhaps the bulk of a mustard-seed or tare. These are hemispherical in form, adherent by broad bases, smooth on the surface, and uniformly solid. They are scattered irregularly, sometimes sparsely sometimes in great abundance, on the lips, and other parts of the surface of the oral cavity and pharynx. Under the microscope these bodies are found to consist of the oval spores of a cryptogamic plant, called the 'oidium albicans,' the mycelium of which infiltrates the subjacent epithelium. The source of this parasite has not been clearly ascertained, but Hallier regards it as identical with the oidium lactis. If this be so the explanation of its occurrence in infants at the breast, and in persons wasted with disease, is not far to seek.

It must be added that the oidium may often be recognised in the

form of aphthæ first described. It seems therefore probable, not only that it may be derived from milk; but that it attacks the mucous membrane under various circumstances, sometimes directly, sometimes at the seat of excoriations or of inflammatory patches—in other words that aphthæ are sometimes primarily, sometimes secondarily, parasitic.

Symptoms and progress.—Aphthæ are of very common occurrence in young children, more particularly infants at the breast; but are frequent also in the course of many diseases, especially when they are attended with hectic fever or the typhoid state. In young children thrush is generally preceded by and attended with feverish symptoms —heat of skin, fretfulness, and drowsiness—diarrhœa, or other morbid conditions of the bowels, loss of appetite, vomiting, and unwillingness to take food. Gastro-intestinal disturbance, indeed, is rarely absent; and it is believed by many that aphthæ of the mouth indicate a similar condition of the stomach and alimentary canal. The lips usually are dry, and the tongue, especially at the tip and edges, redder and drier than natural, and with a tendency to get furred on the dorsum and towards the base. The anus and its vicinity in such cases are sometimes reddened and excoriated, and aphthæ have been described as existing there. Aphthæ may subside after a few days, or last continuously or with remissions for many weeks. Thrush is not in itself a dangerous affection or necessarily an indication of danger in the affection which it attends. It must not be forgotten, however, that it frequently accompanies gastro-intestinal lesions which prove fatal, and that its presence cannot but add something to the danger of an already dangerous disease. When thrush supervenes in the course of diseases affecting adults, although it is not necessarily an indication of impending death, it is yet often a symptom of grave omen.

A trivial form of the affection is observed in persons who are liable to dyspepsia. The dyspeptic symptoms, which are probably inflammatory, are attended with stiffness and soreness of the back of the tongue and fauces, and sometimes of the anterior part of the tongue as well. There may be considerable pain on deglutition; and acid or stimulating articles of diet, and such as are in hard and angular fragments, cause intolerable smarting. On inspection in some cases, little or no visible departure from the healthy condition can be observed; in other cases, however, there is more or less obvious redness; and often the presence of cracks or fissures, or even of small patches of excoriation along the edges of the tongue, and elsewhere at the back of the mouth, may be recognised.

Treatment.—In the treatment of thrush and of the forms of inflammation related to it, it is important in the first place to attend to the general health and especially to the condition of the alimentary canal. In children it is generally best to commence the treatment with a dose of castor oil, or of rhubarb in combination with carbonate of magnesia or grey powder, and then to administer medicines calculated

to improve the tone of the stomach and bowels. According to the particular symptoms present may be prescribed limewater with milk, small doses of rhubarb with ginger or some other aromatic, aromatic confection with chalk and opium, or vegetable bitters. Locally, relief may be given by the application of mel boracis, solution of tannin, sulphate of zinc, or nitrate of silver; by washing out the mouth with a solution of chlorate of potash; by rinsing it with mucilaginous fluids; or by the use of lozenges containing gelatine or mucilage. With the object of destroying the parasite present in some forms of aphthæ, solution of sulphurous acid has been strongly recommended.

III. ULCERATIVE STOMATITIS.

Causation and morbid anatomy.—A peculiar affection of the mucous surface of the cavity of the mouth is sometimes met with, chiefly if not entirely in children below the age of puberty, which has a close relation, at all events anatomically, to that observed in cattle affected with the foot-and-mouth disease. It is impossible to deny that there is also some resemblance between this affection and both thrush and the early stage of gangrenous ulceration. Yet the appearances are so peculiar, and the whole progress of the affection so like that of a specific disease, that there is good reason to regard it as an affection *sui generis*. It consists in the formation of excoriated patches, chiefly limited to the surface of the gums, and corresponding parts of the cheeks, but occurring also on the dorsum and sides of the tongue, mainly towards the base, on the palate, and general surface of the buccal mucous membrane. The excoriations vary in size and shape, but are mostly irregular and tending to run together; their surface is raw, red, and weeping, sometimes bleeding; and the surface of the mucous membrane between them is thickened and opaque. The tongue (excepting the spots of excoriation) generally gets covered with a thick, tough, opaque, whitish fur, and its surface looks not unlike a piece of wash-leather.

Symptoms and progress.—The approach of the malady is usually indicated by some degree of feverishness and malaise—symptoms, indeed, differing little if at all from those that usher in an ordinary cold. Then, after a day or two, some soreness is experienced in masticating, speaking, and deglutition; and if the mouth be examined, the morbid phenomena above described will be recognised in an early stage. The progress of the affection is attended with febrile symptoms—heat of skin, flushing of face, listlessness, drowsiness, thirst, loss of appetite, and the like. And these, together with the local phenomena, usually subside in the course of a week or ten days. In some cases the affection

of the mouth assumes a more chronic character. We are not aware that it ever leads to serious consequences.

Treatment.—For local treatment mel boracis, or chlorate of potash in solution, seems to be indicated. Internally, a little chlorate of potash or other febrifuge medicine may be administered.

IV. NOMA. (*Gangrenous Stomatitis.*) GANGRENE OF FAUCES.

A. *Noma.*

Causation.—Gangrenous ulceration of the mouth occurs almost exclusively in children under twelve years old, and indeed is mainly limited to those whose ages lie between one and five. Its cause is not very obvious. There is no doubt, however, that it is especially apt to become developed during convalescence from acute febrile disorders, among which measles stands pre-eminent, and in children who have been badly fed or are anæmic.

Morbid anatomy.—The gangrene may commence at any part of the buccal surface, and in several parts at one time. But it usually originates in the sulci between the gums and cheeks, and chiefly (according to Barthez and Rilliet) in those connected with the lower jaw. It begins variously: sometimes with ulceration or the formation of a superficial slough; sometimes with congestion, thickening and tension of the substance of the cheek or other soft parts circumscribing the oral cavity. In any case there soon appears on some part of the mucous surface of the mouth an irregular greyish or black sloughy patch surrounded with a rim of intense and somewhat livid congestion. This tends to spread rapidly both in area and in depth—its extension being preceded and accompanied by infiltration, hardening, and congestion of the tissues. The cheek in the affected neighbourhood frequently becomes tense, shining and livid. With the extension of the gangrene, the gums may be eaten away, the alveoli necrosed, and, if the patient live sufficiently long, the teeth and portions of the jaw may exfoliate, and the soft palate, fauces, and tongue, each and all, be more or less extensively destroyed. Very frequently the cheek gets perforated, and the destructive process may then spread almost indefinitely, involving in turn the mouth, the entire cheek, and it may be the nose, the eye, and other contiguous parts.

Symptoms and progress.—The symptoms which attend noma are, at all events in many cases, much less severe than one would expect them to be. It often happens that the gangrene has made some progress in the interior of the mouth before anything has occurred to call special attention to what is going on there; and indeed it is not a rare thing to find patients in whom gangrene has committed the most extensive

and frightful ravages, and for whom recovery is hopeless, who neither suffer pain nor have suffered it, who maintain a good appetite, and continue sensible and even cheerful. The special symptoms, in addition to swelling of the cheek and the actual progress of the gangrene (which is obvious enough if looked for), are: more or less profuse salivation, the discharge often being bloody and foul; extreme fetor of this discharge and of the breath; and more or less swelling of the neighbouring lymphatic glands. As above indicated, the patient often suffers very little pain or uneasiness, remains sensible, talks, and takes an interest in whatever is going on about him, and retains his desire for food and the power of taking it. But notwithstanding this, the pulse rises in frequency and gets small and feeble; the surface grows pale and cold; drowsiness or delirium comes on; diarrhœa perhaps sets in; and death from asthenia supervenes at the end of a few days. In a small proportion of cases recovery takes place, with more or less deformity.

B. *Gangrene of Fauces.*

Causation.—But gangrene, not specially limited in this case to young children, may commence in the fauces or pharynx. In some cases this is due to diphtheria or scarlet fever, or results from the mere intensity of the inflammation in ordinary tonsillitis. But it may also occur independently of such special diseases, and, like noma, be traceable to profound impairment of the general health.

Symptoms and progress.—The symptoms in these several cases differ in some degree according to the nature of the disease to which the gangrene is due. Eliminating, however, the symptoms referrible to the several specific affections which have been named, gangrene of the fauces would be revealed by tumefaction of the tissues, the appearance of sloughs upon the surface, fetid discharge and breath, swelling of the glands beneath and behind the jaw, and, in addition to these phenomena, difficulty and pain in deglutition, and probably, before long, more or less difficulty of respiration. The situation of the morbid process necessitates the presence of much more pain and discomfort than are usually associated with noma; and here, as in the other case, very extensive destruction of tissue may take place, and perforation ensue. The general symptoms are: feebleness of pulse, sometimes with quickening, sometimes with marked diminution of frequency; pallor; coldness of surface; tendency to collapse; and not unfrequently, before death, copious perspirations, diarrhœa, and impairment of consciousness, delirium or coma.

C. *Treatment.*

In treating gangrenous affections of the mouth and throat, it is in the first place of paramount importance that the patient's strength should be maintained by the regulated administration of nutritious

food and alcohol, and of tonic medicines, or these combined with diffusible stimulants. Opium here, as in all similar cases, may be of great service. For local treatment, it is necessary to keep the parts cleansed; to wash them frequently with antiseptic fluids, such as solutions of either chlorinated soda, chlorine, hydrochloric acid, permanganate of potash, or chlorate of potash; and to treat the gangrenous tracts themselves freely with escharotics, of which probably the most valuable are pure hydrochloric or nitric acid, and the actual cautery.

V. INFLAMMATION OF THE GUMS IN DENTITION.

Cutting the teeth is always attended with more or less discomfort, if not absolute pain. Previous to the actual eruption, the implicated gum generally becomes congested, swollen and tense, and often distinctly inflamed. Occasionally suppuration or ulceration takes place. The eruption of the second teeth is seldom attended with symptoms which call for the notice of the physician. The eruption of the first set, however, is a fertile source of infantile ailments. This is especially the case when it occurs early. It is well known to mothers and nurses that infants who are on the eve of cutting their teeth begin to dribble and to bite the finger or any other hard substance which may be introduced into the mouth; and, looking upon these symptoms as an indication for treatment, they give the babe an ivory or india-rubber ring or a piece of coral to bite.

So far the symptoms may be regarded as normal; but in many cases the congestion of the gum produces feverishness and fretfulness, interferes with the infant's rest, and induces sickness and diarrhœa. When these phenomena ensue, each may be treated according to its importance: the vomiting may be allayed by the exhibition of some aromatic, or the addition of a small quantity of lime-water to the milk; the diarrhœa may be rectified by the administration of a little castor oil or Gregory's powder, followed, if necessary, by a little aromatic confection and chalk; the restlessness may be met by minute doses of opium. In the great majority of cases, however, the most efficacious and the best treatment is freely to lance the inflamed gum.

In some instances convulsions are referrible to the irritation of the emerging teeth. Under such circumstances, in addition to the appropriate treatment for convulsions, lancing of the gums must be efficiently performed.

Many other maladies besides the above are commonly regarded as consequences of dentition, the principal of them being eczema, lichen, and impetigo in various forms, bronchitic affections, and paralysis. It is doubtful, however, whether dentition has any other effect upon them than that of aggravating them.

VI. GLOSSITIS.

Causation.—Besides the superficial forms of inflammation in which the tongue shares with the other parts bounding the oral cavity, the organ is liable to become inflamed throughout its whole substance. This occurrence, which is rare, may take place under the influence of the mercurial poison, or as a consequence of direct injury, but now and then arises independently of all such obvious causes.

Symptoms and progress.—Idiopathic glossitis is said to be preceded in some cases by premonitory febrile symptoms. In other cases the inflammation is certainly, so far as one can judge, primary, although attended probably from the commencement with more or less febrile disturbance, and even with rigors. It sometimes commences in the tongue itself, at other times in neighbouring parts, especially the fauces. The tongue then becomes swollen, stiff, and painful, and incapable of executing its proper functions. The swelling is usually general, although sometimes limited to one half, or it may be some lesser portion. In the first case the organ gets enlarged in all its dimensions, sometimes so thick as to render inspection of the back of the mouth out of the question, so wide as to project on either side between the molar teeth, so long as to protrude beyond the lips, and even exerting serious pressure upon the upper part of the larynx. The pain is usually of a throbbing or burning character, and increased by all attempts at movement, so that mastication, deglutition, and articulation are in some cases almost impossible. Saliva accumulates in the mouth, and the patient's sufferings consequently become much aggravated. The surface of the tongue may in the first instance be redder than natural, but very soon gets enveloped in a thick white creamy fur. Occasionally suppuration takes place and an abscess forms. The affection usually attains its height in the course of three or four days, and, if free from complication, subsides in the course of a week or ten days. Permanent hypertrophy of the tongue has sometimes resulted.

The sufferings of a patient with glossitis are usually out of proportion to his danger. Some of them have already been referred to; but one of the most serious is the sense of impending suffocation which is often present, and which alone may be sufficient to prevent all sleep and forbid even temporary ease. It is quite possible, however, that from extension of œdema or inflammation to the larynx dangerous symptoms, and even death, may ensue. The disease, therefore, is one which needs close and careful watching.

Treatment.—The patient should have his mouth cleansed, by gargling (if he can effect it) or otherwise, with solution of chlorate of potash or other detergent lotions; his strength should be sustained with liquid nourishment, which, if it cannot be swallowed, should be

administered by the nose or rectum. Fomentations may be applied to the throat externally, and even leeches may be deemed advisable. It may also be necessary (and the practice is very efficacious) either to apply leeches to the tongue itself, or to make longitudinal incisions into it. If an abscess form, it should, of course, be opened. For general treatment, febrifuges may be given; and opium is of paramount value. It requires, however, to be given with much caution. If suffocation threaten, tracheotomy may be performed.

VII. QUINSY. (*Tonsillitis.*)

A. *Acute Tonsillitis.*

Causation.—The surface of the tonsil becomes inflamed in a greater or less degree whenever spreading or general inflammation involves the mucous membrane of the mouth and fauces. Hence, in catarrh, aphthæ, and the like, the tonsils are necessarily implicated. Again there are several affections in which inflammatory involvement of the substance of the tonsils forms an important and characteristic feature. We especially refer to scarlet fever and diphtheria. Deep-seated or parenchymatous inflammation of the tonsils, however, is like ordinary catarrh a frequent consequence of exposure to cold or wet; the two conditions, indeed, are not unapt to concur. Nevertheless it is a fact that many persons who are subject to catarrh, with all its usual associations, never suffer by any chance from tonsillitis; and it is equally a fact that tonsillitis often occurs independently of the special symptoms of catarrh. The symptoms and course of tonsillitis, moreover, are very characteristic, and the affection, therefore, calls for independent consideration. Tonsillitis is mostly a disease of childhood, but when once it has developed, it is apt to recur, and thus to be perpetuated into the period of adult life.

Morbid anatomy.—Simple or non-specific inflammation of the tonsils is characterised by inflammatory swelling of the tonsils themselves and of the soft tissues in their immediate neighbourhood, especially the pillars of the fauces, the soft palate and uvula, the base of the tongue, and the pharynx. The tonsil (for one is generally first and often solely affected) becomes increased in size, deeply congested, and infiltrated with inflammatory exudation and growth. The crypts upon its free surface produce superabundant epithelium, which accumulates in their orifices, forming opaque, yellowish, creamy pellets. The lymphatic nodules of the interior undergo inflammatory overgrowth, and often soften, suppurate and run together, and ultimately form an abscess. The soft palate and pillars of the fauces become of a vivid red hue, swollen, tense and shining, and more or less displaced;

and thus, if the swelling of the tonsil and surrounding parts be extreme, we find the soft palate on the affected side pushed downwards, forwards, and inwards, the anterior faucial pillar correspondingly displaced, and both together forming a smooth, tense, vividly red swelling, with the convexity facing forwards. The swelling and displacement indeed of the surrounding parts are sometimes so great that the enlarged tonsil itself is almost concealed. When both tonsils are involved, their affection is sometimes concurrent, more frequently in sequence. Often indeed the one is getting well when its fellow first shows signs of disease. When the tonsils are both very large, they may meet one another in the mesial line, becoming flattened and sometimes ulcerated from mutual pressure, and between them almost completely closing the faucial canal. The uvula, which is usually swollen, tense and congested, often clings to one of them; it may be so much elongated as to hang into the upper part of the larynx. Further, the tongue gets covered with a thick creamy fur, and the glands at the angle of the jaw, and sometimes the salivary glands, share in the inflammation, and become hard and large.

Symptoms and progress.—The invasion of tonsillitis is almost always marked by the occurrence of severe febrile symptoms, associated with soreness, itching or tingling, dryness and aching in the region of the fauces. The febrile symptoms increase in severity with the increase of the local affection, and with the cessation of the latter gradually, or, it may be, suddenly subside. At the beginning the patient experiences alternate flushes of heat and chills, and even distinct rigors; his temperature rises, and often reaches an elevation of at least 102°; not unfrequently, indeed, by the time the disease has attained its maximum, it mounts to 104° or even 105° and upwards; his pulse increases in frequency, rising to 100 or 120, and is at the same time more or less full and firm; his skin is hot and pungent, with a marked tendency to remittent sweats; he complains of headache, pains in the back and limbs, thirst and anorexia; his bowels are confined, his urine dark-coloured and scanty. The appearances which the tonsils and interior of the mouth present may be gathered from the description which has been given of these parts. It remains to say that the patient has severe pains at the back of the throat and base of the tongue whenever he moves his jaws, or speaks, and especially whenever he opens his mouth widely or attempts to swallow. The pain then not unfrequently shoots along the Eustachian tubes to the ears. He has a constant desire to swallow in order to relieve his uneasiness, but the pain and difficulty of swallowing are so great that he permits the secretions to accumulate in his mouth; and, in attempting to swallow, fluids not unfrequently pass up into the nose. The quality of the voice is nasal and characteristic. There is often deafness, and always more or less fulness and tenderness behind the angles of the jaw. The swollen tonsils indeed may be felt in these situa-

tions. If one tonsil only be inflamed, or both be simultaneously affected, the malady will probably attain its height in three or four days, and end in convalescence at the end of a week or ten days. Occasionally its course is yet more rapid, and the patient is well, or nearly so, in three or four days. But when one tonsil is affected after another, the course of the disease is necessarily protracted. If an abscess form, as is usually the case when the attack is severe, the severity of the symptoms progressively increases up to the moment at which the abscess breaks. Then the tonsil suddenly shrinks within moderate dimensions, and the patient is probably at once restored to comparatively good health. The matter which escapes is fetid and thick, and usually swallowed. The symptoms of tonsillitis are severe out of all proportion to the seriousness and danger of the affection. Any other termination than that of recovery within a brief period is very rare. The interference with swallowing, which seems so serious, never prevents the taking of food for more than a very limited period. Occasionally, however, death results from suffocation, due either to the sudden bursting of a large abscess and the entrance of its contents into the larynx, or to the mechanical impediment which the inflamed and swollen parts interpose to respiration.

Treatment.—Tonsillitis is one of that large number of diseases which take their own course. It may, nevertheless, be relieved by appropriate measures. The patient should be submitted to the same plan of general treatment that has already been recommended as suitable for catarrh. Nor need there be much difference in respect of local treatment. Hot fomentations, or flannel, or cotton wool may be applied to the exterior of the throat; and the patient be persuaded to gargle his fauces frequently with warm milk, or to allow the steam of boiling water to play upon them, or to suck black-currant jelly and such-like substances. Swallowing lumps of ice, however, and the application of ice-cold compresses to the neck often give far greater relief than warmth. Astringent and stimulating gargles are often recommended, as is also the application of nitrate of silver. Such treatment, however, is more suitable to the period of convalescence, at which time also tonics and good food may be specially needed. Opium judiciously administered generally gives great relief. When the swelling of the tonsil is extreme and the congestion intense, and the patient at the same time is suffering severely, relief may sometimes be afforded by scarifying or puncturing the tonsil. The value of such treatment, however, is chiefly seen when suppuration has taken place. Care should be exercised in puncturing the tonsil not to wound the large vessels which run along its outer aspect. The point of the lancet should be directed backwards, with an inclination inwards. But even if no large vessel be injured, dangerous hemorrhage occasionally ensues.

B. *Chronic Tonsillitis.*

Symptoms and progress.—As a consequence, sometimes of frequently repeated attacks of acute tonsillitis, sometimes of chronic inflammation, the tonsils undergo gradual hypertrophy, and form indolent tumours, which more or less seriously diminish the size of the faucial passage, and occasionally come into actual contact with one another. The presence of such tumours may be scarcely apparent to the patient himself; but in many cases, especially if large, they give a peculiar quality to the voice, which is indescribable, but impossible not to recognise when once it has been pointed out; and not unfrequently there is associated with them some chronic thickening of the mucous membrane of the pharynx and Eustachian tubes, with more or less deafness. Further, such patients are generally liable to frequent exacerbations of the affection.

Treatment.—Tonic medicines, iron and quinine and the like, good diet, fresh air, and healthful exercise are of essential value in the treatment of chronic tonsillitis. It is commonly held that the application of strong solutions of nitrate of silver or of the solid caustic, or other such agents, is serviceable in promoting the disappearance of these bodies. Such applications are no doubt frequently beneficial in allaying inflammation affecting their surface. But the only effectual way of dealing with them is to remove them by the knife.

VIII. RETRO-PHARYNGEAL ABSCESS.

Causation.—Retro-pharyngeal abscess is usually due to caries of the cervical vertebræ, and is sometimes one of its earliest indications; it may be connected also with suppuration in and about the tympanum and Eustachian tube, even when the bone is not involved. We have met with it in a case of aortic aneurysm.

Symptoms and progress.—A retro-pharyngeal abscess is situated, as its name indicates, between the posterior wall of the pharynx and the anterior aspect of the vertebræ, and forms a convex protrusion of greater or less extent and prominence at the back of the pharynx. It may be so high or so low as to escape detection by the usual method of observation; but in most cases it forms a visible bulging at the back of the throat. It is sometimes symmetrical, sometimes more or less one-sided, soft and yielding to the touch, and not necessarily presenting superficial congestion. It is liable to undergo perforation from time to time, to allow of a more or less free temporary escape of matter, and consequently to vary in bulk. Its presence is sometimes

productive of pain and difficulty in swallowing, and has been known to impede respiration and even to cause death by such impediment; but not unfrequently it is, for a time at least, simply a source of discomfort to the patient, in consequence of the pus which it exudes, the foul taste which it gives, and the fetor which it imparts to the breath. The progress of the abscess mainly depends on that of the disease which produces it.

The *treatment*, apart from the use of tonics, which is generally clearly indicated, is essentially surgical.

IX. OZŒNA.

Causation.—This term is applied to all those cases which are attended with fetid discharge from the nose. The causes of ozœna are in some cases mere chronicity of inflammation of the mucous surface, in some ulcerative destruction or gangrene, and in a large proportion of cases, caries or necrosis of the nasal bones. These several morbid conditions are for the most part connected, either with a scrofulous condition, with syphilis, with lupus, or with polypoid or malignant growths occurring in the nasal cavities.

Symptoms.—The discharge which escapes from the nostrils varies considerably both in character and in quantity. Sometimes it differs little in appearance from ordinary mucus, often it is thick and purulent, sometimes it contains blood, sometimes it is thin and ichorous. It frequently also tends to concrete in the cavities of the nostrils into thick crusts. The accumulation of unhealthy discharges in the antrum and other sinuses connected with the nose often leads to their decomposition, and to fetor; and the escape of such discharges is apt to take place at irregular intervals. The nature of the stench which is emitted varies greatly both in quality and in intensity. In some cases it is horribly disgusting. Ozœna is generally attended with more or less complete loss of smell.

The source of fetor may, even in the absence of discharge, be readily ascertained by making the patient respire alternately through the mouth and nose, and ascertaining under which of these conditions it is chiefly developed.

Treatment.—For this purpose the determination of the cause is of fundamental importance. If it be syphilitic, antisyphilitic remedies must be given; if connected with enfeebled constitution, tonics and good diet must be enjoined. Under any circumstances the nose should be kept clean; it should be frequently washed out by means of either a syringe or the nasal douche, with a weak alkaline solution, or a weak solution of quinine, Condy's fluid, chlorinated soda, chlorate of potash,

or carbolic acid; and either stronger solutions of the same agents should be occasionally employed as injections, or appropriate powders should be frequently blown in or sniffed up For the latter purpose Trousseau especially recommends bismuth diluted with an equal part of some inert powder, or white precipitate mixed with about forty times its weight of finely-powdered sugar.

X. MORBID GROWTHS.

A. *Tubercle.*—Miliary tubercles are described by Virchow as occasionally affecting the mucous surface of the tongue, palate, and nose, and there producing shallow sinuous ulcers, such as characterise the tubercular process in other mucous membranes. Miliary tubercles are difficult of recognition in these situations during life; at the same time it is a fact that shallow, intractable ulcers, not improbably due to this cause, are not altogether uncommon in the fauces and soft palate of phthisical patients, even at an early period of their disease.

B. *Syphilis.*—Syphilis in its secondary and tertiary stages, is very apt to affect the tract of mucous membrane now under consideration. 1. Erythematous patches, for the most part symmetrical, may appear on the pharynx or palate, inside the lips, or elsewhere in the mouth during the prevalence of the secondary cutaneous eruption. 2. Mucous tubercles may get developed during the same period, principally on the lips, dorsum and edges of the tongue, tonsils, and palate, and in the pharynx; and shallow ulcers, secondary to these tubercles, or of independent origin, are not unfrequent in the same situations. 3. At a later period of the disease deep ulcers appear, most commonly in the soft palate, tonsils, fauces, and pharynx, frequently spreading in a serpiginous manner, and gradually involving a wide extent of surface or penetrating deeply, and in either case leading to serious destruction of tissue. 4. Lastly, gummatous tumours are not uncommonly developed in the soft palate, and pharynx, but more especially in the substance of the tongue.

For a further account of these affections, and their treatment, we must refer to the article upon syphilis.

C. *Malignant tumours.*—Tumours of various kinds originate in, or involve, the mucous membrane of the mouth and fauces, or the organs which are contained within the mouth. But it scarcely falls within the province of the physician either to investigate or to treat them. Malignant affections of these parts alone have any medical interest. They are not uncommon.

In persons advanced in years, epithelioma of the lips (more especially of the lower lip) is apt to occur; in those who have attained or

passed middle life a similar affection of the tongue is not uncommon; and not unfrequently, under the same circumstances of age, malignant disease, mostly epithelioma, but sometimes carcinoma, sometimes sarcoma, becomes developed in some part of the fauces or pharynx. Again, malignant tumours (commonly some soft variety of carcinoma or sarcoma) occasionally form in connection with the mucous membrane of the nose, for the most part in young children or persons advanced in life. Further, sarcomatous and carcinomatous tumours, originating either in periosteum or in bone, form outgrowths from the bones of the upper and lower jaws, from those bounding the nasal cavity, and from the cervical vertebræ.

Malignant tumours of the mucous membrane are nearly always primary; they are often slow and insidious in their progress, and apt at first to be mistaken for some trivial affection; they are especially liable when they have made some progress to be confounded with syphilitic affections. That they are not syphilitic is, however, soon revealed by the total inoperativeness upon them of antisyphilitic treatment, and by their further progress. They gradually and surely invade the surrounding textures, gradually ulcerate and slough—causing more and more extensive destruction and yielding a foul discharge—and always before long involve the neighbouring lymphatic glands. These then form gradually enlarging tumours, which presently undergo precisely the same changes as the primary tumour. The diagnosis of these cases, which is often very uncertain in the beginning, rests mainly upon microscopic examination and on careful observation of their gradual and characteristic progress.

Their *treatment* is purely surgical.

Section II.—DISEASES OF THE ŒSOPHAGUS.

I. INTRODUCTORY REMARKS.

Anatomical relations.—The œsophagus commences at the cricoid cartilage, opposite the lower border of the fifth cervical vertebra, and runs down along the spine, a little to the left side, as far as the ninth dorsal vertebra, opposite which it penetrates the diaphragm and opens into the stomach. In the neck it has the trachea in front of it, with the recurrent laryngeal nerves between them, and on either side the common carotid artery. In the chest it is covered in front by the lower part of the trachea and then crossed by the left bronchus, after which it comes into contact with the pericardium. On either side of it is the pleura. The transverse and descending arch of the aorta cross the front and left side of the œsophagus on the level of the second and

third dorsal vertebræ; and the thoracic portion of that vessel lies to its left and behind it throughout the rest of its course, excepting just as it perforates the diaphragm, when the aorta slips altogether behind it.

II. INFLAMMATION OF THE ŒSOPHAGUS.

Causation and morbid anatomy.—The œsophagus is liable to share in all those inflammatory conditions which affect the pharynx and larynx. We have pointed out that the inflammation of a simple 'cold' may travel downwards along this tube; and when inflammation of special intensity involves the organs in relation with it, the œsophageal inflammation may be equally intense. Occasionally, indeed, under such circumstances thickening of its walls, with purulent infiltration of them and of the surrounding connective tissue, may extend from the pharynx to the cardiac orifice of the stomach. Further, the specific eruptions of some of the infectious fevers may involve the œsophagus, the diphtheritic false membrane may pervade its whole extent, and aphthous patches may form here and there upon it. Inflammation is sometimes also the result of swallowing boiling water or corrosive substances, such as the mineral acids, caustic alkalies, and other chemical agents.

Symptoms.—In nearly all the above cases the œsophageal inflammation is associated with similar but probably more severe inflammation, either of the larynx, pharynx, and fauces above, or of the stomach below; and the graver symptoms of these other affections tend to mask more or less completely the presence of the œsophageal complication. The special indications of inflammation of the œsophagus are: the presence of heat and pain in the course of that tube; aggravation of pain in the same situation during the act of swallowing, and in very severe cases inability to swallow; and tenderness on pressure applied to the neck in the situation of the œsophagus. The absence, however, of such symptoms does not disprove the presence of either general slight inflammation, or limited tracts of inflammation.

III. CHRONIC AND OBSTRUCTIVE DISEASES OF THE ŒSOPHAGUS.

A. *Ulceration.*

Causation and morbid anatomy.—The most frequent cause of ulceration is either mechanical violence, or the operation of destructive re-agents, to which may be added perforation of the œsophagus from without.

Small ulcers and mere excoriations doubtless get well, as a rule, without leaving any permanent ill effects behind; but when ulcers are extensive and deep, even though they be free from any malignant taint they are liable sooner or later to induce serious results. Of these the most important is cicatrisation, with consequent contraction of the calibre of the tube, and the supervention of a stricture which tends to become more and more tight. Other results are the formation of a sinus between the œsophagus and trachea, or left bronchus, and the perforation of an artery.

B. *Morbid Growths.*

Morbid anatomy.—The œsophagus is occasionally the seat of syphilitic disease with ulceration which by cicatrisation may cause more or less serious contraction and obstruction. Of all adventitious formations, however, the most common and the gravest are of a malignant character. These are chiefly met with after the age of 40 or 45, and in the great majority of cases are of primary origin. The most frequent variety of malignant disease, probably, is epithelioma; but encephaloid and scirrhous cancers are not unfrequent; and colloid cancer also has been observed. The seat of the disease is very various. In some cases it occupies the upper extremity of the tube, probably then involving also the contiguous pharynx and larynx; in some cases it is found at the lower extremity, when it is often associated with similar disease of the neighbouring cardiac extremity of the stomach; but in the greater number of cases it occurs in some intermediate spot, and very frequently in that part of the tube which is in relation with the trachea and bronchi. The affection, when primary, usually commences at some spot in the thickness of the mucous and submucous tissues, whence it spreads: superficially, so that before long it probably occupies three or four inches of the length of the œsophagus and its whole circumference; and in depth, so that sooner or later it implicates the whole thickness of the walls, and probably invades also the trachea or other neighbouring tissues and organs. The free aspect of the growth is at first somewhat nodulated; but the nodules running together soon form more or less flattened elevations, in connection with which, before long, ulceration, sloughing, and the formation of fungous outgrowths take place. The thickened walls and nodulated outgrowths reduce the calibre of the œsophagus, and sometimes render it almost impervious. The subsequent ulcerative destruction occasionally leads to its imperfect restoration. When the disease is of the colloid variety, the close-set vesicles of the growth open on the mucous surface, and abundant, clear, glairy fluid escapes.

In the progress of malignant disease various accidents are apt to arise. Sometimes the trachea or left bronchus gets perforated, and a more or less free communication between it and the œsophagus esta-

blished; sometimes the œsophagus opens into the posterior mediastinum, or externally, or communicates by ulceration with one of the œsophageal or intercostal arteries, or the left subclavian. And besides the mere spread by contiguity, œsophageal malignant growths, like those of other parts, soon cause secondary disease in the neighbouring lymphatic glands, and, if the patient survive sufficiently long, disease of remote organs. The involvement of lymphatic glands, especially if they be those of the neck, is very often valuable as an aid to diagnosis. Further, it not very unfrequently happens that the recurrent laryngeal nerve, especially that of the left side, gets implicated, and paralysis of the corresponding vocal cord induced.

C. *Affections implicating the Œsophagus from without.*

Causation and morbid anatomy.—The œsophagus is apt to be pressed upon or otherwise affected by tumours and other morbid conditions originating externally to it; and the patient's sufferings in many such cases are mainly, if not entirely, due to interference with the functions of this canal. Thus it may be compressed by an overgrown thyroid body encircling the trachea and acting upon it laterally; by a carotid or innominate aneurysm, or an aneurysm of the descending arch or thoracic aorta; by enlargements of the bronchial glands and other mediastinal growths; by tumours springing from the vertebræ; by abscesses; and even by a distended pericardium or dilated auricles.

Again, aneurysms and abscesses not unfrequently open into the œsophagus with a sudden and copious escape of blood or pus. Occasionally they open simultaneously into the œsophagus and trachea or one of the bronchi, causing more or less free communication between these tubes. And further, rupture of an aneurysm of the lower part of the thoracic aorta occasionally causes an accumulation of coagulum around the cardiac end of the œsophagus with complete obstruction of its passage.

D. *Dilatation.*

Causation and morbid anatomy.—Whenever a stricture of the œsophagus has existed for any length of time, a tendency shows itself for the part of the tube below to contract and even to undergo atrophy, and for the part of the tube above to become dilated and at the same time hypertrophied in respect of its muscular parietes. The same results indeed follow here as follow in the case of the bladder when there is stricture of the urethra. This dilatation and hypertrophy are in the majority of cases not strikingly apparent; sometimes, however, they are considerable, and especially when the stricture is situated low down, is non-malignant, and has been in existence for many years. Under such circumstances the œsophagus becomes dilated either in its whole length, or in a part of its length only,

forming an elongated pouch, which may have a circumference of five or six inches. Such dilatations are sometimes discovered in cases where their development cannot be traced to the existence of any mechanical impediment. It seems obvious, however, that they must even here be due, partly to distension by accumulated contents, and partly to powerful and sustained efforts of the muscular tunic to drive these contents onwards; and that hence there must have been in the first instance some weakness or sluggishness of the tube, some virtual impediment, permitting of such accumulation.

E. *Spasm and Paralysis.*

Spasmodic stricture of the œsophagus generally occurs in nervous persons, and especially in hysterical women. It may appear, however, without obvious cause, in persons of quite different nervous organisation; and not unfrequently supervenes in the course of organic œsophageal disease, causing temporary aggravation of the patient's symptoms.

Paralytic conditions of the œsophagus are rare. They may be hysterical, or dependent on profound affection of the central nervous organs.

F. *Symptoms. Dysphagia.*

A common symptom of nearly all the above lesions is dysphagia, or difficulty or pain in swallowing. It is this symptom, indeed, which generally first attracts attention to the œsophagus as the seat of disease; and it is only by the subsequent history of the case, by the supervention or non-supervention of other phenomena—oftentimes mere hints—that we are enabled, with more or less accuracy, to ascertain the exact nature of the disease which is present.

But dysphagia is a symptom of many other morbid conditions besides these; and especially of affections of the mouth, fauces, larynx, and pharynx. But dysphagia, due to morbid states of the parts here enumerated, is for the most part merely a subordinate symptom of diseases otherwise well characterised. It is very different, however, when the impediment to swallowing exists in the course of the œsophagus; it is then not merely a symptom, but it is *the* symptom by which alone, in many cases, the presence of disease is indicated.

The symptoms of organic obstruction are usually of slow development; the patient perhaps first experiences an occasional hitch in the passage of food to the stomach—a hitch which is chiefly obvious when solids are being swallowed. This is variable, partly because the bulk and character of the swallowed bolus differ from time to time, and partly from the occasional superaddition of more or less spasmodic contraction. Further, it is probably always referred to a definite point, and is not unfrequently associated with more or less well-marked

soreness or pain there. For some time probably these symptoms have little attention paid to them; but gradually they increase in severity and constancy, and attend the swallowing of both liquids and solids; further, the food before long begins to accumulate above the seat of obstruction, and hence to be regurgitated after a longer or shorter period of time with a kind of gulp—an effort which often has little or no resemblance to ordinary vomiting. The patient then finds it necessary to restrict his diet to slops, and ere long finds that he can take even such food as this only in the smallest quantities, and with difficulty and distress. He then rapidly emaciates, and if no fatal complication ensue, dies after a shorter or longer period of suffering from simple starvation. Such deaths are usually exceedingly distressing, because the patient, as a rule, retains his mental powers unimpaired to the last, and craves for nourishment which cannot be administered to him. These are the general symptoms of œsophageal obstruction ending fatally. But the progress of the case is usually largely modified by the nature of the disease on which it depends.

If the case be one of simple stricture from a cicatrix, its course is generally much protracted. And although such cases are often ultimately fatal, instances are on record in which patients have lived, though with more or less discomfort, to a good old age, and have then died of some other ailment. It is in them especially that dilatation of the tube above the stricture with compensatory hypertrophy takes place—conditions which, confined within certain limits, tend to neutralise the effects of the stricture.

If the case be one of malignant disease, this fact is often for a while incapable of determination. The points which specially indicate it are: the comparative rapidity with which the case goes on from bad to worse; the advanced age of the patient; the appearance of indurated glands in the neck; and the discharge from the œsophagus, in company with regurgitated food, of offensive, puriform, or sanious matter or detritus. Further, the sudden discharge of blood in large quantity, or the establishment of a communication between the œsophagus and air-passages, strongly indicates, though it does not absolutely prove, the presence of a malignant ulcer.

The symptoms due to the pressure of external growths differ but little from those arising from actual disease of the œsophageal walls; indeed the latter usually after a time become distinctly implicated. To aid our diagnosis we must carefully explore the neck and thorax, in order to ascertain whether there be an enlarged thyroid body, a mediastinal growth, an aneurysm, or any other form of tumour. But although in many such cases we may be enabled to form a correct diagnosis, in many all our efforts will necessarily be fruitless.

We have stated that organic obstruction is usually of slow development; nevertheless it occasionally arises with sudden completeness. In the case, for example, of obstruction from the compression exerted by

a circle of effused blood around the cardiac orifice, the symptoms occur quite suddenly, and the patient dies probably of starvation at the end of ten days or a fortnight.

An important point in reference to œsophageal obstruction is to ascertain its exact seat. It is important, partly in connection with the treatment to be adopted, partly as an element in determining the exact nature of the obstruction. Its site may be pretty correctly determined in many cases by the sensations of the patient. It is often indicated to some extent by the phenomena which follow the ingestion of a few mouthfuls of milk or other food; thus, if it be at quite the upper part of the tube, regurgitation immediately follows the act of deglutition, and is probably attended with the intrusion of some of the fluid into the larynx; if it be seated near the cardiac orifice, the return of food may be delayed for some seconds or minutes. It is, however, on the passage of the bougie, and the determination of the exact point at which its progress gets arrested, that our main reliance must be placed. Another useful method is that of auscultating the œsophagus. If the stethoscope be applied to the back in the course of this tube, and the person examined be made to swallow a mouthful of some fluid, its momentary passage in the form of a compact mass is distinctly audible. If, however, an impediment exist, especially if the impediment be considerable, there will be some obvious delay in the passage of the mass at its seat; and, moreover, the mass, instead of passing in a compact form, will probably trickle through in driblets, and its passage be attended with comparatively prolonged gurgling. It is not sufficient, however, to determine on one occasion the existence of gurgling at a particular spot. We must ascertain, by repeated observation, whether that localised gurgling is permanent or not.

Dilatation alone of the œsophagus is an impediment to the act of deglutition. The presence of dilatation, even if there be muscular hypertrophy, necessarily renders the œsophagus a less efficient instrument for the propulsion of its contents. These, instead of being driven readily and rapidly onwards, accumulate in the flaccid bag, and thence find their way fitfully into the stomach. One of the most interesting phenomena connected with dilatation is the tendency which there often is for the accumulated contents of the tube to be regurgitated by an effort, more or less voluntary, into the mouth, as in the act of rumination.

Spasmodic stricture is apt to come and go more or less suddenly, and, if it be long continued, to present intermissions or variations of severity. It is attended with many of the symptoms of organic stricture, and may even lead to death by starvation. The diagnosis rests partly on the patient's history and general state of health, partly on the variableness of the œsophageal obstruction, and partly on the evidence furnished by the unopposed passage of the bougie.

The symptoms due to paralysis of the gullet are also mainly those

of obstruction. The food fails to be transmitted onwards to the stomach, and at the same time tends to accumulate in the tube and to distend it. The bougie passes without impediment.

G. *Treatment.*

Œsophageal obstruction is, in a very large proportion of cases, difficult and unsatisfactory of treatment. If it be functional only, the passage of a bougie will sometimes at once restore the capability of swallowing. The permanent cure, however, of such cases is to be obtained only by curing the nervous conditions on which the obstruction depends. If, on the other hand, the obstruction be organic, the tendency of the disease is to render the occlusion of the tube more and more complete, and actual cure is probably out of the question. We have no drugs which promote the absorption of cicatricial bands, or of carcinomatous or other tumours. We can, however, in some cases, by surgical means, check the progress of contraction, and even cause dilatation of a part already strictured. We have pointed out the importance, for diagnostic purposes, of passing an œsophageal bougie. The careful passage of a bougie through a stricture, and the repetition of the operation at intervals with instruments of gradually increasing size, will not only aid us in diagnosis, but in some cases relieve the stricture materially, and maintain that relief. The passing of a bougie, however, through an obstructed œsophagus is an operation of much delicacy, and attended with no inconsiderable danger, especially if the impediment consist of a tract of soft ulcerating cancerous material, or be due to the pressure of a thoracic aneurysm. The bougie may in fact, under such circumstances, readily form a false passage into either the trachea, the mediastinum, or the cavity of an aneurysm, and so induce speedily fatal symptoms. So great is this danger, that most practitioners regard this mode of treatment as almost entirely inadmissible; and indeed it must, we think, be conceded that it is quite inadmissible in cases of compression of the œsophagus by an aneurysm, and in cases of malignant disease—especially those in which ulceration or sloughing has taken place. But there cannot, we think, be a doubt of the benefit which may accrue from the regulated use of the bougie, in skilful hands, in cases of simple stricture. The dilator suggested by Dr. M. Mackenzie is well suited for such cases. The passage of the bougie has occasionally ruptured an abscess to which obstruction was due, and in this way cured the patient. When the ingestion of food is largely interfered with, and the patient shows manifest signs of starvation, the question as to whether he may be supplied with food by any other route than the œsophagus arises. The use of nutritive enemata is one of the methods which suggest themselves, and is often useful in prolonging life. Another method is that of laying open the stomach itself through the anterior abdominal wall, and feeding the

patient through the artificial opening. Several such operations have been performed, and although the cases have not been very successful, the feasibility of the operation has been clearly demonstrated.

SECTION III.—DISEASES OF THE STOMACH, INTESTINES, AND PERITONEUM.

I. INTRODUCTORY REMARKS.

A. *Anatomical relations.*

The surface of the abdomen is artificially divided into regions which are convenient in determining the relations of the organs situated within. This division is usually effected by drawing two horizontal lines—one above, from the lowest point to which the ribs descend on the one side to the corresponding point on the other side; one below, between the anterior superior spines of the iliac bones; and then intersecting them by two vertical lines drawn, one on either side, from the cartilage of the eighth rib above to the centre of Poupart's ligament below. Nine unequal spaces are thus mapped out; of which the three occupying the median aspect of the abdomen are, from above downwards, the *epigastrium* or *scrobiculus cordis*, the *umbilical region*, and the *hypogastrium*; and the three on either side are, in the same order, the *hypochondrium*, the *lumbar region*, and the *iliac region*. The hypochondriac and iliac regions are small and triangular; the lumbar extend round to the spine, occupying on either side the whole interval between the ribs and the crest of the ilium, and are, therefore, of considerable extent.

The *epigastric* region is occupied mainly by the stomach, inclusive of its pyloric extremity, portions of the right and left lobes of the liver appearing above on either side of the ensiform cartilage; more deeply seated lie the hepatic vessels, pancreas, cœliac axis, and semilunar ganglia. The *umbilical* and *hypogastric* regions are occupied almost exclusively by the convolutions of the small intestine; along the upper part passes the transverse colon, and into the lower part ascend the distended bladder and the gravid uterus. Deep in these regions lie the third portion of the duodenum above, and the mesentery with its vessels and glands below. The *right hypochondriac* region contains the lower edge of the right lobe of the liver with the gall bladder, and the hepatic flexure of the colon; more deeply the first and second portions of the duodenum; and more deeply still the upper part of the right kidney and the supra-renal capsule. The *left hypochondrium* is occupied by the lower portion of the spleen, the cardiac extremity of the stomach, the splenic flexure of the colon, and more

deeply by the upper part of the left kidney and the supra-renal body. Each *lumbar region* is occupied by the convolutions of the small intestine, laterally by the ascending or descending colon, and further back by the lower half of one of the kidneys. In the *right iliac region* is placed the cæcum, in the *left* the sigmoid flexure.

B. *Examination of the Abdomen.*

The direct examination of the abdomen in all cases of disease of the contained viscera, and in all affections attended with symptoms referrible to these organs, should never be neglected. And in conducting such examinations, and forming our opinions from them, we must always recollect, not only the normal positions of the parts within, but the facts that even in health many organs are liable to considerable changes of bulk and position, and that in disease such changes are often in the highest degree misleading. Apart from rectal, vaginal, and urethral examinations, which we shall not now enter upon, the methods of investigation include inspection, palpation, percussion, and auscultation.

1. *Inspection.*—Much may often be learnt by simple inspection. The *form* of the abdomen in many diseases is no doubt entirely normal; but it is often more or less importantly and characteristically modified. In cases of extreme emaciation, whether from starvation or any other cause, the surface becomes flattened or even concave; and a somewhat similar retraction of the parietes is frequently observed in cerebral disease, especially of children. On the other hand the abdomen is often more prominent than natural. This condition may be due to fat in the parietes, or to anasarca; in which case the general symmetry of the belly is maintained, but the umbilicus is usually deeply sunk. It may depend on distension of the stomach and bowels; when not unfrequently the abdominal walls (especially if they be thin) are moulded in some degree to the alternate depressions and elevations of the subjacent organs. When distension is the consequence of ascitic accumulation, the belly (owing to the influence of gravitation) always has a tendency, as the patient lies on his back, to expand laterally and to bulge in the flanks—a tendency, however, which often disappears when the accumulation becomes very large, and may be masked by coincident tympanites. Enlargement due to enlargement of solid organs, to tumours, or to abscesses, is rarely symmetrical. The *movements* of the abdominal walls are often significant. In pericarditis and pleurisy and especially in paralytic affections of the diaphragm, and peritonitis, with other inflammatory affections of the abdominal organs, the diaphragm becomes inactive, and the surface of the belly remains quiescent during respiration. In cases of distension of the stomach or bowels, especially if it be due to any mechanical impediment to their action, the peristaltic movements of the dilated organs may often be distinctly seen and traced. It need scarcely be added that the movements of the

fœtus in the gravid uterus are distinctly visible. The *condition of the parietes* again may be of service to us. We may note the presence or absence of eruptions, or of dilated veins which generally accompany ascites, tumours, and obstructive disease of the portal vein, vena cava, or iliac veins. We may also observe whether they present circumscribed redness, brawniness or swelling, such as indicates the pointing of an abscess or the extension of inflammation from below, or whether there be any cutaneous or subcutaneous tumour or an umbilical or any other hernia. Further, it may be remarked: not only that abdominal walls which have been the seat of much dropsical effusion or fatty accumulation fall into wrinkles when the fluid or fat disappears, but also that when once the abdomen has been largely distended (whether by pregnancy, ascites, or any other condition) they are liable to present those atrophic lines which habitually follow child-birth.

2. *Palpation.*—By manual or tactile examination we distinguish the different degrees of hardness, softness, resistance, and elasticity of the abdominal walls and subjacent parts, and can thus often determine the size, shape, quality, and relations of tumours. Moreover we may recognise the fluctuation due to the presence of fluid, the pulsation of arteries and aneurysms, the thrill or crepitation resulting from inflammatory deposit, and the peristaltic movements of the stomach and bowels.

When the parietes are flaccid, especially if they be at the same time thin, we may sometimes by careful manipulation map out the form of the kidneys and other deep-seated solid organs. Indeed under such circumstances the kidneys have been mistaken for tumours and the abdominal aorta (especially that part of it which lies on the promontory of the sacrum) for an aneurysm. When the walls are rigid (as in fact they are only too apt to be in those cases where examination is most needed) it is often exceedingly difficult to determine the condition of parts within. Moreover portions of the rigid recti muscles are then very liable to be mistaken for tumours. In this case the patient should be made to lie on his back with elevated shoulders and knees, and heels pressed into the bed; and then the physician with warm hand or hands should press quietly but firmly on the abdomen, making the patient from time to time draw a deep breath. By such means and taking constant advantage of each momentary relaxation, he may often in a short time overcome the muscular rigidity, and learn all that is necessary with regard to the subjacent organs. If these measures fail, the patient must be examined under the influence of anæsthetics.

The source of a tumour is in great measure distinguishable by its site and relations to the abdominal organs. We need not particularise the different localities in which we should expect to discover tumours of the liver, spleen, kidneys, or other viscera. We may, however, point out the importance and mode of determining whether a tumour

be in the abdominal walls or adherent to them, or spring from the back of the cavity, or be connected with some of the movable parts within. A tumour of the walls necessarily rises and sinks with the walls during respiration. A tumour connected with the liver, spleen, stomach, bowels, or omentum ascends and descends with the movements of the diaphragm, and, if unattached to the abdominal walls in front, can be distinctly felt to glide under them. This locomotion dependent on the diaphragm is of course most obvious in the case of tumours resting against the diaphragm or near it. Tumours springing from the kidneys or back of the abdomen are usually fixed, or, if movable at all, generally slightly in the transverse direction. Many growths connected with the stomach, bowels, ovaries, and peritoneum are freely movable, either under the hand of the examiner or with change of position. The form, size, and consistence of tumours, and the presence or absence of fluctuation in them, are points of importance.

The presence of fluid in the peritoneal cavity is generally attended with the sense of fluctuation. This is best obtained by pressing the left hand firmly and flat upon the abdomen, and then giving a sharp tap or fillip with the fingers of the opposite hand. It is most marked when the fluid is abundant, and the walls thin and tense. The sense of fluctuation comprises two elements: the one an instantaneous impulse conveyed through the fluid and not generally very perceptible; the other a wave which travels over its surface and involves the abdominal parietes. The latter is what is usually meant by the term; but it must be observed that it (or something very like it) is occasionally observed in flaccid abdomens free from dropsy, and that it may be arrested or annulled by pressure made on the abdomen between the finger which percusses and the hand which receives the impression. In connection with the presence of ascites it may be observed: that a layer of fluid, varying say from half to one inch in thickness, often intervenes between the upper surface of an enlarged liver and the anterior abdominal walls; and that the presence of the liver may then often be readily detected by pressing the finger perpendicularly with suddenness and force, and thus displacing the fluid and coming into sudden contact with the surface of the solid organ.

3. *Percussion.*—On percussing the abdomen we obtain as a general rule resonance or dulness, according as we operate over the stomach and bowels or over solid organs. Abdominal resonance is higher pitched and more musical than that elicited over the lungs. It is also much more variable in health, owing to the varying distension of the different parts of the alimentary canal. The percussion note is of course higher according as the tube percussed is narrower; hence deeper notes are usually obtained over the stomach than over the colon, and over the colon than over the small intestine. But in morbid states (which need not here be specified) the stomach may contract so as to

yield a note like that of the healthy ileum, or the ileum or colon may become so much dilated, as to furnish a note like that usually belonging to the stomach. Although percussion for the most part gives a dull sound over solid organs, it is an important fact that distinct resonance may often be elicited over the thin edge of the liver, due to the liver and abdominal parietes vibrating together over the subjacent stomach. Occasionally also such resonance may be elicited over the spleen. The determination of the exact distribution of resonance and dulness is often very important in reference to the diagnosis of abdominal tumours—the course, for example, which the ascending, descending, or transverse colon may take in relation to a tumour, often deciding for us whether it arises in the kidney, liver, retro-peritoneal glands, or some other part. It is necessary, therefore, to bear in mind that a line of bowel lying superficially to a solid mass may be readily overlooked, if care be not taken in the examination; for if, as in ordinary percussion, the finger of the left hand be pressed upon the part to be percussed, the bowel may be readily flattened under its influence, and dulness result. In all such cases, and indeed generally in abdominal percussion, when we are anxious to make a minute and critical examination of the condition of parts lying immediately under the walls, it is best to percuss by simply filliping the surface with the nail of the right forefinger. Of the peculiar prolonged thrill often observed on percussion over an hydatid tumour we shall speak hereafter.

4. *Auscultation.*—Of course gurgling and musical sounds of all sorts may be heard with the stethoscope over the stomach and alimentary canal, but little or nothing is to be learnt from them. Besides these, arterial murmurs due to the presence of aneurysm, or to the pressure of tumours, or of the stethoscope, venous murmurs in connection with the gravid uterus, abdominal tumours, or dilated veins, the beats of the fœtal heart, and friction sounds in connection with hepatic or splenic peritonitis, may be met with under different circumstances.

II. GASTRITIS.

Causation.—Acute gastritis in its severest form is exceedingly rare, unless as the result of the direct application of irritant or corrosive substances to the mucous surface of the stomach. Its milder varieties, on the other hand, are very common at all ages and in both sexes, and from their mildness not unfrequently escape notice. The causes of gastritis are various; and include the ingestion of irritant or corrosive substances, the use of food which is ill-masticated, too abundant, or unwholesome—therefore, excess in eating and the abuse of alcohol—exposure to cold, and other atmospheric influences. Among

predisposing causes must be enumerated constitutional debility, tuberculosis, various acute febrile complaints, heart, lung, and renal disease, and cirrhosis of the liver.

Morbid anatomy.—Slight inflammation, though obvious enough when seen, as in the case of Alexis St. Martin, during life, often leaves little trace of its existence after death. It is indicated by patchy congestion; enlargement of the epithelial cells, with a more or less cloudy condition of their protoplasm, and the appearance of fat granules within them; similar changes in the cells of the mucous glands; and hypertrophy of the lymphatic tissue. These conditions involve some degree of thickening and softening of the mucous membrane, and are attended with the formation of a greater or less abundance of ropy alkaline mucus, and diminished secretion of the true gastric juice. But these are not the only changes. Frequently, small extravasations of blood take place here and there into the substance of the mucous membrane, and small quantities of blood may even escape into the cavity of the stomach; and sometimes erosions, shallow ulcers, or superficial sloughs are developed. Some of the latter appear to be connected with previous hemorrhagic infiltration, if not dependent on it. When inflammation is due to the action of corrosive substances, the morbid appearances are determined largely by their several peculiarities of chemical action. There is usually, however, intense congestion, with more or less extensive destruction of the mucous membrane. Inflammation involving the whole thickness of the gastric walls is rare as an idiopathic affection. In these cases they are swollen in their entire thickness, sometimes infiltrated with simple inflammatory exudation, or pus, sometimes presenting scattered abscesses.

The morbid anatomy of chronic inflammation differs little from that of the acute affection. There is generally, however, less congestion and more degeneration. The mucous membrane is usually thicker than normal, pale, and comparatively tough. It may present extravasations of blood, and excoriations or ulcers. But more frequently it is studded here and there with black or slate-coloured spots, which are the pigmental remains of old extravasations or congestions; and with opaque white patches, which are due to fatty degeneration of the epithelial contents of groups of gland tubes, and even of the corpuscles of the connective tissue between them, and are often associated with atrophy and shrivelling of the glands, and a tendency to the formation of cysts.

Symptoms and progress.—1. In severe idiopathic gastritis, as also in gastritis due to irritant poisoning, the symptoms are of an exceedingly violent character. The patient suffers from intense burning, and shooting pain in the epigastrium and lower part of the chest in front, and between the shoulders, attended with rigidity and retraction of the abdominal muscles; extreme tenderness on pressure in the epigastric region; aggravation of pain on drawing a deep breath, with conse-

quent shallow respiration; nausea, retching, and vomiting, not only after everything that is taken into the stomach, but even when the organ is empty; total loss of appetite; intense thirst; and collapse, marked by extreme feebleness of pulse, coldness and pallor of surface, cold perspirations, and tendency to faint. Besides these symptoms, distressing hiccough usually supervenes, and the bowels may become loose. The character of the vomit depends on circumstances. Generally, however, it comprises mucus (which is often mingled with, more or less, altered blood), and bile, and of course such matters as have been swallowed. The supervention of collapse, which forms so marked a phenomenon of the affection, is preceded by heat of skin and other febrile symptoms which, however, soon subside. When the case ends fatally, death is mostly due to prostration; and the patient usually retains consciousness to the last. The date at which death supervenes varies generally between one and six or seven days. If recovery take place, it is usually protracted.

2. In the commoner and milder forms of acute gastritis, the symptoms are essentially the same as those which characterise the graver attacks;—namely, heat or aching in the region of the stomach; tenderness on pressure in the epigastrium, with more or less rigidity of the abdominal muscles, especially the recti, and the endeavour to obtain ease by bending the body forwards, and restraining the action of the diaphragm; irritability of stomach, with tendency to eructation and to reject by vomiting whatever is taken into it; anorexia, thirst, and febrile disturbance. Besides which, the tongue is usually coated, and there is more or less headache, with intolerance of light, depression of spirits, and disturbed sleep. The symptoms are subject, however, to great variety, and even the most characteristic of them may be absent.

Pain in the stomach may fail wholly, or exist as a mere sensation of warmth, or it may be replaced by a violent craving for food. Ingestion of food, however, in such cases does not usually give the anticipated relief, and often brings on pain and induces vomiting. Irritability of the stomach may be extreme; on the other hand, it may be indicated by frequent eructations only. Under any circumstances, however, the taking of food or drink, except in moderation, will probably ensure its rejection and bring on epigastric pain. The vomit consists of ropy and tenacious mucus, mixed with matters which have been swallowed, and (if the vomiting have been prolonged) with bile. Blood, in small quantities, may be contained in it. The breath is usually febrile or offensive, and not unfrequently fetid. The eructations occasionally have the odour of sulphuretted hydrogen. Thirst is generally a marked feature, but now and then is wholly absent. The temperature is usually elevated above the normal, but rarely exceeds 100°; it presents variations during the day, and, for the most part, an afternoon or evening rise. The patient often feels chilly, and

even has distinct rigors. The skin is hot, but disposed to be moist at times. In most cases the tongue becomes early covered with a thick whitish or brownish creamy fur, through which the congested fungiform papillæ protrude; but it may be abnormally red and clean, and then often dry, glazed and fissured. In some cases it is little changed from the normal. Taste is usually perverted; and there is often a sensation of bitterness or a metallic flavour. Headache is usually very severe, of an aching or throbbing character, and limited to some particular region. In some cases it is difficult, if not impossible, to distinguish it from that of megrim. Chiefly when the headache is frontal there is disturbance of vision and photophobia. The patient is commonly more or less irritable and restless, yet depressed; he is often drowsy, yet unable to obtain refreshing sleep—his rest being disturbed by dreams. Further, the action of the heart frequently becomes enfeebled, the pulse quick and small, the extremities cold; and there may be palpitation, faintness, dyspnœa, and confusion of mind. Associated with gastric inflammation there is very often more or less disturbance of the bowels; generally flatulence, and either constipation, griping and purging, or irregularity of action. These disturbances, however, are in many cases due to concurrent inflammatory implication of the mucous membrane of the bowels.

In some cases the symptoms of the milder forms of acute gastritis scarcely differ from those of enteric fever; while in some they are little more than such as constitute an ordinary sick headache; and in others amount collectively only to that vague sense of illness to which the term 'malaise' is commonly applied. In young children, drowsiness and other cerebral phenomena, such as coma and convulsions, are not unfrequent accompaniments of the gastric disorder; and it is among them that diarrhœa is chiefly common. When gastritis arises in the course of other affections, its symptoms are liable to be overlooked.

3. The symptoms of chronic gastritis necessarily present a considerable resemblance to those of the acute disorder, but are on the whole more vague, and merge into those included in the collective term dyspepsia. The patient, moreover, is in many cases liable to remissions, during which he appears to enjoy comparatively good health. In other cases he ails continuously. Febrile symptoms, on the whole, are slight, and often altogether absent; thirst, anorexia, vomiting, and uneasiness or pain in the epigastrium and between the shoulders, are all more variable and generally less severe than in the acute disorder; vomiting, however, of an abundance of glairy mucus is often a characteristic phenomenon; the tongue varies in its condition, as it does in the acute affection, and often gets furrowed or intersected with fissure-like depressions; the breath is offensive; the bowels usually are confined; and the patient becomes restless, irritable, nervous, and hypochondriacal, but rarely suffers so severely from headache as

those who labour under the more acute disorder. With its continuance emaciation and debility come on, with defective circulation, coldness of extremities, and tendency to palpitation and faintness. Numberless other symptoms and consequences, of more or less importance, are commonly, and no doubt in the main correctly, attributed to chronic gastritis. For the most part, however, they constitute no essential part of it, and are connected with it only as they are with many other affections in which the processes of nutrition are profoundly involved.

Treatment.—1. In the treatment of severe acute gastritis, local measures are of great importance. Leeches—twelve, twenty, or more—may be applied to the epigastrium; or warm fomentations may be employed, or ice, or mustard poultices and other counter-irritants. Which of these applications should be selected must depend on the severity, or stage, or other conditions affecting the case. The irritability of the stomach renders the introduction of food and medicine in bulk into that organ impossible or undesirable. A little ice may be sucked, or ice-cold water or milk sipped; and opiates in large doses should be administered. If given by the mouth they should be in the form of pill, powder, solution of morphia, or undilute liquid extract of opium. The association of opium with bismuth or magnesia is often very efficacious. The best mode, however, of introducing opiates is undoubtedly by subcutaneous injection.

2. In less severe cases, local bleeding need scarcely ever be resorted to, but warm fomentations and counter-irritants are of benefit. Here also the use of ice, or minute quantities of ice-cold water, often affords much relief. And generally it is desirable to avoid as far as possible the administration of food or drink until irritability and pain have in great measure subsided. In some cases opium is of great value; but generally it is not called for. Bismuth, magnesia, lime-water, nitrate of silver, effervescent alkalies, and hydrocyanic acid are often beneficial. When constipation is present, or there is evidence of implication of the bowels, purgatives are valuable, especially perhaps castor oil, calomel in combination with rhubarb, and enemata. When food is given it should be of light quality and easily digestible. Milk and farinaceous substances are most suitable. Later on, animal broths, fish, and chicken may be allowed. Alcoholic drinks are not desirable, unless there be marked tendency to depression or collapse. Under similar circumstances ammonia is often serviceable.

3. Chronic gastritis usually requires much attention to hygienic conditions. The patient should be enjoined to take moderate and regulated exercise, to seek change of air and scene, to keep good hours, and generally to adopt such a mode of life and such habits as are conducive to health. The diet should be strictly regulated, but it is difficult to lay down definite rules with respect to such regulation. The patient's own experience is usually an important, if not the best, guide. He

should carefully avoid all those articles of diet which he has found to be prejudicial to him, however wholesome theoretically we may suppose them to be. Milk, well-cooked farinaceous substances, fish, fowl, and well-roasted mutton and beef in small quantities are probably on the whole the most suitable. Salted meats, rich and highly-seasoned dishes, pork and veal, should be especially eschewed. Tea often disagrees. Alcohol is seldom beneficial, and should only be used sparingly and in a dilute form. The particular beverage to be employed must depend on circumstances. As to medicinal treatment, the bowels should be regulated by occasional laxatives or mild purgatives; and tonics—especially quinine or nux vomica in combination with hydrochloric acid, and calumba or gentian associated with alkalies and rhubarb, or bismuth—effervescing medicines, lime, silver, zinc, hydrocyanic acid, belladonna, opium, and pepsine, have all been found more or less useful under different circumstances and in different cases.

III. ENTERITIS.

Causation.—Acute inflammation of the bowels, like the corresponding affection of the stomach, presents every degree of severity. The simplest, or catarrhal, form may be caused by the local action of irritating ingesta, or those external conditions which are commonly instrumental in exciting idiopathic inflammations. Young children, mainly about the time of teething, are specially liable to it; and it is said to be common in scarlatina and other specific fevers. Occasionally it becomes chronic, and is then apt to be associated with morbid states of other organs, to which indeed it is often secondary. The stomach especially, under these circumstances, is frequently the seat of some chronic morbid process. But enteritis in the usual sense of the term—the 'phlegmonous enteritis' of Cullen—is rarely of idiopathic origin; it is generally the result of some mechanical injury, and thus complicates strangulated hernia, intussusception, the impaction of gall-stones and other foreign bodies, and intestinal stricture.

Morbid anatomy.—1. Acute catarrhal inflammation of the bowel is characterised anatomically by congestion, tumefaction, and dryness of the mucous membrane, speedily followed by the more or less abundant secretion of mucus, which is ropy or watery, irritating, and sometimes mixed with blood. When the inflammation assumes a chronic form the mucous membrane becomes condensed and hardened, congested, and studded with black pigmentary deposits. There is often atrophy of the Lieberkühnian follicles, with granular or fatty degeneration of their epithelial contents; and atrophy, or even enlargement, of the solitary and agminated glands.

2. Occasionally, under conditions which are not well understood, membranous pellicles in patches arise, especially in the large intestine, in connection with chronic enteritis. They consist of corpuscular elements cemented together by a coagulable exudation, and are for the most part prolonged by rootlets into the Lieberkühnian follicles. Their development usually is attended with much greater congestion and thickening of the mucous membrane than is the simple catarrhal affection, and not unfrequently hemorrhage, suppuration, or gangrene ensues. In the large intestine the pellicular inflammatory patches are sometimes linear, sometimes irregularly polygonal or stellate, and occupy, for the most part, the prominent ridges of the mucous membrane, especially the edges of the intersaccular constrictions. In some cases, while still occupying the more prominent parts, they form a coarse irregular network over large tracts of surface; in other cases they coalesce into uniform patches of considerable extent. In the small intestine pellicular inflammation may be found, either affecting only the free edges of the valvulæ conniventes, or spread over large areæ. Cases sometimes come under observation in which patients pass per anum shreds of false membrane, or even membranous casts of the bowel, of soft texture, various thickness, and a dirty greenish or brown hue. This discharge is generally, if not always, the consequence of dysenteric ulceration.

3. The morbid changes discoverable after death in phlegmonous enteritis are such as are produced by intense inflammation of a limited tract of bowel. The affected part, which is mostly in the small intestine, and may vary in length from an inch or two to two or three feet, is as a rule much dilated. Its serous surface presents a general dusky red, slaty, or purplish black colour, due to the condition of the parts internal to it; it is marked, too, by lines or patches of more or less intense superficial congestion, may present blotches of subserous extravasation, and is often covered to a greater or less extent with adherent lymph. Its mucous and submucous tissues are mostly somewhat thickened and softened, sometimes only moderately congested but presenting spots and streaks of extravasation, sometimes black from combined congestion and escape of blood, sometimes pale and infiltrated with lymph or pus, sometimes distinctly gangrenous. And its middle coat, sharing in these changes, is also more or less swollen and soft, congested or œdematous, or the seat of some form of inflammatory exudation. The inflamed tract usually presents fairly well-defined limits, terminating abruptly below in pale and healthy but contracted and nearly empty bowel, above in bowel which may also be healthy, but is dilated like the diseased portion, and filled like it with fæcal contents. The diseased intestine frequently contains, in addition to fæcal matter, more or less sanguineous exudation, or a thick pitchy fetid fluid; and traces of the same may often be discovered in the contracted bowel below.

Symptoms and progress.—1. Catarrh may affect the lower bowel only, causing mild dysenteric symptoms; but very often it begins in the upper bowel, or stomach, and spreading thence downwards gradually traverses the whole length of the intestinal canal, causing in its progress more or less uneasiness, aching, and griping—frequently attended with nausea and sickness while the inflammation is still high up, with diarrhœa and expulsive pains and efforts when it reaches the large intestine. The tongue generally is more or less furred and dry, the breath offensive, and the appetite impaired; but these symptoms vary, and are often absent, especially when the large intestine alone is affected. Some degree of general febrile disturbance, indicated by heat and dryness of skin, sense of chilliness, increased frequency of pulse, lassitude and headache, is usually attendant on the local disorder. In children, in whom inflammatory affection of the gastro-intestinal mucous membrane is sometimes associated with aphthæ, the disease not unfrequently causes serious results and death, either from the debility which follows persistent diarrhœa and vomiting, or from the supervention of convulsions and coma. It is obvious that the symptoms of this disorder differ but little from those assigned to the commoner varieties of gastritis; but gastritis and enteritis are usually associated more or less intimately, and their respective characteristics consequently get intermingled.

The symptoms of the chronic disorder vary greatly, but may be briefly summarised as combining, in various proportions, imperfect digestion of the alimentary matters received into the intestine, excessive secretion of more or less watery mucus, increased peristalsis with griping pains, looseness of the bowels, discharge of watery, yeasty, or otherwise unhealthy and offensive evacuations, and innutrition from imperfect absorption of food.

2. The symptoms which attend enteritis with the formation of membranous pellicles are not special; they vary, on the one hand, between those of diarrhœa and dysentery, and on the other hand between those of mere colic and typical enteritis; moreover the affection is often overlooked from the fact that it is apt to occur as a complication of the later stages of many grave disorders, as, for example, acute pneumonia, Bright's disease, cirrhosis of the liver, and cerebral affections.

3. The symptoms of phlegmonous enteritis are, even when the disease is unattended with any of the mechanical lesions which so often complicate it, liable to considerable variety—the variations depending mainly on the degree of inflammation and its extent, and on the situation of the affected portion of bowel. The principal factors in producing the characteristic symptoms are inflammation, on which the various febrile phenomena depend; and paralysis of the inflamed tract of bowel, which permits of its passive dilatation by the accumulation of contents, opposes a more or less complete bar to their

transit, and thus induces, on the one hand constipation, on the other vomiting.

Heat of skin, rigors, and quickness and hardness of pulse, not unfrequently mark the onset of the attack; but the invasion is in many cases insidious and unattended with obvious febrile symptoms. There is mostly some dryness and clamminess of the mouth, if not absolute thirst; and the tongue, which is occasionally pretty clean at the beginning, generally soon gets thickly coated, and ultimately dry.

A special feature of enteritis is the association of the abdominal pain and tenderness of peritonitis with the tormina of colic. Pain and tenderness are certainly present in most cases, at least in the beginning, and in dependence upon them the dorsal decubitus so characteristic of peritoneal inflammation. They are sometimes, however, scarcely appreciable from first to last, and generally subside in the progress of the case. It can readily be understood that, when the peritoneal surface is largely involved, pain and tenderness will generally be proportionately severe; that when an extensive length of bowel is affected there will be correspondingly extensive uneasiness and tenderness; and that when, as sometimes happens, the serous surface is not inflamed, or the affected portion of bowel is small, pain and tenderness may be not only limited in extent, but no greater than we find them in colic or simple ulceration of the mucous membrane. It may be observed that limited pain and tenderness are very commonly referred to the region of the umbilicus. Tormina are often at the onset very agonising, and are then probably due in some measure to the spasmodic movements of the inflamed bowel; but they continue after paralysis is established, in consequence of the violent but ineffectual efforts of the bowel above to overcome the impediment which the disease produces. But tormina are sometimes scarcely recognisable, and frequently, like pain, cease comparatively early.

Constipation and vomiting are among the most important symptoms of enteritis. Constipation, in the uncomplicated affection, is due simply to want of contractile power in the inflamed length of gut. It is therefore not necessarily absolute; there is no reason why the attack should commence with constipation, or why the bowel below the seat of disease should not empty itself in the progress of the case, or even why a certain amount of fæcal matter should not slip through the inflamed region into the healthier bowel below. Nevertheless the inflamed bowel is really a substantial impediment, constipation is a striking incident in the disease, and purgatives as a rule fail to produce a purgative effect. The vomiting of enteritis is probably at the commencement mainly functional, but ultimately it is due, like the constipation, to intestinal obstruction. In the first instance, no matter where the obstruction, the vomited matters are merely the secretions of the stomach mixed with alimentary substances; but soon bile is added, and before long glairy mucus and bile alone are discharged. Then the

eructations become fetid, and the fluid brought up is turbid and brownish, and by degrees comes to resemble the contents of the lower part of the small intestine; but it becomes fetid also—far more fetid, indeed, than the contents of a healthy bowel ever are. This discharge of 'stercoraceous' matter by the mouth is due, not to inverted peristaltic action, but to the fact that the general contents of the distended bowel are gradually churned up, as it were, and intermingled, by the constantly recurring peristaltic movements of their muscular walls.

Hiccough is often a distressing symptom. Tympanites is probably always present; slight at the beginning, but increasing as the case progresses until the belly becomes greatly distended, tense, and drum-like. It is due mainly to the distension of the inflamed bowel, and that above it with fæcal matter and flatus. But now and then it is connected with rupture of the distended intestine and escape of gas into the peritoneal cavity.

The pulse usually is accelerated and hard at the beginning, but varies in different cases in frequency, volume, and strength, and is sometimes nearly normal in character; but as the fatal issue approaches it gets more and more feeble, and sometimes at length wholly imperceptible at the wrist. It generally also becomes quicker, but sometimes slower, and not unfrequently irregular.

The temperature of the skin is usually in the first instance more or less elevated, and the surface dry; but even then sweats are apt to break out, especially during the colicky paroxysms; subsequently the temperature falls, the extremities and face get cold and pale or livid, with sometimes a slight tinge of jaundice, and all parts of the surface bathed in profuse cold sweat. The expression is generally indicative of anxiety and distress, and the features pinched and shrivelled.

The patient as a rule retains his senses throughout his illness, and even up to the moment of death; but this event is often preceded by a period of quiescence or lethargy, and occasionally by slight rambling and partial unconsciousness. There is generally almost complete suppression of urine.

Enteritis in its most violent form is an extremely dangerous and indeed generally a very rapidly fatal malady. Death may occur within twenty-four hours, and is rarely delayed beyond a week.

Treatment.—The treatment of the milder forms of enteritis is so intimately connected on the one hand with that of inflammatory affections of the stomach, and on the other with that of diarrhœa and dysentery, that the reader may be safely referred to the articles on those subjects for all necessary details. As regards the treatment of the more severe forms of the disease, two main principles seem to be fairly well established:—namely, first, to relieve pain, and prevent as far as may be all movements of the bowels; second, to avoid every attempt (at least until all grave symptoms have ceased) to force the bowels by the administration of purgatives. Constipation, lasting for

a few days, or even prolonged for a week or two, in itself is generally a matter of very little consequence; it is, however, a matter of very serious consequence to intensify the pain from which patients are already suffering, to fret and irritate inflamed organs, and to subject to unwonted violence bowels unnaturally soft, enfeebled, and ready to undergo laceration. Clearly if patients are to get well, their recovery must in the first instance depend on the recovery by the diseased bowels of their healthy tone and capability of peristaltic action, and on the relief of pain and irritation. For these purposes, opium in large and frequent doses is generally our most valuable resource. No absolute rule can be laid down with regard to the quantity of this drug to be given at one time, or the frequency with which the dose should be repeated; the patient should, however, be got well under its influence and kept under its influence. For many reasons it is best administered by subcutaneous injection.

But our treatment need not be limited to the use of opium. The abstraction of blood is often of the greatest value. This is most efficacious early in the disease, and may be effected either by the opening of a vein in the arm, or by the application of ten, twenty, or thirty leeches to the surface of the belly. Warm but light applications, and hot fomentations, generally soothe; and sometimes mustard plaisters and similar mild counter-irritants give relief. In the same way enemata of warm water or gruel are at times useful. To relieve nausea and vomiting, ice, hydrocyanic acid, alkalies, lime-water, bismuth, carminatives and the like may be tried, and may be of much efficacy; but, when the vomiting is simply the consequence of over-distension of the bowels, as it sometimes is late in the disease, such remedies necessarily fail. The extreme prostration which so early manifests itself is a strong indication of the need of food and stimulants; but their exhibition by the mouth tends to increase distension, already probably painful, and to promote sickness; and under such circumstances they are little likely to be absorbed. It is obvious, indeed, that alimentary matters, if given by the mouth, must be given in very small quantities, and in a form suitable for their ready absorption. They are best administered in the form of enemata.

IV. ULCERATION OF THE STOMACH.

Causation.—The occurrence of excoriation or superficial ulceration in the course of ordinary gastritis has already been referred to. Such lesions have rarely, however, any special importance, and as a rule speedily undergo spontaneous cure. But the stomach is also liable to become the seat of ulcers, which tend to spread widely and deeply, are

productive of serious symptoms and sometimes of death, and the origin of which is to some extent enshrouded in mystery. These ulcers are seldom observed previously to the age of ten or fifteen, but subsequently to that period they seem to increase in frequency with advancing life, not, indeed, absolutely, but in relation to the numbers of persons living at each successive period. They appear to be two or three times more common in females than in males. They are often associated with amenorrhœa and anæmia, or chlorosis, and in both sexes (but more especially in men) with the cachexiæ which follow from habits of drinking and dissipation and from syphilis. It is possible that these conditions of the system may be the actual causes of the ulceration; it is more probable, however, that they tend to promote the spread and retard the healing of ulcers which have begun in the first instance independently of them. Virchow considers that ulcers originate mainly in affections of the vessels connected with the diseased areæ, especially embolism or degenerative change in the arteries, attended with arrest of circulation and necrosis, or obstruction of branches of the portal system of veins followed by interstitial hemorrhage. But it seems not improbable that the superficial ulcers which form in gastritis, and which as a rule readily heal, may under certain circumstances remain open, and be irritated into active enlargement. The progressive spread, and unwillingness to heal, of gastric ulcers, are readily explained by the constant irritation to which they are subjected by the ingestion of food, the pouring out of gastric juice and the movements of the stomach in digestion.

Morbid anatomy.—Gastric ulcers vary in size from that of a four-penny-piece up to that of the palm of the hand. The smaller ones are usually circular or oval in shape; the larger are more or less irregular, either from being formed by the coalescence of several smaller ulcers, or in consequence of irregular extension. When small an ulcer usually appears as if it had been made by punching out a bit of the mucous membrane. Its edges are more or less perpendicular; and the tissues entering into their formation are infiltrated, indurated, and probably thickened to some little distance around. Its floor may be smooth, flocculent, or even superficially gangrenous; and may be formed, according to the depth to which the ulcer has reached, by either the submucous tissue, the muscular coat, or simply the serous membrane. In an ulcer of large size the tissues which surround it are usually considerably thickened and indurated from inflammatory overgrowth, and often much congested; the edges, which are specially thickened, usually slope downwards to the floor of the ulcer, which thus becomes smaller than the superficial area of ulceration; sometimes, however, they are perpendicular; sometimes undermined, and overhanging. The floor of a large ulcer may be formed like that of a small one by any of the gastric tunics except the mucous membrane itself; but it may be formed also by the substance of the liver, pancreas, or any

other organ or tissue which has become adherent to the stomach, and involved in the progress of the ulceration. The floor may be smooth, irregular and flocculent, or sloughy, or may present granulation-like bodies due to the projection of the lobules of the eroded pancreas.

Gastric ulcers not unfrequently cicatrise. The surrounding thickening then diminishes, the sloping edges become undistinguishable, on the one side from the contiguous mucous membrane, on the other from the floor of the ulcer. The ulcerated surface contracts, radiating puckers form, and the central raw area grows smaller and smaller, and at length heals. The result is an opaque, whitish, smooth, tough, depressed area, surrounded by more or less obvious radiating folds of mucous membrane, and often attended with marked and it may be serious deformity of the stomach. It is not uncommon to find ulcers partly healed, or cicatrising at one part while undergoing extension elsewhere.

Unfortunately gastric ulcers do not always heal. In many cases they remain quiescent; in many they slowly extend; in many they end in perforation. Perforation sometimes takes place at once into the peritoneum; but sometimes the base of the ulcer previous to perforation becomes adherent to some neighbouring part, so that, while extravasation of the contents of the stomach into the peritoneal cavity is prevented, a communication becomes established with the transverse colon, or small intestine, with the pleura or lung through the diaphragm, or with the external air through the abdominal parietes. In other cases some artery—the splenic, coronary, gastro-epiploic, or one of their branches—or even the hepatic artery, or portal vein, becomes eroded, and profuse hemorrhage ensues.

Gastric ulcers are usually solitary, but occasionally two, three, or more are present at the same time. They may occur at any part of the stomach; but are more frequent in the pyloric than in the cardiac half, in connection with the posterior than the anterior wall of the organ, and in the neighbourhood of the smaller than in that of the larger curvature. Perforation is believed to be relatively more frequent in females than in males; and is a not uncommon termination of ulcer, especially in young women.

Symptoms and progress.—The symptoms which attend gastric ulcer present much variety. In a few cases the disease proves fatal by perforation or hemorrhage without having ever been attended with symptoms to attract attention to the stomach as the seat of disease. In most cases, however, the patient suffers from dyspeptic phenomena, of which the most common and characteristic are pain, vomiting, and hæmatemesis. As ulcer of the stomach is mainly a chronic disease, so the symptoms to which it gives rise generally assume a chronic character. They creep on for the most part gradually, probably sometimes intermitting for a while, often presenting exacerbations, but, on the whole, tending to become more and more pronounced.

At first possibly the patient complains of distension, flatulence and uneasiness, especially after food, and of impairment of appetite; but soon the uneasiness becomes pain; and sickness presently supervenes. The pain varies somewhat in intensity and character. It usually begins in, and may remain limited to, the epigastrium, which becomes tender on pressure; or it is referred to the region of the spine corresponding to the last two or three dorsal and first two or three lumbar vertebræ, or to the interscapular region—the muscles on either side often being tender; or it occupies the umbilicus, or some other point or area in the neighbourhood; and generally, when it is severe, it radiates from its point of chief intensity, upwards towards the œsophagus, backwards to the loins, or downwards and laterally over the greater part of the abdominal cavity. The pain, when severe, is of a burning, boring, or shooting character, often attended with a sense of soreness; it is aggravated by taking food, and in some cases occurs only then. It usually comes on a few minutes after ingestion, but is occasionally delayed until half an hour or an hour afterwards. It is doubtful how far the situation of the pain serves to indicate the situation of the ulcer; but both Dr. Budd and Dr. Brinton are inclined to believe that pain occurring chiefly in the pit of the stomach indicates the presence of an ulcer in the anterior wall of the stomach, and that pain in the back implies a corresponding situation for the ulcer. Further, Dr. Brinton regards the decubitus of the patient as suggestive in this respect—the patient lying as a rule on that aspect of the body which is farthest removed from the seat of ulceration.

Vomiting may be absent from first to last; it usually comes on, however, during the progress of the case, for the most part subsequently to the pain; and is then very persistent. The attacks are determined by the taking of food, usually come on a little later than the pain, and not unfrequently by emptying the stomach cause the pain to subside. The vomiting may be attended with violent spasmodic efforts, or may be effected in the manner of simple regurgitation. Hemorrhage is a frequent consequence of gastric ulcer—taking place sometimes from the congested mucous membrane which bounds it, sometimes from the general surface of the ulcer, sometimes from a vessel which has undergone erosion. In the last case especially the bleeding is apt to be very profuse, and to be repeated from time to time; and consequently large quantities of blood are vomited and subsequently passed by stool.

The long-continuance of dyspeptic symptoms, with pain induced by taking food and having the characters which have been described, and with vomiting coming on pretty constantly at some variable period after ingestion, is alone strong presumptive evidence of the presence of a gastric ulcer. And if to these be added the occurrence of profuse hæmatemesis there can be little room for doubt.

The most frequent termination of gastric ulcer is no doubt in con-

valescence. There is, however, a great tendency for healed ulcers to break out again, and consequently for patients who seem cured to have relapses. When the disease ends fatally, death may be due to simple asthenia—the patient sinking, worn out by the combination of long-continued pain, vomiting, and want of food; or it may be caused by the sudden loss of a large quantity of blood, or by the repetition, at longer or shorter intervals, of smaller but still copious hemorrhages; or it may result from perforation. When perforation takes place into the peritoneal cavity, sudden intense abdominal pain and collapse occur, speedily followed by general peritonitis; and the patient usually dies in from five or six hours to two or three days after the occurrence of the accident. When, however, perforation takes place into any of the hollow viscera or other cavities than that of the peritoneum, the symptoms which arise are usually much less sudden and grave, though still in many cases leading sooner or later to a fatal result.

Treatment.—Attention to diet is of the utmost importance in the treatment of gastric ulcer. The patient must be nourished; and yet all the digestive actions of the stomach are inimical to the cure of the lesion. We must consequently be especially careful as far as possible to avoid overloading the stomach or causing gastric pain, uneasiness, or vomiting. With this object it is important to administer as little food as is compatible with the maintenance of life, and to give it in small quantities at a time, and at short intervals; it is important also to select food of such a kind as will impart nourishment without causing undue irritation of the stomach; and, in reference to this matter, it may be observed that few articles of diet are so suitable as milk, which may be thickened, if necessary, with biscuit powder, arrowroot, or similar substances. Milk, however, sometimes disagrees, and then recourse must be had either to farinaceous substances mixed with water, or to animal broths and jellies. Liquids are generally ill borne when hot; and hence it is usually best to administer them tepid or cold. Hot tea and coffee especially are injurious. As the case progresses towards recovery eggs may be given, and tender, easily digested meats. Alcoholic stimulants should, if possible, be avoided; if given, they should be in a dilute form and cold. In some cases it is necessary to feed the patient for a time by means of nutrient enemata only. The chief medicinal agents which have been employed for the cure of ulcers are nitrate of silver, bismuth, the carbonated alkalies, and opium. It is certain that the combination of bismuth, in doses varying from ten to twenty grains, with opium is often very efficacious in relieving pain and vomiting and apparently in promoting the cure of the ulcer. Iron and the vegetable tonics are indicated when the more distressing symptoms have been relieved and the patient seems convalescent. When hemorrhage or any other serious complication occurs, special measures will be needed.

Counter-irritation and other external treatment applied to the epigastrium are often serviceable.

V. ULCERATION OF THE BOWELS.

Causation and morbid anatomy.—1. Intestinal ulcers are much more common, and various in character, than those of the stomach. Their causes for the most part are equally obscure. In many cases, no doubt, simple inflammation of the mucous membrane is followed by excoriation; which either rapidly heals and gets effaced, or, in consequence of continued irritation, becomes a veritable ulcer. Such ulcers may arise from simple mechanical irritation. They are roundish or irregular in form, vary in size, and present congested and well-defined margins, and irregularly excavated shreddy greyish surfaces. The margins and the surrounding tissues are in some cases considerably thickened and indurated, in others present little obvious departure from the normal state. Ulcers of this kind are not unfrequently met with in the duodenum, and in many cases are not improbably due to the same causes (whatever they may be) as the so-called 'chronic ulcers' of the stomach. They are also occasionally met with here apparently as the result of extensive superficial burns. The large intestine, however, is their most common seat; and they are produced here for the most part by the mechanical irritation of retained fæces or intestinal concretions. They are often found in the cæcum and its appendage, where such accumulations are very apt to form; but they may be developed at any part of the larger bowel. In cases of long retention of fæces, whether from simple constipation or from stricture, it is not rare to find the mucous surface studded with tracts, varying from one to many square inches in area, and consisting of groups of circular ulcers from half an inch in diameter downwards separated from one another by a network of congested and partly undermined bands of mucous membrane. Again, such ulcers may arise in any part of the intestine, whether large or small, from the effects of the passage or impaction of gall-stones or other solid bodies, especially when impaction occurs above a stricture or other such impediment.

2. In other cases ulceration is connected with the formation of a membranous pellicle; a linear, stellate, or irregularly polygonal patch of mucous membrane becomes congested and swollen, and soon covered with an opaque whitish or buff-coloured exudation, which is friable and granular, and extends by rootlets into the Lieberkühnian follicles. This, after a time, separates, leaving sometimes a sound surface, sometimes a slight excoriation, or even a distinct ulcer, with a

grey ish or yellowish floor and a well-marked margin of congestion. These ulcers may be met with in any part of the bowels, but are much more common in the large than elsewhere. In the small intestine they affect chiefly the free edges of the valvulæ conniventes; in the large, either the projecting ridges formed by the intervals between the sacculi, or those corresponding to the longitudinal muscular bands. Sometimes we find extensive tracts of congested bowel studded or intersected with patches or bands of membranous exudation, or consecutive ulceration, or both intermingled. This condition is met with under various circumstances: especially perhaps in pneumonia, and many chronic affections, such as Bright's disease, cirrhosis of the liver, cancer and chronic phthisis.

3. Sometimes ulcers originate in foci of submucous suppuration, as occurs in pyæmia, or in patches of deep-seated sloughing like ordinary boils. Among the latter may perhaps be reckoned the ulcerative inflammation of the follicles of the colon, which Rokitansky describes, and is believed to constitute the early stage of dysentery. The follicles enlarge to the size of a tare or pea, become surrounded by a halo of congestion, and then undergoing suppuration form each an ulcerated opening, which eventually enlarges and constitutes a circular ulcer, with overlapping edges.

4. Ulceration may be due to the formation and detachment of a superficial slough. Circumscribed patches of intense congestion or extravasation appear in the substance of the mucous membrane, which, shortly dying, come away bit by bit, or in mass. The above process is often effected with little obvious change in the immediately surrounding parts, and the resulting pits are for the most part speedily effaced. This affection is not uncommon in small-pox, typhus, and other such diseases. It frequently involves only the valvulæ conniventes, or the corresponding projections of the large intestine. It may be due to sudden arterial obstruction.

5. But sloughing, to a much more serious extent, is sometimes met with, especially in the large intestine: patches of mucous membrane become livid, brown, or nearly black with congestion; and their central areæ assume a grey or ashy colour, get shrunken, depressed and softened, and break down into a soft, shreddy substance, which partly becomes detached, and partly adheres to the floors of the excavations and to their not yet broken-down edges. The process tends to spread.

It is not pretended that all non-specific ulcers arise in one or other of the modes here enumerated, or that they necessarily maintain in their ulterior progress the distinctive characters of their origin. Yet, independently of their exciting causes, and early peculiarities, all ulcers are apt after a time to present certain common varieties of appearance, dependent mainly on the processes which are actually taking place in them. Thus when they are healing, we find the general surface smooth and clean or granulating, the edges little thickened or

congested, perhaps puckered, and probably sloping more or less obviously to the ulcerated area, with which they are in fact continuous; when they are sluggish, the edges are more or less tumid and rounded, probably overhanging, and the general surface smooth; when they are spreading, the surrounding mucous membrane presents more or less intense congestion and swelling, and the immediate margin is either flocculent and ash-coloured, or presents a vivid red, raw, bleeding wall, or forms a more or less complete ring of distinct gangrene, and the floor is irregular and flocculent. The base of an intestinal ulcer is generally constituted by the submucous tissue, but not unfrequently the transverse muscular fibres are exposed; and when an ulcer tends to perforate the bowel the muscular coat itself becomes opaque, softened, and in part destroyed.

The above account applies mainly to individual ulcers. But very often, and much oftener in the large than in the small intestine, many ulcers are present at the same time, and tend to increase either in number or in size or in both of these respects, and to coalesce. And then, according to the stage to which the lesion has advanced, we meet in different cases with: either a number of ulcers separated from one another by an imperfect network of mucous membrane; or interlacing networks of ulceration and mucous membrane; or islets of mucous membrane in an expanse of ulceration; or, lastly, extensive tracts from which the mucous coat has been wholly removed. In these cases the transverse muscular fibres are often freely exposed, and the remains of mucous membrane are red, swollen and rounded, and in the form of tubercle-like excrescences. The affected bowel, moreover, is frequently much contracted, and the muscular walls hypertrophied.

This is not the place to discuss the important subject of specific ulceration of the bowels. Yet specific ulcers constitute by far the most formidable class of intestinal ulcers. The more important of them are the following:—First, syphilitic ulcers; these have not been certainly recognised in the alimentary canal excepting in the neighbourhood of its inlet and outlet; syphilitic ulceration of the rectum is a well-recognised, and for the most part very intractable, lesion. Second, the ulcers of enteric fever: these affect mainly Peyer's patches, and are most abundant and large in the lower part of the ileum; they not unfrequently involve also the solitary glands of the large intestine, especially in its upper part. Third, tubercular ulcers, which originate for the most part in the same glands and situations as enteric-fever ulcers. And, fourth, the various kinds of ulcer due to the breaking down of carcinoma and other varieties of malignant disease.

Many intestinal ulcers cicatrise and leave behind them little or no trace of their existence. In other cases, however, and indeed in a large proportion of them, results of more or less serious importance follow. Sometimes, as we see in the rectum, when a vast continuous surface has been destroyed, the wound never heals; and, even in cases where

the destruction has been much more limited, the ulcer may assume the characters so often presented by chronic ulcer of the stomach, and be ready, if it cicatrises, to break out again and again. But generally, when a large ulcer heals wholly or in part, especially if it has involved the whole circumference of the bowel, some degree of contraction of the bowel, or stricture, results. In many cases hemorrhage takes place either from the congested surfaces or margins of ulcers, or from vessels perforated in their progress; and such hemorrhage may be so frequently repeated, or so abundant, as to prove fatal. In many cases, also, perforation of the bowel takes place. This accident is usually due to a sudden tear in the floor of an ulcer, which has got unusually thin, and undergone softening, or become weakened in some other way; and not unfrequently depends immediately on some violence inflicted from without, or some undue pressure from within, such as may result from over-distension, or violent peristaltic movement. The rupture usually takes place at once into the peritoneal cavity, causing extravasation of fæcal matter and generally fatal peritonitis. But not unfrequently inflammation arises on the peritoneal aspect of the ulcerated bowel; adhesion takes place between it and some neighbouring viscus; and consequently the threatened perforation becomes, for a time at least, averted. In many cases, a communication becomes established between the bowel and some neighbouring hollow organ—a result preceded either by the formation of adhesions or by the development of a circumscribed abscess between the two organs. The latter mode of communication is especially liable to take place when the ulcer opens on the mesenteric aspect of the small intestine, or in the corresponding part of the larger bowel, and consequently into the connective tissue, with which the bowel is in these situations closely invested. Thus, we occasionally find contiguous portions of the small intestine communicating with one another, or small intestine with the transverse or some other part of the colon; the rectum, sigmoid flexure or ileum with an ovarian cyst, the urinary bladder, or vagina; the duodenum or transverse colon with the gall-bladder; the stomach with the transverse colon; or, lastly, almost any part of the intestinal canal with the external surface.

Symptoms and progress.—The symptoms of ulceration of the bowels are so constantly associated with those of the different morbid states of the system on which it depends, or those due to the various complications which follow upon it, that we seldom have the opportunity of studying them in their simple form. It may be stated generally: that ulceration of the bowels is often attended with more or less obvious febrile symptoms, which assume, if the disease become chronic, a distinctly hectic character; that the affected bowel is often more or less tender on pressure—a characteristic which is especially observable if the ulceration be extensive, or occupy the cæcum or some other part of the large intestine; that there is almost necessarily some im-

pairment of nutrition marked by emaciation, debility, and feebleness of circulation; that there is more or less abdominal soreness, aching, or griping; and that, above all, there is something abnormal in the action of the bowels and in the evacuations. The symptoms will vary according to the seat of disease. If the ulcer be high up, and especially if it be in the duodenum, the symptoms will approximate to those of gastric ulcer; there will probably be pain coming on some time after food, and vomiting, but no material interference with the function of defæcation. If the ulceration occupy the central portion of the small intestine, there may be nothing beyond gradually increasing emaciation, and occasional colicky pains, to indicate that the bowels are affected; and indeed extensive ulceration may be present even in the lower part of the ileum without occasioning any obvious modification of the stools; there may, indeed, be constipation from first to last. Usually, however, if there be ulceration in the last-named situation, and especially if the large intestine be involved, more or less diarrhœa may be looked for. The stools are then generally liquid, and contain an abnormal quantity of the fluid secretions of the bowels, and not unfrequently more or less blood; they are, moreover, often pease-soup-like in colour and consistence, and more offensive than in health; further, they are usually passed much more frequently than natural, and the patient suffers from frequent colicky pains and tenesmus. As the ulceration approaches nearer and nearer to the lower part of the large intestine, the evacuations assume more and more of the so-called 'dysenteric' character. They are then passed with extreme frequency and great tenesmus; are scanty, mucous, often sanguinolent, and not unfrequently entirely free from true fæcal matter. The latter may be only passed occasionally in small hard lumps, invested in mucus; indeed, constipation, so far as regards the passage of fæcal matter, is often one of the most troublesome and distressing symptoms of ulceration of the rectum and lower part of the colon. It is in the dysenteric form of the disease, moreover, that the evacuations become most offensive, the fetor sometimes being putrid and almost insufferable. Besides the slight oozing of blood which tinges the evacuations in dysenteric diarrhœa, hemorrhage to a considerable amount sometimes takes place; and this may be either continuous or recurrent, and sufficient to destroy life. Many of the communications which have been described as taking place between the intestine and other organs as a result of ulceration are doubtless of little practical importance; but some are dangerous, or present features of clinical interest. Among the latter may be especially mentioned: communications between the stomach or duodenum and the colon, which lead to the occasional or constant vomiting of actual fæces and to the escape of undigested food into the large intestine; and communications with the urinary bladder, which occasion the escape of flatus and fæces into that viscus, with other consequences which are easy

to imagine. Rupture into the peritoneum generally causes fatal peritonitis.

Treatment.—Our aims in treating ulcers of the bowels should be: first, to promote the healing of the ulcers and prevent as far as possible the local mischances which are apt to follow; second, to check abdominal discomfort and diarrhœa; and, third, to support the patient's strength. It is of course doubtful how far remedies given by the mouth can act locally on ulcers low down in the bowels, and how far, therefore, substances like bismuth, nitrate of silver, iron, copper, mineral acids, and the like can promote cicatrisation; still they are often employed with this object, and sometimes apparently with benefit. But it is of great importance that the bowels should be kept at rest, and violent peristaltic movement as far as possible restrained. Purgatives, therefore, should be in great measure, or wholly, eschewed; while astringent medicines—iron, copper, lime, chalk, tannic acid, or vegetable astringents—will probably prove serviceable. Opium is especially valuable; and the compound kino powder, and the combination of aromatic chalk powder with opium, are useful preparations. It is important, however, to note that opium cannot always be taken in these cases; for chronic ulceration of the bowels is often attended with an irritable state of the mucous membrane of the mouth and stomach which the use of opium is apt to augment. If this drug cannot be employed, it may be replaced to some extent by other sedatives, such as hyoscyamus, belladonna, Indian hemp, or hydrocyanic acid. Opium may often be given with advantage in the form of enema or suppository. It is obvious that the various measures which have just been enumerated, while they check peristalsis, act with equal efficacy in fulfilling the second indication of treatment—namely, the arrest of diarrhœa. The maintenance of the patient's strength must be effected by the use of tonic medicines and the careful administration of suitable food and stimulants. The form of tonic must be adapted to the special requirements of the case, and to the other details of treatment it may be considered necessary to adopt. As regards food, this should be well-cooked, well-masticated, easy of digestion, given in moderate quantities, and at regular if not frequent intervals. Farinaceous foods are in many cases most suitable; but eggs, fish, and fowl may often be used with advantage. Butcher's meat is sometimes wholly inadmissible.

VI. PERFORATING ULCERS OF THE CÆCUM AND RECTUM.

(*Typhlitis, Perityphlitis, and Periproctitis.*)

There are certain parts of the bowels which are especially liable to become the seat of non-specific forms of inflammation and ulceration,

or to be involved in inflammation originating in their neighbourhood: these are the duodenum and the large intestine—more particularly the cæcum and lower part of the rectum.

As to the duodenum, we have already pointed out that it is not unfrequently the seat of ulcers which resemble chronic ulcers of the stomach, and of ulcers arising in connection with extensive burns of the skin. We may add that from its situation and attachments it is liable to become perforated from without by abscesses of the gall-bladder and liver, and by abscesses originating, no matter how, in the upper part of the retro-peritoneal tissue.

So, also, the large intestine, from its peculiar relations with the peritoneum, and from the extent to which it is in many places devoid of peritoneal covering, and continuous, therefore, with the sub-peritoneal connective tissue, and thus brought into almost immediate connection with the various organs lying beneath the parietal peritoneum, is peculiarly apt to be involved in extraneous inflammation and suppuration. For similar reasons (at least in great measure) inflammation originating here is very liable to induce inflammatory thickening and abscess in the surrounding tissues.

A. *Typhlitis. Perityphlitis.*

Causation and morbid anatomy.—The terms 'typhlitis' and 'perityphlitis' (the former signifying inflammation of the walls of the cæcum, the latter inflammation of the tissues surrounding it) are commonly employed in reference to those cases in which inflammation of the cæcum or its vermiform appendage involves, either by perforation or by simple extension, the connective tissue of the iliac fossa or the peritoneal cavity. Ulceration of these parts very frequently takes place (in enteric fever and phthisis to wit) without causing the special phenomena of typhlitis. There is reason, indeed, to believe that in most, if not all, cases where inflammation spreads from the cæcum to the surrounding tissues, its spread is referrible to ulcerative perforation. The causes of the lesion in question are no doubt various. It may be due to the extension of tubercular, typhoid, or dysenteric ulcers, to simple but extreme distension of the cæcum, to the fretting of its surface by accumulated fæcal contents, to the mechanical effects of bristles, pins, or bits of bone which have been accidentally swallowed, or to the lodgment of intestinal concretions. Concretions are mostly found in the vermiform appendage and are the usual causes of perforative ulceration of this part. They vary from the size of a pea to that of a date-stone, are sometimes of a waxy consistence and lustre, sometimes brown, opaque, laminated, and for the most part fæcal, sometimes composed mainly of earthy phosphates, but consist in all cases of an admixture in unequal proportions of ordinary fæcal matters and the secretions from the mucous surface, and are occasionally developed around small extraneous bodies.

In some cases the ulcer perforates that portion of the bowel which is devoid of peritoneal covering. Fæcal matter then escapes into the surrounding tissues, leading to more or less extensive inflammation and induration. If the escape be slight inflammatory swelling alone may take place, and after a while subside. Often, however, an abscess forms, which enlarges more or less rapidly, and then extends in a direction determined in great measure by its original seat: in one case descending into the pelvis and opening into the rectum; in another passing out with the pyriformis muscle and presenting in or below the buttock; in another forming a swelling in the groin immediately above Poupart's ligament, or passing along the inguinal canal into the scrotum, or along the psoas and iliacus muscles into the upper part of the thigh. In most cases no doubt it presents itself in the iliac region superficial to the position which the cæcum normally occupies. An abscess of this kind may get cured by discharging its contents either through the orifice in the cæcum which gave rise to it or through an opening at any one of the spots which have been enumerated; or, burrowing extensively, it may form a sinus or series of sinuses which are never obliterated. The communication between the abscess and cæcum is sometimes maintained; at other times it closes more or less speedily, and the abscess appears henceforth to be independent of the bowel. In some cases (especially if the part affected be the vermiform process), local peritonitis precedes or accompanies the perforation, which would otherwise have been direct into the general peritoneal cavity; and a circumscribed abscess forms, the indications and progress of which differ little, if at all, from those of the abscesses previously considered. In other cases rupture takes place directly into the peritoneal cavity and fatal peritonitis is excited. It may be added that the circumscribed abscesses themselves may rupture ultimately into the peritoneum.

The most common form of fatal typhlitis is that connected with perforation of the vermiform appendix—an accident which occurs mainly in early life, and apparently oftener in males than in females.

Symptoms and progress.—The symptoms of typhlitis are, in the first instance, pain, tenderness, and swelling in the region of the cæcum, together with signs of inflammatory fever, and sometimes rigors. The local symptoms are for the most part those which may be caused by inflammation of whatever origin occupying the venter of the ileum. If an abscess forms, but extends downwards into the pelvis, or remains deep-seated, the case is naturally obscure. If, however, it tends to point anteriorly, the fulness and hardness get more and more pronounced, and gradually develop into a fluctuating hemispherical protuberance over which the integuments become œdematous and congested. Sometimes, even at this stage, the swelling gradually subsides and disappears, owing to the abscess having discharged itself into the bowel; but more frequently it still enlarges and ultimately

opens externally, discharging a greater or less amount of fetid pus, sometimes having a fæcal odour, sometimes containing fæcal matter and bubbles of gas. The further progress of the case may be towards either more or less speedy recovery, or the formation of successive abscesses or fistulæ, or the establishment of an artificial anus. When peritonitis arises from perforation of the cæcum or its appendix, its occurrence may be quite sudden and unpreceded by any form of premonitory symptoms; but occasionally it is heralded by localised uneasiness or pain, or (as we have pointed out) supervenes in the course of well-marked perityphlitis.

The functions of the alimentary canal are by no means necessarily disturbed to any great extent in typhlitis. Sickness is often absent. Constipation is not unfrequently present during the earlier period of the disease; while diarrhœa is apt to supervene at a later stage. But none of these symptoms has any particular uniformity or value. It may be remarked that, from the close proximity of the cæcum to important veins and nerves, typhlitis is apt to induce painful neuralgic symptoms and œdema of the right lower extremity. Its duration is necessarily very uncertain. Sometimes the patient speedily recovers, sometimes he lingers indefinitely with a constantly discharging abscess or a succession of abscesses. If, however, perforation take place into the peritoneum death rapidly follows.

Although inflammation beginning in the cæcum is a very common and important cause of inflammatory swelling and suppuration in the right iliac fossa; it must not be forgotten that this part is also a common seat of inflammation and abscess from other causes, and further, that such abscesses are liable to form communications with the cæcum, and hence still further to simulate primary typhlitis. Among the affections here referred to may be enumerated: inflammation of the ovary and connective tissue in its neighbourhood; idiopathic abscesses of the venter ilii, or in the course of the psoas muscle; psoas abscess from caries of the spine; renal abscess; and all descending retro-peritoneal abscesses, whether from the interior of the spinal canal, the pleura, lung, or liver.

Treatment.—The treatment of typhlitis is in principle, and indeed in most of its details, the same as that of enteritis and other forms of intestinal ulceration. It consists mainly in keeping the bowels quiet by the aid of opium, and in the use of local applications. It is almost more important in typhlitis than in any other affection to avoid opening medicines; for, especially if the disease be in the appendix, rupture into the peritoneum is in many cases prevented solely by slight adhesions. This danger often continues, indeed, for some time after the local inflammation seems to have subsided; and caution, therefore, should be exercised in respect of the use of purgatives for some time after apparent restoration to health. If the bowels need to be relieved simple enemata are the safest agents, and are usually sufficient. The

local measures to be employed comprise leeching, fomentations, and the application of ice; and if an abscess form, its speedy evacuation. Those who have once suffered from typhlitis are liable to recurrences of the disease, and require to take great care in respect of diet, exposure to cold, and other conditions likely to act injuriously.

B. *Periproctitis.*

Causation and morbid anatomy.—Inflammation and suppuration about the lower part of the rectum are even more common than the corresponding affections of the cæcum; and their causes are equally various. In many cases this affection is traceable to ulceration (perforative or other) of the mucous membrane; in others it probably originates in the connective tissue which surrounds the rectum. Further, the rectum (again even more frequently than the cæcum) gets involved in inflammation and suppuration originating in the various pelvic and even distant organs. Abscesses, in fact, arising in the abdominal cavity or its walls, or implicating them, are peculiarly apt to gravitate into the pelvis, and to communicate with the rectum. Rectal abscess is frequently connected with the presence of tuberculosis.

Symptoms and progress.—Inflammation in the neighbourhood of the lower part of the rectum necessarily produces tumefaction and induration, which may usually be readily detected by digital examination per anum, or by their presence in the perinæum in the immediate vicinity of the anus. In connection with the swelling there are always more or less severe pain and tenderness, which often prevent the patient from sitting down, and are greatly aggravated during the act of defæcation. If suppuration take place, the swelling rapidly increases in size, and the abscess presently opens either into the rectum (usually a little within the internal sphincter), or externally by the side of the anus, or in both of these situations, and discharges exceedingly fetid pus. Simple inflammation around the rectum may subside spontaneously; but an abscess almost invariably results in the formation of a fistula which is a peculiarly obstinate affection, and rarely yields excepting to direct surgical treatment. When an abscess opening into the rectum is connected directly with suppuration of some remote organ, the ultimate prospects of recovery are by no means satisfactory.

The *treatment* consists in the application of fomentations, poultices, or leeches, and the opening of the abscess as soon as the presence of pus is ascertained. The bowels, moreover, should be regulated either by laxatives or by enemata.

VII. DYSENTERY.

Definition.—We have already, in describing inflammation and ulceration of the bowels, discussed the various inflammatory processes which take place in the large intestine, and considered the symptoms to which they give rise. These affections, especially if they involve its lower segment, always induce so-called 'dysenteric' symptoms, and are usually included in the generic term 'dysentery.' But dysentery is also the name of one of the most widespread and fatal of diseases—a disease which, under special circumstances, assumes an endemic or even epidemic character, and is hence not unnaturally regarded as a specific disease, in the same sense as ague and enteric fever are specific diseases.

Causation.—Dysentery prevails largely in tropical regions, and more especially in those places which are low and swampy, and surcharged with decaying vegetable matter—in regions indeed which are, for the most part, malarious and breed intermittent fevers. It occurs, however, under conditions and in places which are not productive of ague; it has been in all ages one of the greatest scourges of armies in the field, of beleaguered cities, and of starving populations. According to Sydenham and others of our older writers, it was once a formidable disease in this country; whence in an aggravated and epidemic form it has now almost entirely disappeared. It is probable, however, that enteric fever formed a large proportion of the cases then termed dysenteric. From its frequent coincidence in area of distribution with ague it is by many regarded as being, equally with that disease, a product of the malarial poison. But the facts that aguish districts are not necessarily also dysenteric; that dysentery, even in an epidemic form, occurs in places and under circumstances which never yield ague; and that ague and dysentery no more graduate into one another than do enteric and typhus fevers, render this view of its origin untenable. The influences of foul water, polluted air, insufficient nourishment, and exposure and over-fatigue in its production are unquestionable, but whether as exciting causes or merely as predisposing causes is by no means clearly established. There is reason, however, to believe that polluted drinking water is an especially active agent in the induction of the disease, but whether by the introduction of a specific poison is, at least, doubtful. We are inclined to regard dysentery as both of non-specific origin and non-infectious; and, on these grounds, introduce its description here.

Morbid anatomy.—The morbid anatomy of dysentery has been abundantly described, but the descriptions which have been given of it are various, and do not admit of being readily reconciled. Some

of the most trustworthy of recent observers, such as Parkes and Baly, regard it as a disease essentially of the solitary glands of the large intestine, which rise up in the form of hemispherical buttons, varying from the size of a millet-seed downwards, and occasionally attaining the bulk of a split pea. Associated, however, with glandular hypertrophy there is always more or less intense congestion of the general surface of the mucous membrane, which becomes sepia-coloured, reddish-brown, or almost black; together with inflammatory infiltration of its substance and of the submucous tissue, which may consequently acquire a collective thickness of one-quarter or even one-third of an inch.

It must, we think, be admitted that dysentery commences with congestion, more or less intense, and infiltration, more or less conspicuous, of the mucous membrane, in which changes the solitary glands not improbably take a predominant share. This inflammation (at all events in the first instance) usually occurs in scattered patches, which are linear, stellate, or irregularly roundish or polygonal, are peculiarly liable to involve the prominent folds, and are sometimes limited to them. The patches may be discrete, or they may run together, forming an irregular network, or they may coalesce completely over a more or less extensive area, and even throughout the whole length of the large intestine. It usually happens that, in addition to the interstitial inflammatory changes [illegible]re adverted to, the affected surface becomes early covered with a thin, opaque, granular film, or with such films in patches. These can usually be readily removed from the subjacent surface, bringing with them adherent casts of the Lieberkühnian follicles. They consist, in fact, mainly of an inflammatory overgrowth of the intestinal follicular epithelium.

If the dysenteric attack be slight, the morbid process may cease at this point, and convalescence become established without any material injury to the bowel. But if it be severe, further changes speedily ensue. These present considerable variety, but consist essentially in the formation of sloughs and (by the separation of these) of ulcers. The sloughs vary in colour, size, shape, and arrangement. They may be yellow, like those of enteric fever, or ash-coloured, or black. They are sometimes circular and distinct, studding the surface more or less uniformly and thickly; sometimes they occur in irregular groups, and constitute patches of various, and often considerable, extent; sometimes they so run together and are so arranged as to constitute a network the interstices of which are formed by isolated patches of mucous membrane; sometimes extensive tracts of surface are uniformly and completely destroyed; and in all cases there is more or less tendency for the morbid process to spread, either by simple ulceration, or by the burrowing of pus beneath the mucous surface, or by sloughing. With the separation of the sloughs ulcers are left, sometimes with ragged, sometimes with abrupt, and often with swollen and congested

margins, and with floors formed either by the submucous tissue or by the transverse muscular fibres.

The subsequent progress of the morbid process varies. In some cases more or less perfect cicatrisation ensues; in some, the ulcers assume a chronic character, and remain open, and with little alteration, for an indefinite period; and in either of these cases there is a tendency to the recurrence of active inflammation under slight provocation. When the disease lapses into the chronic form, the affected bowel is apt to remain exceedingly irritable, to become permanently contracted, and as regards its muscular coat sometimes greatly hypertrophied. It must be added: that perforation of the bowel is an occasional complication of dysentery; that inflammation sometimes pervades the whole thickness of the intestinal walls, extending even to the peritoneal aspect; that more or less hemorrhage from the inflamed or ulcerated surface is almost invariable, while in some cases it is so abundant as to cause death; and that the cicatrisation of dysenteric ulcers not unfrequently causes stricture.

Dysenteric inflammation may occupy any part of the large intestine, or the whole of it, and may be prolonged for a considerable distance up the ileum. It is most common, however, in the lower part of the colon and in the rectum, and is usually most severe and most advanced in these situations.

Other lesions besides those of the bowels are often met with in dysentery. The most common are engorgement of the lymphatic glands in relation with the inflamed mucous membrane, and congestion of internal organs, more especially of the liver, spleen, kidneys, and lungs. In association with the dysentery of tropical climates abscess of the liver is not uncommon. This complication is referred by Dr. Geo. Budd to portal pyæmia, taking its rise from the diseased mucous membrane of the bowel. Hepatic abscess, however, sometimes originates simultaneously with the dysentery, sometimes precedes it; and hence it seems more probable that the two lesions are concurrent effects of the same cause, and not dependent the one on the other.

Symptoms and progress.—The symptoms of dysentery comprise those of pyrexia and those due directly to the morbid processes going on in the large intestine—the latter being mainly determined by the excessive irritability and tendency to spasmodic contraction of the larger bowel, and by the fact of the constant discharge into it of the morbid products of the diseased mucous surface.

In the milder forms of the disease, the patient, after suffering, perhaps, for a short time from more or less heat and dryness of skin, clamminess of mouth, and vague griping pains, is attacked almost suddenly with an uncontrollable impulse to evacuate his bowels, and probably passes a solid motion with unusual ease—the mass being invested in a greater or less abundance of greyish or colourless mucus.

The usual sense of relief, however, does not follow, and he probably finds himself compelled to sit straining at stool, with fits of spasmodic violence, during which he discharges small quantities of offensive mucus, and probably a minute fæcal lump or two. With the continuance of the affection the febrile disturbance continues; the tongue probably becomes coated; a constant sense of uneasiness, heat, or burning pervades the anus and adjoining parts of the rectum, and more or less, perhaps, of the rest of the large intestine. The patient suffers from frequent tormina, and impulse to evacuate the bowels—the efforts being attended with much tenesmus, and the discharge mainly of small quantities of mucus. This may be stained with fæcal matter, and is often intimately mixed with blood, and may consequently present very much the appearance of pneumonic expectoration. But, notwithstanding the almost constant efforts at defæcation, there is, so far as actual fæcal matter is concerned, almost complete constipation. A few scybala only are passed from time to time. Cases of this kind may subside in the course of a day or two, and seldom last longer than a week or ten days. Nevertheless some irritability of the bowels, uneasiness after defæcation, and tendency to constipation, may trouble the patient for a considerable time after he seems to have regained in other respects his ordinary good health.

In the more severe forms of dysentery the symptoms are similar in kind, but much more intense. The disease is usually ushered in with high fever, often with alternate chills and flushes of heat, sometimes with distinct rigors, and occasionally even with convulsions. The skin is hot, the pulse accelerated; there are febrile pains and headache, anorexia, thirst, and dryness and furring of the tongue. In this, as in the former case, the intestinal affection is usually first indicated by the occurrence of griping pains, which are presently followed by the evacuation of the contents, often solid, of the lower bowel. But very soon the griping becomes frequent and severe, calls to stool are incessant, and the patient suffers from almost constant tenesmus. The matters discharged from the bowels are at first a whitish, brownish, or olive-coloured glairy or jelly-like mucus; but this soon gets sanguinolent, and not unfrequently intermingled with considerable quantities of dark and more or less clotted blood. After a while the discharges commonly assume those characters which give them a resemblance to 'meat-washings'; they become thin, watery, turbid, reddish, and dirty-looking, and contain brownish or blackish particles, which are fragments either of altered blood-clots or of sloughy mucous membrane. It is at this time also that the patient frequently passes soft membranous pellicles, which are either tracts of mucous membrane detached in bulk, or portions of false membrane. Dysenteric evacuations are further characterised: by a peculiar and almost insupportable fetor, which increases in intensity with the supervention of sloughing; by containing a large quantity of dissolved albumen; and by the occa-

sional presence of small solid fæcal lumps or scybala. They sometimes become purulent. The frequency with which the bowels act is often very remarkable. In some cases the patient seems for a length of time never to cease discharging small quantities of fluid. The bowels are often relieved four or five times in the hour, and sometimes as many as ten or twenty times in the same period. The quantity of fluid passed, however, is not necessarily in relation with the frequency with which the bowels act. In many cases, especially at the beginning, the discharge is scanty; but later on considerable quantities of serous fluid, or blood, or both, are apt to escape, and the total bulk of these discharges in the twenty-four hours is hence often very large.

Associated with tenesmus and alvine flux are burning pain within the anal orifice, and a constant sense of the lodgment there of something which needs to be got rid of; there is also more or less burning pain and tenderness on pressure in the course of the large intestine and especially of those parts which are chiefly involved. At first probably the abdominal parietes are rigid and retracted; but before long flatus accumulates and the abdomen consequently gets enlarged and tympanitic; the tongue becomes thickly coated; the patient complains of thirst, loathes food, and not unfrequently suffers from nausea and vomiting; the urine is scanty and high-coloured, and its discharge sometimes attended with pain or difficulty; the febrile excitement which ushered in the disease rapidly gets replaced by a condition of profound depression; the skin may yet be hot and dry, but the pulse becomes small, feeble, and rapid, the face anxious, and the patient restless, sleepless, and desponding.

Cases which end favourably usually manifest signs of amendment from the sixth to the tenth day; these consist in abatement of fever and other general symptoms, and gradual cessation of tenesmus and the peculiar dysenteric stools. But convalescence is usually much protracted; and some time elapses before the bowels completely regain their normal tone. In those cases which end fatally the pulse increases in rapidity, loses fulness and power, and often becomes scarcely perceptible; the surface tends to grow cool; the face and extremities acquire a shrunken and dusky aspect; the tongue becomes dry and brown or black; hiccough and vomiting come on; and the abdomen grows more and more tympanitic. Although probably continuing restless and desponding, the patient often retains his senses perfect to the last; sometimes, however, he becomes delirious (in some cases, indeed, delirium comes on early), and he may then pass into a state of stupor or coma. It very commonly happens that, with the increase of tympanites, the abdominal pain, colic, and tenesmus all subside and even disappear wholly. The symptoms which precede death, and the mode of death, are necessarily modified to some extent by the special circumstances of the case; they are, for example, somewhat different in such cases as are attended with profuse hemorrhage from what they

are in those in which intestinal perforation takes place, or which are complicated by hepatic abscess, or where the patient sinks under the influence of the uncomplicated disease. Under all circumstances, however, the immediate cause of death is asthenia.

Many cases of acute dysentery, instead of taking either of the two courses which have been considered, become chronic; and the disease continues, with occasional remissions and exacerbations, for an indefinite period. The patient is then an almost constant sufferer from colic and tenesmus, and the discharge of offensive liquid stools containing little true fæcal matter, and from retention, often to a very uncomfortable extent, of his solid fæces; he complains of abdominal tenderness and uneasiness; his tongue is in some cases dry, glazed, and fissured, in others coated, in others almost normal; and his appetite presents equal variations; more or less sickness is often present; and he becomes emaciated, weak, anæmic, anasarcous, and often hectic. If an hepatic abscess be present, the symptoms, or many of them, are aggravated, and probably the indications of hepatic tumour are presently superadded. Chronic dysentery varies greatly in its severity, and in some cases, even though lasting for years or throughout life, is, excepting from the discomfort which attends it, of comparatively little importance.

As a rule, sporadic dysentery is not a very fatal disorder; but the epidemic form is usually attended with a high mortality; and although, even here, the ratio of deaths to attacks is sometimes small, the cases are so numerous and the total mortality usually so high, that it is justly regarded as one of the most fatal of epidemic diseases.

Treatment.—There is little unanimity of opinion with regard to the treatment of dysentery; some authors strongly advocate the copious abstraction of blood, if not by venesection, at any rate by leeches; some place their chief reliance on calomel in large doses; some regard ipecacuanha as almost a specific; some pin their faith to purgatives, some to opiates; while, on the other hand, each of these remedies has been more or less strongly condemned. Of the immediate relief which follows the abstraction of blood there is probably little doubt; but it is obvious that the marked tendency to asthenia which exists in dysentery supplies a powerful argument against the indiscriminate and excessive use of blood-letting. As a rule, it is doubtless unnecessary; but if employed it should be employed early, and preferably effected by the application of leeches to the tender regions of the abdomen. Calomel has been administered (as it was formerly in cholera) in large doses with reputed success; it has, however, fallen into disuse, and probably deservedly. Ipecacuanha has enjoyed a long but various reputation. It was formerly regarded as an almost unfailing specific, and at the present day is very highly esteemed. There are at least two antagonistic principles on which it is administered. By Trousseau and other French authorities it is given in doses of ten or twelve grains of the powder every ten minutes or so, until copious vomiting results—the

essence of the treatment being, according to them, the production of a powerful evacuating effect upon the stomach; by English army surgeons, on the other hand, it is recommended to be given in a large dose (twenty-five to thirty grains), which is to be repeated at the end of eight or ten hours; but it is to be given, guarded by opium, and with every precaution against sickness, in order that the remedy may act directly, or indirectly through the system, on the affected mucous surface. Bretonneau advocated the use of saline purgatives in large doses, and in this advocacy he is strongly supported by Trousseau. Opium and astringents are often employed; but the former (except in infinitesimal doses) is strongly condemned by the last author.

It may, we think, be fairly asked whether there are any good grounds for believing that dysentery is more amenable to treatment, specific or non-specific, than other forms of enteritis are; and whether there is sufficient reason for adopting a radically different treatment from that which has been found generally useful in enteritis? In acute and severe cases we should be disposed in the first instance: to apply hot fomentations to the belly, and if there be much local pain and distress, to abstract blood by means of ten, twenty, or thirty leeches; to exhibit opium, or opium with ipecacuanha, in doses sufficiently large or sufficiently frequently repeated to relieve the tormina, tenesmus, and abdominal pain; and to use enemata either simply to wash out and cleanse the lower bowel, or to soothe it, or for the purpose of applying astringent or other medicaments directly to its surface. We should prefer, in the early stage of the disease, small enemata of gruel containing laudanum, or opium or morphia suppositories. The patient's diet should consist of milk, gruel, broths, eggs, and such-like articles, together with such a proportion of alcoholic stimulants as the case may seem to need. If sickness be present, it must be treated with ice and such remedies as are generally useful in relieving sickness. When the dysentery passes into the chronic state, the use of astringent medicines and of vegetable tonics is indicated. The former may comprise copper, lead, iron, and tannin, together with other vegetable astringents, the latter a wide range of vegetable infusions. At this period also enemata are likely to be particularly serviceable; of which those containing copper, lead, tannin, sulphate of zinc, or nitrate of silver have been strongly recommended in the belief that they have a direct beneficial action on the diseased mucous membrane. We believe it to be a good plan to wash out the bowel night and morning with as large an injection of warm water or gruel as can be introduced without pain, and then to insert a morphia suppository. In treating dysentery it must not be forgotten that in both acute and chronic cases fæcal matter tends to accumulate above the diseased portion of bowel, and that this needs from time to time to be removed. For this purpose it may be necessary to administer an occasional purgative. In mild cases of the disease it is often well to commence the

treatment with a dose of castor oil, and to continue it with mild astringents, such as compound kino powder, Dover's powder, or aromatic chalk and opium.

VIII. PERITONITIS.

Causation.—Peritoneal inflammation is of common occurrence in both sexes and at all periods of life. It is due to various causes. In some cases it is idiopathic, or the result of exposure to cold and wet, or generally to those exterior conditions to which inflammations of other organs are so commonly traceable. Idiopathic peritonitis may attack the robust and healthy; it is more common, however, in those who are anæmic, debilitated, or broken down in constitution, and in those who suffer from obstructive diseases of the heart, lungs, or liver, and especially in such as are labouring under chronic Bright's disease. In many cases peritonitis is due to the simple extension of inflammation from neighbouring parts. It is thus developed, in the course of enteritis or gastritis, in connection with inflammatory affections of the liver, spleen, kidneys, or bladder, and in dependence on pleuritis, pericarditis, or inflammation of the abdominal parietes. The most fruitful causes, however, of grave peritoneal inflammation by simple extension are inflammation of the ovaries, uterus, and other pelvic organs in females, and especially that form of uterine inflammation which follows upon parturition. In many cases, again, peritonitis is caused by mechanical injury—sometimes by external wounds; more frequently by the perforation or rupture of some viscus and the extravasation of its contents or of foreign matters into the peritoneal cavity. Among such cases must be enumerated: ulcerative perforation of the stomach and duodenum; perforation of the small intestine (usually the ileum) by tubercular, typhoid, or other ulcers; perforation of the cæcum, vermiform appendix, colon, or rectum, consecutive to tubercular or typhoid ulcers, dysentery, or mere over-distension; rupture of an hepatic abscess, of the gall-bladder or common bile-duct, of an hydatid cyst, or of a psoas, renal, or other abscess; rupture of the uterus, or ovarian cysts; and, besides these, the laceration, from external violence, of the liver, spleen, kidneys, intestine, or bladder. Further, peritonitis is a frequent concomitant of abdominal tubercle or carcinoma, and a not uncommon result of pyæmic or metastasic processes.

Morbid anatomy.—The morbid changes which take place in the inflamed peritoneum are precisely similar to those attending inflammation of other serous membranes. They consist in dilatation of the minute vessels, with accumulation of blood within them, and infiltration

and thickening of the subserous tissue; and in inflammatory hyperplasia of the epithelial investment, with effusion from the subjacent vessels of modified plasma of the blood, of which part coagulates on the surface, forming, with entangled corpuscles, a false membrane, and part (mainly fluid) accumulates in the cavity. The first visible indications of peritoneal inflammation consist in most cases: in more or less intense capillary congestion, which is usually observed to extend in bands (determined by the pressure of the organs against one another) along the intestines; and in more or less loss of polish, due to the commencement of inflammatory exudation. With the advance of the disease, the congestion becomes more intense, and patchy, and sometimes complicated with subserous extravasations; and the solid inflammatory exudation increases in quantity. This forms in the first instance a thin, greyish, granular lamina; but as it increases in thickness acquires a more distinctly yellow tinge, and becomes, according to its quantity and position, ribbed, villous, papular, or honeycombed. The false membrane varies in thickness from a delicate film to a quarter or half an inch, or more; and in quality from a more pulp to a coherent elastic lamina. It usually acquires toughness with age; and the deeper-seated portions are always tougher than the more superficial. It tends to accumulate in the dependent parts of the peritoneal cavity, and to cause more or less intimate adhesion between neighbouring organs. The fluid effused in the course of peritonitis is often small in quantity, and, subsiding into the pelvis and lumbar regions, apt to escape detection; on the other hand, it is sometimes very copious, and causes much abdominal distension. It is chiefly abundant in chronic cases. It is usually opalescent, containing exudation corpuscles, and fibrinogen, which readily coagulates. The spaces occupied by the fluid are commonly traversed by filaments, bands, or bridles, of coagulated lymph.

Peritonitis, even when of local origin, generally soon involves the whole of the peritoneal surface. In some cases, however, it remains localised. Thus it may be confined to the neighbourhood of the liver, spleen, cæcum, or pelvic organs. The great omentum not unfrequently effectually limits its spread. Convalescence from simple peritonitis is attended with absorption of the dropsical effusion, subsidence of the inflammatory congestion, and organisation of the false membrane, with its gradual conversion into connective tissue. The usual consequences are that the peritoneal surface gets thick and opaque, and the viscera united to neighbouring parts and compressed by the contracting adventitious membrane. Thus the liver and spleen become adherent to the diaphragm; and the small intestines grow together, and are not unfrequently welded into an apparently homogeneous lump. Further, the liver and spleen, and other organs in a less degree, are apt to get more or less closely studded with opaque fibroid patches which may attain a thickness of ¼ inch or more, and present an almost cartilaginous consistence and aspect.

In many cases peritonitis becomes suppurative. Sometimes, as in the puerperal variety, the inflammation presents this character universally and from the beginning: the effused lymph is more abundant, opaque, yellow, and pulpy than in non-suppurative cases, and obvious pus is poured out into the peritoneal cavity. Where inflammation results from the perforation of some viscus or sac and the escape of irritating matters, general peritonitis of the ordinary adhesive character is often at once excited, and thus the effused matters become confined to some limited district. In such cases a circumscribed abscess frequently follows which may possibly undergo cure by the discharge of its contents either externally or into the bowel. But in some cases groups of such abscesses form; and sinuses extend in various directions—either among the peritoneal adhesions, or in the substance of the mesentery, meso-colon, great omentum, and other such parts; and fistulous openings may be established in various situations. General suppurative peritonitis may of course result from the escape of fæcal or other irritant matters into the peritoneum, especially if the escape be sudden and profuse; in which case if the accident be not immediately fatal the false membrane becomes exceedingly thick and tough, and the general surface acquires the usual characters of that of a chronic abscess.

Occasionally in peritonitis, as in other serous inflammations, copious hemorrhage from the newly-formed vessels of the adhesions takes place into the serous cavity.

Symptoms and progress.—The symptoms of peritonitis are mainly those of fever in combination with acute abdominal pain, increased by pressure. They are liable, however, to considerable variety; and many others of more or less importance are usually superadded. The phenomena of peritonitis differ greatly, indeed, in relation to the extent and intensity of the inflammation and to the circumstances under which it arises.

Acute idiopathic peritonitis, although by far the least frequent variety, yet displays the symptoms and course of the disease in their simplest and most typical form. Its mode of onset varies. Sometimes the outbreak of the local affection is preceded by a few days of vague sense of illness; sometimes it is marked by the occurrence of febrile symptoms, and even of rigors; sometimes the first indications of disease are sudden vomiting or purging, or both, or gradually increasing dysuria, or in females the occurrence of menorrhagia. But, whatever the initiatory symptoms, the patient before long complains of more or less marked febrile disturbance, and of burning, aching, pinching, or cutting pain, probably limited to some region of the abdomen, and increased by pressure or movement. The pain is usually, in the first instance across the lower part of the abdomen; and if the patient have not yet taken to his bed, he sits, moves and walks with his body bent into a stooping posture. Soon, however, the signs of

peritoneal inflammation extend and increase in severity; and at the same time the patient's general symptoms assume a more serious aspect. The abdominal pain becomes exceedingly severe, and is aggravated beyond endurance by the slightest movement. He takes to his bed, where he lies motionless on his back; with his head and shoulders elevated, and his thighs and legs flexed so as to diminish as far as possible the pressure of the abdominal walls on the internal organs; and breathing by means of the intercostal muscles only, and shallowly, with the same object. He not only shrinks from the pressure of the hand, but generally cannot endure even the weight of the bed-clothes, or of the poultices or fomentations which may have been ordered for his relief. The pain often is comparatively trivial so long as perfect rest is maintained; but it breaks out afresh whenever a cough, sneeze, hiccough, or deep inspiration takes place, and is liable to periodical and in many cases frequent aggravations, due to the peristaltic movements of the bowels. In association with these phenomena there is generally distinct fever. The temperature may reach 104° or 105°, but is very often not above 100° or 101°. The skin is hot and dry; the face flushed; the pulse increased in frequency and sharpness; the respirations augmented to 30 or 40 in the minute; and the tongue more or less coated and clammy, if not dry. Vomiting is often present, but is no necessary feature of the disease; and thirst is usually complained of. The bowels are generally constipated, but are not unfrequently loose. The urine is scanty, high-coloured, and sometimes retained; or there may be irritability of the bladder with painful micturition. It may be observed: that the presence of marked intercostal respiration indicates involvement of the upper part of the abdominal cavity; and that interference with micturition points to implication of the pelvic peritoneum. Further, there is reason to believe that the occurrence of vomiting or diarrhœa is referrible in some cases to inflammation of the serous surface of the stomach or bowels. If the disease take a favourable turn, which indeed at the end of a few days it usually does, the severer symptoms gradually remit: abdominal pain and tenderness subside, vomiting ceases, the respirations become natural, and the temperature and pulse return to their normal condition. If, on the other hand, the case be about to prove fatal, important changes in the symptoms more or less quickly supervene; the abdomen becomes distended, partly from effusion of fluid, mainly, however, from accumulation of gas in the intestines; pain and tenderness, though sometimes continuing and even becoming aggravated, very frequently undergo great diminution and sometimes cease entirely; sickness probably increases, and hiccough supervenes; the temperature falls, the extremities get cool or cold, the face pale or livid, and pinched and anxious in expression, and the skin suffused with cold sweats; the pulse increases in frequency, rising it may be to 130, 140, or 160 in the minute, and gets small, thready, and weak; the respirations quicken,

reaching, perhaps, 40 or even 60 in the minute; and the tongue becomes more thickly coated, and this and the lips dry. The patient, in fact, for the most part retaining his consciousness, falls rapidly into a state of profound collapse, in which he presently dies. Sometimes more or less delirium comes on before the fatal event, and death may then be preceded by coma.

The tendency to failure of circulation and to collapse is one of the most remarkable characteristics of peritonitis, as it is of enteritis. And it is important to bear this fact in mind; for even in the early stage of the disease, when the pulse is little accelerated, and sharp, and perhaps strong, and the patient appears to be suffering from what is termed 'the sthenic form' of peritonitis, a little over-exertion, some unwonted effort, may readily induce dangerous collapse.

It may be added that, while the presence of dropsical effusion adds to the distension of the abdomen, it does not, as a rule, materially aggravate the danger of the case; and that, if sufficiently abundant, it may be detected either by its causing dulness and bulging in the flanks, or by the presence of fluctuation; and further, that peritoneal inflammation constantly causes basic pleuritis, which may possibly be recognised during life; and that peritoneal friction may also occasionally be detected either by the fremitus it occasions or by auscultation. Death may occur as early as the second or third day of the attack, or may be delayed to the end of a week or ten days. When, however, the disease is prolonged beyond this date, it usually lapses into the chronic condition; in which, either inflammation of little intensity is kept up by the development of tubercles or some other cause, or the chronic symptoms are due to the formation of a circumscribed abscess.

Puerperal peritonitis differs from the idiopathic affection chiefly in the circumstances under which it arises, in its usually rapidly fatal course, and in the fact that it is often associated with, if not dependent upon, pyæmia taking its origin in inflammation of the uterine mucous membrane. It generally begins within a few hours or a few days after parturition, with severe rigors, attended with high elevation of temperature, and soon followed by intense pain across the lower part of the belly, and suppression or modification of the lochial discharge. The main points in its symptomatology, by which it differs from the simple form of the disease, are the speedy supervention of collapse, and the more general and early implication of the sensorial functions. Further, the symptoms are, in many cases, compounded of those of the local affection and those of pyæmia.

Peritonitis from perforation is one of the most common and interesting forms of the disease, and by far the most fatal of them. When perforation takes place in a person who appears to have been, up to the very moment of the accident, in the enjoyment of good health—as sometimes happens in cases of penetrating ulcer of the stomach, rupture of the urinary bladder, or perforation of the ileum

in mild enteric fever, the symptoms usually are: sudden and intense pain in the region of the lacerated organ, speedily followed by all the local indications of violent peritoneal inflammation; and extreme and immediate collapse, shown by pallor and coldness of surface, cold sweats, scarcely perceptible pulse, fainting, and vomiting. In some cases the patient dies of this primary collapse in the course of a few hours; and there may be little in the history or symptoms of such a case to distinguish it from one of Asiatic cholera, fatal before the supervention of diarrhœa; or from one of sudden effusion of blood into the stomach and bowels, fatal without hæmatemesis or melæna; or from one of ruptured heart or internal aneurism. But more frequently the patient rallies somewhat, and the collective symptoms of inflammation, fever, and peritoneal mischief become more clearly developed. Collapse, however, generally soon reappears, and the patient usually sinks after a period varying between twelve hours and two or three days. But the symptoms of perforative peritonitis are not always so intense and striking. Indeed, they are very often exceedingly difficult of recognition, and vague, when they occur in the course of abdominal diseases, whose proper symptoms tend to mask them: for example, dysentery, enteritis, and those rare cases in which peritoneal suppuration causes perforation of the bowel from its serous aspect. By far the most common cases of masked perforative peritonitis are those which occur in the second or third week of severe enteric fever, when the patient is prostrate with diarrhœa, and is dull, confused, and delirious, and to a large extent insensible to painful and other impressions. The evidences of perforation are then to be sought, not so much in obvious sudden collapse or intensity of abdominal pain, as in the general indications of failing strength—namely, increased weakness and rapidity of pulse, coldness of extremities, lividity of face, and diminution of intelligence, and of power over the limbs and sphincters; and in the supervention or increase of tympanites, with general abdominal tenderness, as shown by the expression and actions of the patient when pressure is made upon the surface of the abdomen. But although peritonitis from perforation is a well-nigh hopeless affection, there is reason to believe that it is not entirely hopeless. We have known of a case in which the patient certainly survived the accident for a fortnight; and several cases have been put on record in which there are good grounds for believing that a cure was effected after the formation of an abscess and its discharge by the bowel or some other route.

Peritonitis is not always the serious disease which has been above described. In a large number of cases it is, even if general, slight; and in a large number of cases, also, it is of local origin, and continues localised. The symptoms of partial peritonitis are the same in kind as those of the more general and more severe affection; but the local indications are limited to some comparatively small area, and the

general symptoms, if there be no serious complications, are comparatively slight.

It must not be forgotten that the adhesions which peritonitis leaves behind are not unfrequently a source of discomfort or danger. In some cases the compression of the bowels which they induce keeps up a tendency to colicky pains and intestinal disturbance; in some cases slowly contracting adhesions gradually compress a length of bowel and render it practically impervious; while in other cases, again, bridles or bands are formed, behind which coils of bowel are apt to slip and get incarcerated or strangulated.

Peritonitis is liable to be confounded both with enteritis and with colic; but is generally distinguishable from enteritis by the absence of intestinal obstruction, and from simple colic by the fact that the latter is unattended with fever, and that its pain is usually relieved in some degree by pressure.

Treatment.—The principles of treatment in peritonitis are sufficiently simple: they are, the maintenance of perfect rest, the administration of opium, and the application of leeches and other remedial agents to the surface of the abdomen. The patient should be placed and propped up in that position which he finds easiest, usually upon his back, with his knees and shoulders elevated. His abdomen should be defended from the weight of bed-clothes by means of a suitable cradle. Opium or morphia should be given sufficiently frequently, and in sufficiently large doses, to assuage the patient's pain and keep it in abeyance, to quiet the action of the bowels, and to promote comfort and sleep; it may be given by the mouth or by subcutaneous injection. If the case be severe and in an early stage, from ten to thirty or forty leeches should be applied to the surface of the abdomen; and bleeding should be promoted by fomentations or light poultices. Subsequently hot fomentations, turpentine epithems, mustard plaisters, or blisters may prove serviceable. On the other hand, cold applications—evaporating lotions, cold compresses, and ice-bags—have been largely advocated, and in many cases have proved of great advantage. It is important, moreover, in many cases, to relieve accidental complications, such as nausea and vomiting, dysuria, and the like. To meet the former indications, recourse must be had to ordinary antiemetic measures; to meet the second, the catheter may need to be employed. It is of course essential to maintain, as far as we possibly can, the patient's bodily strength; for which purpose nourishing diet, mainly in the fluid form, must be frequently administered in small quantities, and alcoholic stimulants, in amounts depending on the condition of the patient, combined therewith. If he cannot retain food on the stomach, it must be given by the rectum. It need scarcely be said that cases of peritonitis passing rapidly into collapse, and especially therefore cases of puerperal peritonitis, bear depletory measures less well than others; and that hence such treatment is admissible only in

quite their early stage. These cases, moreover, demand, more than others, early and considerable stimulation; and ammonia and ether, or similar agents, may be employed in addition to alcohol. When peritonitis is caused by perforation, our main reliance must be placed upon opium; and here especially it is of vast importance that the movements of the bowels be restrained, that purgatives be religiously avoided, and that the stomach be not overloaded with nutriment. If the patient survive for two or three days, some hope (remote, no doubt) may be entertained of his final recovery. But in order to promote this consummation, it is always desirable to investigate carefully from day to day the condition of the abdomen in order to detect the presence of any circumscribed abscess there, and as soon as may be to evacuate its contents.

IX. CIRRHOSIS OF THE STOMACH AND BOWELS.

Morbid anatomy.—Fibroid infiltration or thickening—a condition also termed 'cirrhosis,' and having a close anatomical relation with cirrhosis of the liver—occasionally takes place in the walls of the stomach and intestines. Thickening, which differs little, if at all, from this, is usually present in the neighbourhood of chronic ulcers of the stomach. When occurring independently, all the coats of the stomach as a rule are implicated, but more especially the muscular coat and the submucous tissue—the mucous surface being thrown into prominent folds over the affected area. The whole stomach is sometimes thus diseased, and is then usually diminished in size, tough, and retaining its form like an india-rubber bottle. But commonly the affection is limited to the neighbourhood of the pylorus, which then becomes constricted, and leads to general dilatation of the organ. The gastric walls, especially at the pyloric end, sometimes attain a thickness of half an inch or an inch, and present to the naked eye most of the usual characters of scirrhus. The morbid growth, however, differs from scirrhus in consisting wholly of fibroid tissue, and not possessing malignant properties. The intestines are much less frequently affected than the stomach.

The *symptoms* referrible to cirrhosis are exceedingly vague. They resemble indeed, for the most part, those of the early stages of carcinoma. When the pylorus is obstructed, the symptoms of that condition necessarily manifest themselves; when the large intestine is involved, the phenomena of stricture presently supervene.

X. TUBERCLE. (*Abdominal Phthisis.*)

Morbid anatomy.—Tubercular disease of the mucous membrane of the stomach is so rare, and so little is known about it clinically or otherwise, that it is needless to do more than record the fact of its occasional occurrence.

1. *Bowels.*—The mucous membrane of the bowels, on the other hand, is one of its most frequent seats; and, indeed, intestinal ulceration is, in a very large proportion of cases, of tubercular origin. Tubercle of the bowels occurs in rather more than one-half of the total number of cases of pulmonary phthisis, and rarely, if ever, independently of it; it is frequently associated, also, with tuberculosis of the peritoneum and other abdominal organs. It affects primarily Peyer's patches and the solitary glands; and in the small intestine, therefore, is always most abundant and most advanced immediately above the ileo-cæcal valve, from whence upwards, even though it extend throughout the whole ileum and jejunum, it gradually diminishes. It attacks the cæcum more frequently than any other part of the large intestine, involving also the ileo-cæcal valve and vermiform appendage; but it may form patches throughout the whole length of the colon. The large and small intestines are affected with equal frequency, and are affected conjointly about twice as often as each is affected separately. The tubercles appear as grey granules, or yellow cheesy masses, in the substance of the glands, and generally soon undergo softening, producing small, deepish ulcers, with thickened, overhanging edges. When several tubercles have softened side by side, as occurs in Peyer's patches, the ulcerated area presents in the first instance a kind of honeycombed appearance—the small ulcers being separated from one another by bridles of thickened mucous membrane; and the general margin, which is also thickened, presents a sinuous or scalloped outline. Tubercular ulcers generally tend to spread by the successive formation and softening of tubercles at their edges; and thus often creep over a considerable area. The whole mucous lining of the cæcum is sometimes destroyed in this manner; and extensive tracts of ulceration often stud the surface of the colon, at more or less distant intervals. In the small intestine tubercular ulcers have a remarkable tendency to spread transversely, and frequently form bands, from half an inch to an inch or more wide, occupying its whole circumference. In most cases the ulcerative process progresses up to the patient's death, and occasionally leads to serious hemorrhage or to perforation. Sometimes the ulcers cicatrise more or less perfectly—some, indeed, cicatrising while others are spreading or new ones forming. Tubercular cicatrisation is very apt to lead to considerable contraction of the bowel and even to the production of stricture. Sometimes, again,

tubercles dry up or get absorbed without undergoing ulceration, leaving behind them pigmented cicatrix-like patches which have some resemblance to the scars left in the skin by superficial lupus. It may be added that extensive ulceration of the large intestine, presenting all the characters of chronic dysenteric ulceration, is often met with in cases of chronic phthisis, where there is no discoverable tubercle in any part of the bowels except the ileum, and where, therefore, it may be a question as to whether the ulceration is of tubercular origin, or has arisen in mere non-specific excoriation such as might be caused by the constant passage of irritating secretions from the tubercular bowel above.

2. *The peritoneum and abdominal lymphatic glands* are often affected. Generally in cases of tubercular ulceration of the bowel, and certainly in all cases of extensive ulceration, grey granulations, in greater or less abundance, stud the serous surfaces corresponding to the diseased areæ. But such formations are for the most part purely local, and of little importance. There are other cases, however, far less common, yet not unfrequent, in which the tendency to the growth of tubercles is general throughout the serous membrane, and in which ulceration of the bowel is not only not their starting-point, but often altogether absent. Peritoneal tuberculosis is almost always associated with similar disease of other parts; most commonly with pulmonary phthisis, but not unfrequently with tubercular affection of the bowels and other abdominal organs. It complicates a very large proportion of those cases in which the pleuræ, spleen, liver, kidneys, uterus and Fallopian tubes, or brain is involved. Peritoneal tubercles are sometimes miliary and grey, and from the size of a poppy-seed downwards. Sometimes they form lobulated masses from the bulk of a tare up to that of a hazel nut—presenting for the most part an opaque buff-colour, often mottled with black points or patches; and exhibiting a cheesy aspect and consistence, which are modified by the greater or less abundance of fibroid material which invests and permeates them. Sometimes, again, but much more rarely, there are found, lying between organs which are adherent, tubercular laminæ of considerable thickness and extent. Peritoneal tubercles, indeed, rarely exist independently of the effusion of lymph and the presence of false membranes. The larger tubercular masses are usually comparatively few in number; the miliary tubercles, on the other hand, are, as a rule, thickly set and innumerable. Further, in the latter case the peritoneal surface is often found covered with a layer of greyish, transparent, adherent, and toughish lymph, which not only invests the abdominal organs, but unites them more or less with one another, and in the substance of which tubercles are disseminated as opaque grains.

In association with the presence of tubercles all the usual phenomena and sequelæ of simple inflammation, such as streaky redness, fibrinous effusion, and dropsical accumulation, are apt to manifest them-

selves; sometimes, also, suppuration, sometimes profuse hemorrhage. Further, it occasionally happens that, during the progress of peritoneal tuberculosis involving the intestinal walls, perforation of the latter takes place. The most important of these phenomena from its frequency is undoubtedly ascitic effusion.

The abdominal lymphatic glands are a frequent seat of tubercle; mainly, however, the glands of the mesentery, and more especially those of them which are in relation with tuberculous intestine. Tubercle of these organs is mostly secondary to tubercle either of the intestines or of the peritoneum. It appears in them, and for the most part in their peripheral portions, in the form of minute, hard, grey points, which occur in groups and tend gradually to run together, and to form imbedded masses which soon undergo caseous change. Glands thus affected may suppurate and even rupture into the peritoneal cavity; or they may get slowly converted into mortary or calcareous lumps. Tubercular glands are usually more or less enlarged, sometimes, indeed, attain the size of a pigeon's egg. When, however, they undergo calcareous change they contract and become invested with an indurated capsule. Tubercular mesenteric glands sometimes, especially in children, collectively form masses easily detectable through the abdominal walls; but there is little doubt that most of those cases of extreme enlargement of these glands which were formerly regarded as tubercular were really cases of lymphadenoma or some other form of malignant disease.

Symptoms and progress.—1. *Bowels.*—The symptoms of tubercular ulceration of the mucous membrane are in no degree specific; but they vary according to the part of the bowel which happens to be affected. When the disease is limited to the ileum there is probably more or less pain and tenderness in the region of the cæcum, with frequent griping. The bowels may be confined or loose, but are more often, perhaps, irregular. When the large intestine is involved, the symptoms closely resemble those of chronic dysentery, and, indeed, are by no means necessarily distinguishable from them. The points of chief clinical importance in reference to intestinal tuberculosis are: first, that the disease is for the most part a progressive one, and that hence diarrhœa having once declared itself tends to become progressively more and more severe and intractable; second, that during its progress the patient rapidly undergoes extreme emaciation, becomes excessively feeble, and suffers in an aggravated form from night sweats, imperfect circulation (indicated by blueness of nose and coldness of extremities), and the other phenomena which attend rapid impairment of nutrition; and, third, that it is usually associated with well-marked indications of tubercular disease in other organs. Hemorrhage, perforation, and stricture are not special to tubercular ulceration, and their symptoms need not now be discussed.

2. *Peritoneum.*—The symptoms which attend the progress of peri-

toneal tuberculosis present much variety and are often vague and misleading. Often, indeed, and not only in those cases in which the peritoneal affection is slight, or in those in which it is as it were overshadowed by the preponderance of disease in other parts, but in those cases in which it is the predominant or sole affection, they fail to indicate clearly the peritoneum as the seat of disease. Further, they are so generally complicated with symptoms due to coexisting tubercular disease in other organs, especially the lungs, pleuræ, and intestines, that it is impossible altogether to dissociate them from the latter. Most cases of tubercular peritonitis, attended with obvious symptoms, may perhaps be somewhat roughly arranged in two classes: the first (the acute class), in which the symptoms have a close resemblance to those of enteric fever; the second (the chronic class), in which the symptoms correspond for the most part with those of chronic peritonitis.

In the acute form the patient, sometimes in the midst of perfect health, more often after an indefinite period of languor and loss of flesh and strength, begins to manifest febrile symptoms attended with remissions, and indicated by heat and dryness of surface, quickened pulse, pains in the limbs, loins, and head, diminution of the secretions, and perhaps drowsiness. At the same time probably the abdomen becomes hard, tumid, and tender, and more or less uneasy or painful. Generally, also, there is some disturbance of the digestive functions—dryness or furring of the tongue, thirst, loss of appetite, nausea or sickness, and constipation, diarrhœa, or irregularity of bowels. And possibly, with no material change in his symptoms beyond what may be due to increasing debility and emaciation, and the gradual supervention of 'typhoid symptoms,' the patient gradually sinks, and at the end of a few weeks dies. Among the chief points by which this affection may be distinguished from enteric fever are: the absence of rash, and of pain specially limited to the cæcal region; the probable presence of tubercular disease in other organs; and the fact that the temperature, although it may be considerably elevated, does not present that regularity of morning remissions and evening exacerbations which are so characteristic of enteric fever.

In the chronic variety of peritoneal tuberculosis, the disease sometimes commences with more or less typical symptoms of acute peritonitis; sometimes it creeps on with the utmost insidiousness; but in either case the symptoms gradually merge into those of chronic peritonitis, with which (unless our diagnosis be aided by the discovery of tubercular disease elsewhere) we cannot well avoid confounding them. During the progress of the disease a more or less diffused tumour is apt to be developed—due for the most part to thickening of the great omentum—which may suggest the formation of a circumscribed abscess or a malignant growth. This sometimes forms a transverse bar, in a line with or above the umbilicus. Ascites is very apt to

ensue. The duration of chronic tubercular peritonitis may vary from a month or six weeks to a year or two.

Tubercular peritonitis tends, as a rule, to a fatal result; at the same time, there are good grounds for the belief that recovery occasionally ensues.

Treatment.—The general treatment of abdominal tuberculosis is identical with that of pulmonary phthisis and generally of scrofulous disease. It comprises careful attention to hygiene, removal if need be to a more suitable climate, a good wholesome and nutritious dietary, and the use of cod-liver oil, iron, and vegetable tonics. If the mucous membrane of the bowels be specially affected, and the patient be suffering from exhaustive diarrhœa, treatment must of course be specially directed to relieve this condition. For the details of treatment in this case we must refer the reader to the articles on intestinal ulceration and dysentery. When the peritoneum is the part principally involved, abdominal pain may need to be relieved by the application of counter-irritants, fomentations, or even leeches; sleeplessness, weariness, and pain may require to be overcome by the use of opiates or other sedative or narcotic medicines; and further, nausea, sickness, diarrhœa, and intestinal obstruction may all in turn call for relief by the various measures on which in such conditions reliance is usually placed.

XI. TUMOURS.

A. *Non-Malignant Tumours.*

These are not uncommon, but on the whole are of little medical importance.

Pedunculated fibrous tumours or *polypi* are sometimes very small, very numerous, and of wide distribution throughout both the small and the large intestines. Sometimes, on the other hand, they are few in number or solitary, and then often attain large dimensions. The latter are occasionally observed in the ileum, but chiefly affect the lower part of the rectum. In the former situation they are believed to be in some cases the determining cause of intussusception; in the latter they often produce irritation, bleeding, tenesmus, and other discomforts. Those only can be diagnosed and treated which are within reach, and for them removal is the only effectual remedy.

Villous growths are in many cases malignant. Some, however, and especially such as are met with in the large intestine, appear to be non-malignant. These usually occupy a limited and well-defined area, and sometimes encircle the bowel. The intestinal walls in the situation of the growth, and especially the mucous and submucous

coats are generally much thickened; and from this thickened area as a base, close-set, elongated, complex villi take their origin. These growths frequently cause hemorrhage, which is occasionally serious; and diarrhœa, which is sometimes of a dysenteric character. When situated near the anus they may be removed by operation.

B. *Malignant Tumours.*

Morbid anatomy.—Malignant growths commence, sometimes in the mucous membrane of the stomach or intestines, sometimes in the peritoneal tissue, sometimes in the mesenteric or retro-peritoneal glands. In the first case, the disease usually takes its origin at some particular spot; whence it spreads over a greater or less extent of the contiguous mucous membrane, then gradually involves the whole thickness of the parietes, and having reached the serous lamina, diffuses itself in a greater or less degree over it, and further implicates the mesenteric or other glands. In the second case, the growth tends rapidly to generalise itself over the surface of the serous membrane, and to infiltrate the subserous tissue; but it is often a long time before it penetrates the muscular wall of the stomach or bowels. Sooner or later, however, this is invaded at points, and then the mucous membrane becomes involved. The lymphatic glands necessarily also suffer. When the disease begins in the mesenteric or retro-peritoneal glands these gradually enlarge; and presently the morbid growth extends from them into the surrounding connective tissue, infiltrates it, and thence spreads to the serous membrane on the one hand, and to the intestinal walls on the other. It will thus be seen that, although the site in which malignant disease commences exerts a more or less important influence over its distribution and consequences, the ultimate tendency is in each case to its general diffusion.

1. *Scirrhous cancer*, originating in the walls of the stomach or bowels, causes thickening and induration of the parts which it affects. If it attack the submucous tissue, this becomes greatly hypertrophied; and presently the superjacent mucous membrane getting incorporated with it, its natural structure gradually disappears, and its free surface, at first perhaps thrown into rigid folds, grows irregular and nodulated. Whilst this process is going on, the muscular wall becomes invaded; the morbid growth extends along the intermuscular septa, converting them into irregular but thick vertical scirrhous bands, and the muscular tissue thus divided into strands at first hypertrophies, and subsequently undergoes fatty degeneration. At length the subserous and serous tissues get implicated; they, like the mucous tissue, become dense, hard, and thick, and small wheal-like excrescences or nodules spring up upon the free surface. Sooner or later in the progress of the case erosion and destruction of the affected mucous membrane takes place, and a smooth excavated ulcer

results; in some cases sloughs form, and the destruction is more rapid and irregular; and frequently carcinomatous nodules sprout up from the edges and floor of the ulcerated surface. Sooner or later also adhesions form between the affected viscus and neighbouring organs, and along them the morbid process may be propagated.

Peritoneal scirrhus always commences in the form of hard, lenticular, white spots, measuring a line or so in diameter, which, though projecting above the surface, tend specially to invade the subserous tissue. They are in the first instance scattered thinly or irregularly, but soon become aggregated in parts or generally, and then coalesce so as to form patches of various sizes. These may be uniformly smooth, or may still present traces in their surface or outline of their mode of development. They rarely, however, form outgrowths, and not very often invade subjacent organs; rarely, too, do they become more than a line or two thick, except where they involve folds or processes of peritoneum. The appendices epiploicæ become converted into small hard lumps, the mesenteric and other like duplicatures thickened and indurated, and the great omentum contracted into a thick band, stretching transversely across the abdomen in the course of the transverse colon. Scirrhous cancer, indeed, whether affecting the gastro-intestinal tube or the peritoneum, tends rather to cause contraction and thickening than outgrowth, and thus, as a rule, leads to constriction of the cavities or canals which it involves, and especially therefore to constriction of the cardiac or pyloric orifice or other parts of the alimentary tube.

2. *Colloid cancer* most commonly takes its origin in the serous lamina, whence it spreads to the mucous membrane. When appearing first in the latter tissue, it causes, as other forms of cancer do, more or less considerable thickening, and manifests itself at the surface in the form of scattered masses, which have a resemblance either to the wheals of urticaria, or to groups of herpetic or eczematous vesicles. Like scirrhus, it invades the muscular coat—running along the intermuscular septa, and causing the muscular tissue to become thickened and hypertrophied; then attacks the subserous tissue and the serous membrane itself, causing these also to become thickened; and finally produces at the free aspect groups of vesicles, varying individually, perhaps, from the size of a mustard-seed to a scarcely visible point. At the mucous surface the affected patches become eroded and excavated, but remain pretty smooth, and discharge in abundance the transparent glairy fluid with which the interstices of the cancerous matrix are filled. Colloid cancer of the peritoneum, in its early stage, appears in the form of groups of vesicles, which are more or less elevated above the general surface, and spread sometimes in tortuous and anastomosing lines as though taking the course of the lymphatic vessels, sometimes by forming scattered, isolated, more or less pedunculated growths. The morbid process tends to spread both in surface and in depth. It

always involves the sub-peritoneal tissue, which may attain very considerable thickness; and it extends thence most frequently to the muscular and mucous coats of the stomach and intestines, less frequently to the substance of the mesenteric glands, pancreas, liver, spleen, and other viscera. In extreme cases nearly the whole of the peritoneum is affected; it is then irregularly thickened—the various duplicatures being especially hypertrophied, and the great omentum either converted into a large lobulated mass, or contracted, as it is in scirrhus, into a thick, irregular, transverse band. In the progress of the disease erosion of the surface is apt to take place, and the glairy fluid which it yields is discharged in some abundance into the abdominal cavity.

3. *Encephaloid cancer*, when affecting the abdominal organs, is characterised, as it is elsewhere, by its softness, milkiness, and rapidity of growth. If it commence in or beneath the mucous membrane of the stomach or bowel, on the one hand it soon invades the mucous and submucous tissues, and on the other spreads to the muscular coat, and through this to the tissues on the outer aspect of the viscus. The extension of the growth both in thickness and in surface is usually very rapid, and before long results in the formation of a more or less lobulated tumour, which often attains a very considerable bulk. The encephaloid mass is of course liable to undergo all those interstitial changes to which encephaloid cancer is usually liable; but especially it tends to ulcerate. Ulceration begins, as a rule, early, and is almost invariably attended with more or less abundant sloughing of the cancerous mass, which becomes consequently deeply and irregularly excavated. But while this is going on, the edges of the ulcerated chasm still furnish lobulated outgrowths, and moreover such outgrowths not unfrequently take place from the ulcerated surface itself. In some cases encephaloid tumours give rise from their mucous aspect to a pile of highly vascular villous outgrowths, constituting the so-called 'villous cancer.' Encephaloid cancer of the peritoneum appears in the form of discrete nodular outgrowths, which are small and rounded, and differ from those of scirrhus not only in their greater softness but in their greater prominence. They are hemispherical, spherical, or pyriform, and often distinctly pedunculated. In its further progress encephaloid cancer presents great varieties. In some cases it seems, like scirrhus, to invade more particularly the substance of the peritoneal folds and to involve subjacent organs; and under such circumstances we sometimes find the mesentery converted into a thick, plicated, cancerous mass, with the cancerous growth extending from the mesenteric attachment over the surface of the intestines, or the greater or lesser omentum or the subperitoneal tissue of other regions affected in like manner and forming more or less distinct tumours. In other instances it tends rather to form outgrowths which are sometimes

small and clustered, sometimes more or less distinct from one another, rounded and massive. In the former case the whole peritoneal surface may be found beset with small lobulated or bunch-of-currant-like excrescences, and the great omentum converted into a large loose mass of such bodies. In the latter case the tumours, though more or less abundant, are isolated; and while many probably are small, others attain the size of an orange, or even of a child's head. So far as we know, the melanotic variety of encephaloid cancer always manifests itself in the latter form.

4. *Epithelioma* affects the rectum and anus exclusively. It is sometimes of primary origin, sometimes due to extension from the uterus or vagina.

5. *Adenoid cancer*, or cylindrical epithelioma, which has a close resemblance to encephaloid, is not uncommon in the intestine. It is probably more common than any as a primary disease of the mucous membrane, and is especially apt to cause stricture.

6. *Sarcomatous and lymphadenomatous* growths may be regarded clinically as mere varieties of encephaloid cancer. Still they present some peculiarities of habit. Sarcomatous growths are exceedingly uncommon, and arise mainly in the substance of the walls of the stomach, where they constitute tumours of considerable size, which tend more or less rapidly to ulcerate, and comport themselves generally as do encephaloid tumours. Lymphadenoma is especially a disease of the lymphatic glands and textures; and hence when the abdominal organs are its seat, the abdominal glands usually reach an enormous volume, and the spleen undergoes more or less considerable enlargement. In its further progress the morbid growth involves the connective tissue around the already diseased glands; and hence the substance of the mesentery and other similar folds of the peritoneum become thickened and infiltrated, and nodular outgrowths sometimes appear upon their surfaces. As the affection still progresses the morbid growth creeps from the mesenteric attachment on to and around the small intestine, confining itself almost, if not quite exclusively, to the peritoneal membrane and subperitoneal tissue; and thus the intestine, while remaining pervious and probably healthy as to its mucous membrane, becomes converted into a thick-walled rigid cylinder. The large intestine and even the stomach may be similarly affected.

Whenever carcinoma or any other form of malignant disease affects the peritoneum, stomach, or bowels, it may spread by continuity to almost any neighbouring organ; and hence the liver, pancreas, and spleen are liable to be invaded when the stomach or peritoneum in the vicinity is its seat, and the various pelvic organs when the rectum is diseased; and further, the mesenteric and retro-peritoneal lymphatic glands, or some of them, when secondarily affected, often develop into large tumours. Such tumours are produced most rapidly, and attain

their largest dimensions, when the disease to which they are secondary is some soft form of malignant disease.

There are certain parts of the gastro-intestinal tube which are more liable than others to be the primary seat of malignant disease. They are the stomach and certain tracts of the large intestine. Of these the stomach is much the most frequently affected; and, although no portion of its surface enjoys absolute immunity, there is no doubt that its pyloric extremity most frequently suffers. When the cardiac orifice is the seat of disease, the adjoining portion of the œsophagus is commonly affected. When the pylorus suffers, the morbid process usually encircles that portion of the stomach which adjoins it, but very rarely extends into the duodenum. The effect of malignant disease upon the stomach is in many cases to cause irregular contraction and deformity, and especially to cause stricture at the cardiac or pyloric orifice. If the pylorus be alone affected and resist the onward transmission of food, the stomach often becomes preternaturally dilated; if, on the other hand, there be impediment to the entrance of food from the œsophagus, the organ necessarily shrinks. Of the large intestine, the parts most liable to suffer primarily are, first, the rectum, and second, the sigmoid flexure; and here, as at the orifices of the stomach, the disease tends to circumscribe the tube and to cause stricture. It may be added that while all parts of the gastro-intestinal canal are apt to be implicated in the progress of malignant disease commencing in the peritoneum or lymphatic glands, the lower part of the rectum is especially liable to become involved in the extension of uterine, vaginal, or other pelvic growths. Further, it must not be forgotten: that malignant disease, whether of the stomach or bowels, may be attended with rupture into the peritoneal cavity or the establishment of communications with adjoining hollow organs; that hemorrhage (sometimes profuse), with foul or fetid discharges, is apt to take place from the congested or ulcerated mucous surface: and that (especially when the peritoneum is largely involved) peritoneal inflammation, ascites, or obstruction to the return of blood from the lower extremities, frequently supervenes.

Of the various forms of malignant disease affecting the organs under consideration, scirrhus is undoubtedly the most common; scarcely any of them, however, is absolutely rare. Taking all forms together, it may be said that they mostly occur after the age of forty—a rule, however, which is more absolute as regards primary gastric or intestinal disease than that of the peritoneum; indeed malignant disease of the peritoneum (especially in connection with similar affection of the ovaries) is not uncommon in young adult females. Sex, on the whole, exerts but little influence numerically. Carcinoma of the stomach is one of the most frequently fatal forms of malignant disease.

Symptoms and progress.—The symptoms referrible to malignant disease of the several organs now under review simulate those of the

inflammatory (mainly chronic) affections of the same organs. And the differential diagnosis between them often depends, less on the presence or absence of specific symptoms, than on a careful consideration of the history of the case, and a close observance of the phenomena which it presents, and their relation to one another. Thus malignant disease of the stomach has many features in common with chronic gastritis and gastric ulcer; malignant disease of the bowels many in common with chronic ulceration of the bowels and its various sequelæ; and malignant disease of the peritoneum many which it shares with chronic peritonitis, tubercular peritonitis, and, we may add, simple ascites. But malignant disease is always remarkably insidious in its progress; and vague symptoms of ill-health, with loss of flesh and strength, usually manifest themselves long before the patient quite recognises the fact that he is ill, or can quite define the character of his sufferings. The patient, therefore, is generally ill and often markedly cachectic before the specific signs of gastric, intestinal, or peritoneal mischief reveal themselves. Again, the course of a case of malignant disease is always progressively from bad to worse; and this progressively downward tendency is connected as a rule, not simply with the aggravation of the ordinary symptoms due to progressive impairment of function of the organ primarily affected, but to the supervention of complications connected with the special properties of malignant disease, such as the involvement of the liver and other abdominal organs in the morbid growth, and the development of disease in more remote organs. The appearance of a tumour and its manifest increase in bulk and change in form, in association with the various phenomena above enumerated, leave little room for doubt. Febrile symptoms, thirst, and dryness or foulness of tongue are no necessary accompaniments of the disease.

1. *Stomach.*—The special symptoms of malignant disease of the stomach are as various as those of ordinary dyspepsia. They comprise mainly loss or capriciousness of appetite, pain, and vomiting. Anorexia is a very constant and ordinarily a very early symptom; but it is very variable in its presence, and is sometimes absent from first to last. Occasionally the appetite is excessive. Uneasy feelings—weight, and fulness—in the region of the stomach, are frequently complained of, especially after taking food. In most cases also there is absolute pain of a more or less intense character, variously described as aching, burning, cutting, or stabbing, and referred either to the epigastrium or interscapular region or to other neighbouring situations. This comes on in paroxysms, which are probably at first 'few and far between,' but increase in frequency, duration, and severity with the progress of the disease. It is often brought on or increased by the ingestion of food, or by pressure applied to the epigastrium. Pain, however, like anorexia, is sometimes of little severity, and occasionally wholly wanting. Eructation is a common but unimportant symptom. Vomiting,

however, supervenes sooner or later in the great majority of cases. This is mostly caused by the taking of food, and comes on at different periods after it; if the cardiac orifice be contracted the food is usually returned at once (as in other forms of œsophageal obstruction) by regurgitation; if the pylorus be affected the vomiting is often delayed for an hour or two or more than that; when the stomach is irritable vomiting may (as in gastric inflammation) take place almost immediately after food has entered the stomach. The vomited matters in the earlier periods of the disease are chiefly altered ingesta combined with mucus and the acid secretions of the stomach. Later on (especially if ulceration have taken place) small quantities of blood escape from the diseased surface, and, mingling with the contents of the stomach, give to the vomited matters a sooty or coffee-ground appearance. The persistence of this kind of vomit is very characteristic of gastric carcinoma. Profuse discharge of blood, with hæmatemesis and melæna, occasionally takes place, but is not nearly so frequent relatively as it is in cases of simple ulcer. When sloughing occurs, the vomited matters are often extremely offensive. It is very common, especially when the pylorus is the seat of disease, for them to contain sarcinæ and the torula cerevisiæ. The detection of a tumour depends partly on its size and partly on its situation. A tumour at the cardiac orifice or cardiac extremity can rarely be felt, however large or extensive it may be; and one situated in the posterior wall or lesser curvature is less easy of recognition than one occupying the anterior surface or the larger curvature or the pylorus. The situation of perceptible tumours varies somewhat. They mostly occupy the epigastric or right hypochondriac region, but are sometimes found in the neighbourhood of the umbilicus. Unless they have become firmly adherent to the abdominal walls in front, or have blended with the pancreas or other enlarged glands behind, they are usually moveable to some extent under the abdominal walls, both during the act of forced inspiration and (if the patient is lying down) in rotation of the body from side to side. They are often irregular in shape, generally very hard, and not unfrequently lifted up with the aortic pulsations. They are usually also resonant on percussion. Constipation is almost invariably present. The special symptoms which ensue when rupture of the stomach into the peritoneal cavity takes place, or when a communication becomes established with the transverse colon, need not be detailed.

2. *Bowels.*—The symptoms referrible to malignant disease of the bowels are yet more vague in their indications than those which attend gastric carcinoma. There is generally more or less irregularity of action, sometimes looseness, sometimes constipation, and it may be the occasional discharge of mucus or modified blood. At the same time there is often pain: partly of a colicky character and connected with unwonted movements of certain portions of the bowels; partly burning, aching, or cutting, and referrible to some particular region. Malignant

disease of the large intestine, and more particularly that of the sigmoid flexure and rectum, produce as a rule more or less impediment to the action of the bowels, and finally stricture. It is in this particular case also that, associated with symptoms of obstruction—preceding them, accompanying them, or following them—mucous, sanguinolent, purulent, and fetid discharges, occasionally even profuse hæmorrhages, occur. Further, if the rectum be the seat of disease, the case is apt in its progress to be complicated by the formation of communications between the bowel on the one hand, and the vagina, bladder, or urethra on the other. In malignant disease of the bowels, equally as in malignant disease of the stomach, the presence of a distinct permanent tumour is a fact of capital importance. This may often fail of recognition; moreover phantom tumours, due to accumulation of flatus or fæces, are in such cases specially apt to arise and disappear from time to time and puzzle the physician. When the lower part of the rectum is affected, the presence of a tumour may generally be readily detected by digital examination.

3. *Peritoneum and glands.*—The symptoms of peritoneal and of glandular malignant disease are necessarily very various and easy to be misunderstood. These affections are in a very large proportion of cases associated with similar disease of the stomach, bowels, liver, uterus, or ovaries, and not unfrequently supervene upon them; and hence their special symptoms are liable to be confounded with and masked by those of the latter lesions. On the other hand, many of the symptoms commonly attributed to malignant disease of the stomach and other abdominal organs are strictly referrible to involvement of the peritoneum and lymphatic glands. Among the symptoms which attend the affections now under discussion must be enumerated nausea, vomiting, loss of appetite, and constipation, diarrhœa, or irregularity of the bowels, together with more or less abdominal uneasiness and pain. The most significant point, however, is the progressive enlargement of the belly with the presence of a growing tumour or tumours. These present all varieties of character; they may occur in any region; may be movable or fixed; may vary in size and shape; may be hard and resisting, or soft and almost yielding a sense of fluctuation; and, especially when they are developed in the neighbourhood of the cœliac axis and superior mesenteric artery or over the aorta, may pulsate as distinctly as many aneurysms do. And hence, notwithstanding the important evidence which their presence furnishes, they may be confounded, at some stage at least of their progress, with circumscribed abscesses, hydatid tumours, floating kidneys, or even aneurysms. In cases where (even if the malignant growth be very abundant) the individual tumours are small, the presence of peritoneal outgrowths may altogether escape detection. We may draw attention to the fact that often, when no other signs of tumour are distinguishable, the presence of the thickened and contracted great omentum, which has

been shown to occur so frequently in scirrhous and colloid cancer, may be recognised as a more or less irregular transverse bar extending horizontally from under the margins of the left ribs across the upper part of the umbilical region to the neighbourhood of the umbilicus. To the above statement it must be added: that the peritoneal affection often becomes complicated by ascites, by peritonitis of a subacute character, by involvement of the gastro-hepatic omentum with obstruction of the vena portæ or common bile-duct and consequent jaundice, or by anasarca of the lower extremities; and that occasionally also the kidneys get affected, the ureters obstructed, or the pelvic organs involved.

Treatment.—The treatment of the above affections can unfortunately only be palliative. When symptoms are chiefly referrible to the stomach they must be treated, and may for a time be benefited, by such measures as have been recommended for gastric ulcer. When the intestines mainly are involved, diarrhœa may need to be restrained by astringent medicines, constipation to be overcome by mild laxatives, such as castor oil and the like, or by enemata. And pain, whatever its seat or source, may often be relieved by counter-irritation, fomentations, or leeches. Opium in such cases is generally invaluable, and in most cases becomes at length indispensable—relieving discomfort and pain, soothing the mind, and giving sleep. The patient should of course be sustained by appropriate aliment in sufficient quantities and, if necessary, by stimulants. The quality of the food and the mode of its administration must be determined by the special requirements of the case; but generally it should be wholesome, easily digestible, and administered in small quantities and at frequent intervals. Milk, eggs, beef-tea, broths, fish, and the like are among the most suitable articles of diet.

XII. PARASITIC AFFECTIONS.

A. *Tape-Worms and Cyst-Worms.* (*Cestoda or Tæniada.*)

1. *General Account.*

The general term cestoda or tæniada includes tape-worms and cyst-worms. Of these, though many species are known to exist, four only are of interest and importance to the practical physician. They are the tænia solium, tænia mediocanellata, tænia echinococcus, and bothriocephalus latus, with their respective cystic representatives.

All the tæniada pass through two phases of existence. In the one the characteristic head or *scolex* of the animal, developed in connection with a cyst or bladder-like body, and devoid of sexual organs, lies im-

bedded in the solid tissues of the *host*, or creature that harbours it. In the other the animal, or rather colony of animals, in the form of a tape-worm or *strobilus*, occupies the alimentary canal. In this condition it still presents at its upper extremity a scolex or head by which it adheres to the mucous membrane, while its tape-like body is divided into a series of quadrilateral elements, or *proglottides*, each of which when mature contains male and female organs and must be regarded as a distinct animal. To trace the cycle of events in the life-history of the tæniada it will be convenient to commence with the ripe proglottides, within which are produced enormous numbers of fertile eggs, in the interior of each one of which a peculiar six-hooked embryo is developed. These proglottides usually become detached from the rest of the strobilus, escape from the anus of the host, and either then or previously discharge their ova, which become scattered broadcast. Of these fertile ova some find their way sooner or later into the alimentary canal of some appropriate animal. Then the six-hooked embryo bursts its shell, migrates through the intestinal parietes, and continues its wanderings until it reaches some spot suitable for its further development, where it gradually undergoes those changes which result in the formation of the perfect cystic scolex. The further fate of this scolex depends mainly on that of its host. It cannot migrate, but lies passive in the cavity which it forms for itself, and there at length perishes, unless before that occurrence its host become the prey of some other animal. In this event the scolex enters the alimentary canal, and under the new conditions which then surround it at once enters on a new career of life. It fixes itself to the mucous surface, it loses its vesicular expansion, and from its caudal extremity the strobilus or chain of sexually reproductive proglottides is gradually evolved. Thus two distinct hosts as a rule are needed for the completion of the cycle of existence of these creatures; the one (usually a vegetable-feeder) for the asexual period of its existence, the other (very commonly a carnivorous animal) for the period of its sexual activity. It follows from the above statements that the ova of the tape-worm, even if set free within the alimentary canal, probably never get hatched until after their escape from it. Further, it may be regarded as a general rule, that the same species o animal is not liable to suffer from both the cystic and the sexual forms of the same cestode. Man is in some degree an exception, for he is apt to harbour both the tænia solium and its vesicular representative—the cysticercus cellulosæ. Looking, however, to the facts that patients affected with this tape-worm are not usually also affected with the cysticercus, and conversely, and that man, moreover, is an omnivorous feeder, there is good reason to believe that the exception is apparent rather than real, and that he derives the two forms of the parasite in the orthodox way from independent sources.

2. Tænia Solium, Tænia Mediocanellata, and Bothriocephalus Latus.

a. Tænia solium and Cysticercus cellulosæ.—The tænia solium is one of the most common of human tape-worms. In its perfect condition it usually measures from seven to ten feet long, but often exceeds that length. Its head or scolex, which is about as large as a small pin's head, or, to be more exact, between $\frac{1}{45}$ and $\frac{1}{35}$ inch in diameter, is succeeded by a delicate thread-like neck, which, gradually becoming broader and flatter and wrinkled transversely, merges ere long in the distinctly-jointed body. The joints or proglottides are, in the first instance, much broader than they are long; but gradually with their increase in size this relation ceases; and although they still get broader, their length throughout the greater part of the strobilus exceeds their breadth. Towards the lower extremity, the quadrilateral joints measure on the average a quarter of an inch wide by half an inch long. The globose head presents four projecting suctorial discs placed at equal distances upon and a little above the equator; and springing from its pole a rounded elevation, or *rostellum*, the margin of which is furnished with a double circle of hooks. The apparently homogeneous neck may be seen under the microscope to be transversely wrinkled at a very short distance from the head. The sexual apparatus first becomes visible about a foot below. It comprises male and female organs opening by a common aperture in the lateral edge of each joint—the apertures of the successive proglottides alternately occupying opposite sides. At about two feet from the head the ova become impregnated, and shortly afterwards enter the uterus, which occupies a large portion of the body of the proglottis, forming a longitudinal central canal with several horizontal diverticula on either side. The egg is globular, about $\frac{1}{700}$ inch in diameter, presents a remarkably thick brownish shell, both concentrically and radially striated, and when ripe contains a six-hooked embryo.

The tænia solium is essentially an inhabitant of the small intestine, to the mucous surface of which it fixes itself by its hooklets and suckers. It is usually, as its name implies, solitary; but two, three, or more are not unfrequently associated, and occasionally much larger numbers. From the time of its entrance into the bowel until it reaches its full development a period of three or four months usually intervenes; and it may live in the bowel for many years, during which time it is constantly shedding its ripe proglottides and discharging ova into the alimentary canal.

The *cysticercus cellulosæ* is chiefly known as a denizen of the flesh of pigs, in which it is sometimes present in vast numbers, rendering the pork 'measly.' And it is almost exclusively to the use of such pork in an uncooked or imperfectly cooked condition that the development of tænia solium in the human intestine is due. In the comparatively

rare cases in which the cysticercus infests the human body it seems to occur mainly in the muscles, connective tissue, brain, eye, and serous membranes. It exists under the form of a round or ovoid vesicle, about the size of a pea or bean, but sometimes attaining that of a marble, formed of a transparent plastic membrane containing a clear limpid fluid. Springing from one side of this vesicle is a wrinkled cylindrical neck, terminating in a head precisely similar to that of the tænia solium. The neck and head protrude externally after death, and may be made to protrude by pressure during life; but in the ordinary living state they are retracted within the vesicle, lying coiled up against one side of it. The conversion of the six-hooked embryo into the perfect cystic scolex occupies about two and a half months; and the scolex may remain living in the tissues of its host for many years.

b. Tænia mediocanellata, and *Cysticercus tæniæ m. c.*—This tape-worm, which was formerly confounded with the last, is equally common. It presents a general resemblance to it both anatomically and in habit; but it presents also characteristic differences. It attains a greater length, its joints are longer and broader, and its head also is about three times as thick. The head, moreover, is furnished with four large round pigmented suckers, but with neither rostellum nor armature of hooklets; the uterus, though exhibiting the same general arrangement as that of the tænia solium, is characterised by much more numerous and finer transverse processes; and the ova, instead of being round, are oval, the long diameter differing little from the diameter of the egg of the tænia solium, the short diameter measuring about $\frac{1}{850}$ inch.

The *cysticercus* of this tape-worm seems especially to affect the ox, and it is, therefore, to the eating of imperfectly-cooked beef that the introduction of the scolex into the intestines is due. The cysticercus is a small oval vesicle, similar to that of the cysticercus cellulosæ, but smaller than it, and furnished with a neck and head, of which the latter is identical with that of the adult sexual strobilus. It is not known to affect the human being.

c. Bothriocephalus latus.—This tape-worm is limited in its range to certain European countries, especially Belgium, Holland, Poland, Prussia, Russia, Sweden, and Switzerland. It is the largest of all tape-worms—not unfrequently attaining a length of twenty-five feet and upwards, and a breadth of more than half an inch at its widest part. The head is ovoid in form, measuring about $\frac{1}{10}$ inch in length by $\frac{1}{16}$ in breadth, and presenting two opposite longitudinal deep grooves or suckers, but no hooklets. The neck, which is comparatively narrow, soon becomes transversely wrinkled; and as it widens out and retreats from the head, the wrinkles divide it into successive segments. The segments gradually increase in all their dimensions, but for the most part continue of greater width than length; and are specially characterised, not merely by their general form, but by the facts that the

genital pore is placed in the centre of each flat surface, and that the uterus forms a small rosette, of which this pore is the centre. The ova never become matured within the uterus, and usually escape thence into the bowel, while the proglottis is still a portion of the strobilus. After the discharge of their ova, the joints diminish in size, and become shrivelled and elongated. The eggs are of oval form, measuring about $\frac{1}{170}$ inch by $\frac{1}{170}$, and have a firm brown shell, which opens by a lid at one end. The embryo, on its escape from the egg, is provided with cilia, which it soon loses, and then presents the common six-hooked character. The cysticercus of this tape-worm is at present unknown, as also is its habitat. It is generally believed to infest some fish or other aquatic animal.

Symptoms.—The symptoms to which tape-worms give rise are on the whole trivial and unimportant. Many of those who are infested by them enjoy perfectly good health; and many more make them the scapegoats of all their ailments (imaginary or otherwise) from which they happen to suffer during the residence of these parasites within them. Among the symptoms which are referred to their presence are: pain and discomfort in the belly, capricious appetite, variable condition of bowels, itching at the nose and anus, depression of spirits, emaciation, and hysterical, epileptic, or other nervous phenomena. The list might easily be extended; but when we consider that, notwithstanding all the evil influences which have been attributed to them, they are probably never diagnosed or even suspected to be present until their joints have been detected in the stools, it is obvious how vague and on the whole how apocryphal all these influences are. The only way in which the presence of tape-worms can be recognised is by the discovery, of their joints either in the stools or about the anus or on the body-linen, and of their eggs by the microscopic examination of the fæces.

The cysticercus cellulosæ causes no symptoms unless it be lodged in some delicate or vital organ, such as the eye or cortex of the brain, and even then the symptoms are not specific.

Treatment.—Many remedies have been employed for the purpose of getting rid of tape-worms; but those on which reliance is now chiefly placed are the male fern, the bark of the pomegranate root, kousso and kamala. The liquid extract of male fern may be administered in a dose of from 30 to 120 minims early in the morning on an empty stomach, and be followed shortly by a full dose of castor oil. And if this procedure prove insufficient, the treatment may be repeated either on the next day or from time to time at short intervals. The other varieties of vermifuge are employed in much the same manner. The decoction of pomegranate root is given in large quantities—a pint or more, for example—in two or three portions at short intervals. Kousso is administered similarly, excepting that the powder from which the infusion is made is usually drunk with the infusion. The dose of this is

from four to eight ounces. These drugs rarely fail to bring away large portions of the worm; but no absolute cure is effected, unless the head be brought away as well. This, however, from its small size is very apt to escape detection. It is consequently of great importance to make a very careful inspection of the evacuations which are passed subsequently to the administration of vermifuge drugs. In order to prevent the development of tape-worms in the intestine, it is necessary that flesh, and especially those kinds of flesh which are known to harbour their vesicular representatives, should always be eaten in a well-cooked condition. Underdone and merely smoke-dried beef and pork should certainly be avoided.

With respect to the cysticercus cellulosæ, unless it occupies some superficial part, and thus lies within reach of surgical treatment, we can do nothing for the patient's relief. The ova of the tænia solium are probably taken into the stomach with uncooked vegetables, salads and the like, and hence those who wish to guard against them should content themselves with cooked vegetables only.

3. *Tænia Echinococcus and Hydatid.*

The *tænia echinococcus* is only known to affect the dog and wolf, and is usually found in them in large numbers, adhering to the mucous membrane of the duodenum and jejunum. It is peculiar in comprising in its perfect form four joints only, and in having a length of little more than a quarter of an inch. The first joint is that which includes the head. This measures about $\frac{1}{100}$ inch wide, and is furnished with four suckers, and a central rostellum, provided with a double coronet of hooklets, which vary from thirty to forty in number. The fourth segment, which is as long as the other three joints together, is usually alone furnished with sexual organs and a marginal reproductive papilla. The eggs, like those of the tænia solium, are globular and thick-walled.

The *cysticercus* or larval form of this tape-worm, commonly known as an *hydatid*, is one of the most dangerous to life of all parasites. It differs from the cysticerci of other tape-worms in the fact that it is capable, on the one hand, of almost indefinite increase of size, on the other of almost indefinite multiplication by the formation of gemmæ. Its favourite haunt is the liver, next to that the subperitoneal tissue, and then probably the lungs, kidneys, and brain. It is found also in the heart, muscles, and bones; and indeed has occasionally been detected in almost every organ and tissue of the body. In its early condition it is a small globular cyst, with transparent laminated walls and finely granular contents. At a later stage the cyst has acquired considerable dimensions, the walls have become thick and the contents fluid. The walls are formed of two portions: an outer, comparatively thick, which is transparent, elastic, tremulous, and beautifully laminated; an inner, which is thin, delicate, and composed mainly of deli-

cate cells, often containing oval or globular refractive bodies. The fluid contents are limpid, colourless, of low specific gravity, and peculiar in containing a considerable quantity of salt, and, as a rule, no albumen. In some cases the hydatid experiences no other change than increase of size. Much more commonly, however, it undergoes further development. This consists principally in the formation of other cysts in the substance of its walls, sometimes towards the outer aspect, sometimes in the mid-region, sometimes towards the inner aspect, and then often in connection with the cellular lamina. These secondary cysts in many cases repeat in their growth all the characters of the parent hydatid. In many they remain permanently devoid of the outer laminated wall. But whether they continue thus simple or not, and especially in the former case, their contents often undergo gradual conversion into one or several echinococci or scolex heads—the cysts then forming what are sometimes termed brood-capsules, and remaining permanently of minute, if not microscopic size. The results of these processes going on almost indefinitely are very various. Thus, in some cases, an hydatid tumour as large, perhaps, as a child's head, consists of one hydatid cyst only, with a larger or smaller number of brood-capsules, springing bud-like from its inner surface; in other cases an indefinite production of barren hydatid cysts takes place, so that the original cyst becomes filled with innumerable daughter cysts, each of which has, like its parent, the capacity for growth and the production of new cysts by gemmation; in other cases again (and these are the most common) the parent hydatid ultimately contains both barren and fertile cysts. It occasionally happens: that the hydatids formed in the walls of the primary cyst, instead of projecting at its inner surface, and finally getting shed into its cavity, project outwards and thus form separate tumours; and further, that in the liver the hydatid growth forms a multilocular mass, in which it may be assumed that the walls of the separate cysts are, as it were, fused together.

The scolex or *echinococcus* in its living condition is a rounded or ovoid body, from $\frac{1}{100}$ to $\frac{1}{60}$ inch in length, attached by a depression at one extremity to a cord which fixes it to the wall of the brood-capsule, and presenting at the other extremity an orifice communicating with a central vertical canal, at the bottom of which lie the retracted rostellum and hooklets, and on the sides of which is seated the inverted suctorial region. When the animal is dead all the latter organs are protruded, and the form which it then presents is as nearly as possible that of the first joint of the tænia; the small vesicular body is surmounted by a kind of quadrilateral expansion, the angles of which are occupied by suckers and from the centre of which arise the rostellum and the crown of hooklets. The latter vary in length between $\frac{1}{780}$ and $\frac{1}{330}$ inch. The growth of hydatids is for the most part very slow; they enjoy, however, a long life, often continuing to grow and

multiply for five, ten, fifteen years or more, and it may be during the whole period of the life (however much prolonged) of their host. In many cases, however, they (like all other imbedded parasites) undergo spontaneous dissolution; in which case the tumours shrink; the cyst walls get flattened and compressed against one another; the echinococci break down, shedding their hooklets; the surrounding tissues become thickened and indurated; and an abundant deposition of calcareous matter pervades the capsule and even the hydatid mass.

For the *symptoms* and *treatment* of hydatid tumours we must refer to the diseases of the several organs in which they occur. We need only mention here that no drug that we know of given by the mouth is capable of affecting these creatures injuriously; and that, in reference to prophylaxis, the chief if not sole source from whence we derive them is the excrement of dogs.

B. *Round Worms.* (*Nœmatoda.*)

1. *General Account.*

These are elongated round worms, presenting a distinct integument marked with fine transverse rugæ, a perivisceral cavity, a distinct alimentary canal, provided with a mouth at one extremity, and for the most part an anus on the ventral aspect close to the opposite extremity, and sexual organs. The sexes are always separate; in the male (which is smaller than the female) the genital pore opens in immediate relation with the anus; in the female, the vaginal orifice is usually situated about the middle of the ventral aspect.

It is certain that some species of this sub-class of parasites need (like the tæniada) two successive hosts for the completion of their cycle of existence. The trichina spiralis, for example, passes an asexual life imbedded in the voluntary muscles of the pig or man; and there, unless the affected flesh become the food of some other animal, after a while it dies. If, however, the trichinous flesh be eaten, the cysts in which the trichinæ are contained become dissolved; the animals are set free, rapidly acquire sexual organs and copulate; ova are developed and fertilised and hatched while still in the uterus; and the living embryos on their birth, instead of remaining in the bowel, undergo an active migration through its walls, and ere long reach the tissues in which they are to become imbedded. As regards the ascaris lumbricoides, there is good reason to believe: not only that the ova which are shed in vast numbers into the intestinal canal which they occupy are never hatched there; but that they are taken into the body of some other animal, probably one of the invertebrata, within which (possibly imbedded in the parenchyma) they complete one phase of their existence. There is reason even to doubt whether the common thread-

worms multiply in the region which they infest—whether the ova which they discharge so abundantly become hatched within the anus. Dr. Ransom indeed suggests that in many cases (among young children especially) there may be a kind of re-infection due to the conveyance of the ova from the anus to the mouth by the fingers.

Among the nematode worms are included the *Ascaris lumbricoides*, the *Oxyuris vermicularis*, the *Dochmius duodenalis*, the *Trichocephalus dispar*, the *Trichina spiralis*, and the *Filaria sanguinis hominis*, which will now engage our attention; the *Filaria medinensis*, whose effects are surgical; and several others, including the *Strongylus gigas*, which are of rare or doubtful occurrence in man.

2. *Common Round Worm.* (*Ascaris Lumbricoides.*)

This well-known worm varies in size: in the female from 10 to 14 inches long, and from $\frac{1}{4}$ to $\frac{1}{3}$ inch thick; and in the male from 4 to 6 inches long, with a correspondingly small diameter. The worm is cylindrical, tapering to either end, white with a brownish or reddish tinge, and invested in a firm elastic integument. The ova, of which each female discharges, on the average, 160000 daily, are oval, measuring $\frac{1}{140}$ inch by $\frac{1}{440}$ inch. They have a thick, firm, nodulated shell, and contain, as ordinarily passed from the bowel, no trace of embryo.

This ascaris is found in some few animals besides man. In man its special habitat is the small intestine; but it is apt to wander, and thus to reach the colon on the one hand, or the stomach on the other; and, indeed, it has been known to find its way into the hepatic or pancreatic duct, and also into the nose or larynx. It has been often asserted that it occasionally perforates the wall of the bowel, and thus finds its way into the peritoneum, or some sinus or abscess. It is now, however, generally held that when found in such situations it has simply passed thither through an accidental perforation. The number of ascarides present at the same time rarely exceeds five or six. But authentic cases are on record in which the bowels have been infested with hundreds and even thousands of them. The time during which a worm remains a denizen of the bowels is probably never more than a few months.

Symptoms.—Innumerable symptoms have been referred to the presence of these parasites, as to that of the tæniæ; but there is no doubt that in the great majority of cases they give no indication whatever of their presence, which is not even suspected until one or more have been discharged. The symptoms which might reasonably be referred to them are those of intestinal irritation, which in children are always liable to be attended with some degree of fever and more or less cerebral disturbance. When these worms are harboured in large numbers there is no doubt that they may induce very grave gastro-

enteritic symptoms, but symptoms which are in no sense characteristic. Occasionally too a mass of them causes complete occlusion of the bowel, as any other concretion may do. In all cases where these worms are suspected to be present, and always before a cure can be safely announced, the fæces should be subjected to microscopic examination; when, if they be present, the innumerable eggs which are discharged can scarcely be overlooked. Ascarides are occasionally vomited.

Treatment.—Various remedies have been employed with the object of getting rid of ascarides, and among them those which are in common use against tape-worms. The mucuna pruriens also was formerly much esteemed. The remedy now mainly relied upon is santonica, and more especially its active principle, santonin, of which from one to three grains may be given twice daily to a child, and about twice that quantity to an adult. Violent purgatives are of little or no use; an occasional laxative may, however, be given with advantage during the course of treatment by santonin.

3. *Common Thread-worm or Seat-worm.* (*Oxyuris Vermicularis.*)

This creature is minute, fusiform, white, and, as its popular name implies, thread-like. The female varies from $\frac{1}{3}$ to $\frac{1}{2}$ inch in length, and presents a comparatively long attenuated caudal extremity. The male is about half the length of the female, and its caudal extremity is simply fusiform. The ova are oval, but unsymmetrical, measuring $\frac{1}{190}$ inch by $\frac{1}{110}$. They present a firm shell with three laminæ, of which one is absent at one of the poles. At the time of deposition they contain a developing embryo. Thread-worms are probably the most common of all intestinal parasites; they infest persons of all ages, but children much more frequently than adults. They occur habitually in the colon alone, and indeed are limited almost exclusively to the rectum. They are often present in enormous numbers. The females are apt to migrate through the anus, and to deposit their eggs on the skin and among the hairs in its vicinity; they occasionally also find their way into the vulva, vagina, and urethra.

Symptoms.—The chief symptom to which oxyurides give rise is troublesome itching about the anus, coming on mainly in the evening; it is often intolerable, especially if they have migrated into the vulva or urethra. Children affected with them are said also to suffer from itching at the nose; and many of the functional disturbances which have been attributed to the presence of more formidable parasites have also been attributed to them. The diagnosis of thread-worms can easily be verified by their discovery and that of their ova in the fæces.

Treatment.—Local measures are usually amply sufficient for getting rid of thread-worms. The injection of a strong infusion of green tea, quassia, or any other bitter, or of a solution of perchloride of iron or

salt, repeated if need be from time to time, is usually efficacious; the use of mercurial ointments or other parasiticide applications in and around the anus may be serviceable for the destruction of the ova in these situations; in addition to which measures occasional purgatives may be administered, and the patient put under a course of tonics.

4. *Whip-worm.* (*Trichocephalus Dispar.*)

This is said to be not uncommon. But it is rarely met with in this country. It is especially characterised by having a comparatively thick cylindrical body, terminating anteriorly in a delicate filiform process, which forms about two-thirds of the entire length of the parasite. The male measures about one and a half inches long and the female about two inches. The latter is very prolific. The eggs are oval, about $\frac{1}{400}$ inch by $\frac{1}{1100}$, pointed at either end, and presenting a firm brownish-yellow shell. The normal habitat of this worm appears to be the cæcum, to which it attaches itself by burying its thread-like neck in the substance of the mucous membrane.

It does not appear to give rise to any symptoms, and, indeed, its presence can only be diagnosed by the discovery of ova in the fæces.

No treatment is needed; the measures most likely, however, to be efficacious in effecting its dislodgment are those already discussed in relation to the ascaris.

5. *Dochmius Duodenalis.* (*Sclerostoma Duodenale.*)

This is a small cylindrical worm of which the female slightly exceeds half an inch in length; the male is somewhat smaller. It has not been met with in this country, but is not uncommon in hot climates. The inhabitants of Italy and Egypt are especially liable to be infested with it. It appears to take up its abode in the duodenum and upper part of the jejunum, where it may be present in vast numbers, fixing itself to the villi, sucking the blood, and causing hemorrhages and dangerous (sometimes fatal) anæmia.

No efficacious treatment is known. The measures recommended for the expulsion of the ascaris may, however, be tried.

6. *Trichina Spiralis. Trichinosis.*

The trichina spiralis was known only as an occasional inhabitant of the muscular tissue, and regarded as a mere pathological curiosity until the year 1860; when a case that came under the observation of Dr. Zenker, of Dresden, conclusively showed that, however harmless the encysted parasite might be, the gravest symptoms, and even death itself, might be caused, after its reception into the bowels, during the processes of reproduction which ensued there, and of migration of the

young animals into the voluntary muscles. Since that period the 'trichina disease' or '*trichinosis*' has been fully recognised and frequently observed.

The trichina spiralis is met with in the muscular tissue in the form of a minute worm, measuring about $\frac{1}{35}$ inch in length. Its anterior extremity is somewhat pointed, its posterior thick and rounded; it presents immature sexual organs, and lies coiled up in the interior of an oval cyst. This cyst, which is no essential part of the parasite, but forms around it after it has taken up its quarters, measures about $\frac{1}{70}$ inch in length, is thick-walled, laminated, transparent, and generally studded externally, especially about the poles, with granular calcareous matter. The trichina-cysts occupy the striped muscles of the body, and are often especially abundant in those of the larynx. The heart, however, is rarely if ever involved. They appear in the muscles as minute white grains distinctly visible to the naked eye, of which the long diameter corresponds to the direction of the fibres. Their apparent size is usually increased by the fact of the development of groups of fat-cells in relation with either extremity. The numbers present vary, of course, in different cases. In a cat experimented upon by Leuckart each ounce of muscle was calculated to contain 325000 trichinæ; and on the basis of this calculation Dr. Cobbold estimates that a man of medium bulk may easily harbour 20,000000. The length of time during which these larval trichinæ retain their vitality is very uncertain. There is no doubt, however, that they may live in the muscular tissue for many years, and that they retain life after the death of their host, and even after the putrefaction and disintegration of his tissues. They do, however, perish *in situ* sooner or later, and then usually undergo calcareous changes. Trichinæ have been discovered in the flesh of various animals besides man, but mainly in that of the pig; and indeed it is from the use of trichinous pork that man becomes affected. The trichina-capsules swallowed with the flesh are dissolved by the gastric juice, and the contained parasites are set free. These then undergo rapid development and attain sexual maturity—the female ultimately acquiring a length of $\frac{1}{8}$ inch, the male a length of not more than $\frac{1}{18}$ inch. The ova are hatched within the uterus; and the living embryos, escaping thence into the intestinal canal of the host, at once commence active migration. They attach themselves to the mucous membrane, eat their way through the intestinal walls, and either continue to burrow through all the tissues which lie between them and their destination, or, what is more probable, find their way into the small vessels and lymphatics of the bowels, and are thence conveyed all over the organism. They have been found during this period in almost all parts of the body—in the intestinal walls, abdominal cavity, mesentery and mesenteric glands, and connective tissue, and in an as yet unencapsuled condition in the muscular tissue itself.

The progress of events above described is always very rapid. The

immature trichinæ taken into the stomach become mature on the second day; on the sixth and following days, up to the end of the second or even third week, the embryos are born and commence operations; they probably reach their destination in the course of a week or two, and by the end of a month or a little more have come to the conclusion of their labours.

Symptoms and progress.—The symptoms which attend the development and migration of trichinæ are on the whole very remarkable and suggestive of the disease. They comprise, in the first instance, those of gastro-intestinal disturbance; in the next those of general muscular inflammation; and, associated with these, febrile phenomena.

Within a day or two, or at most a week, after the ingestion of trichinous flesh symptoms not unlike those of enteric fever manifest themselves. The patient suffers from thirst and loss of appetite, with perhaps nausea and sickness; and from colicky pains in the abdomen, with constipation or irregularity of the bowels, or actual diarrhœa. His tongue is coated; and there is more or less mental and muscular prostration, with elevation of temperature, and acceleration of the heart's action. These symptoms, which are ill-defined in the beginning, become aggravated day by day during the first week or ten days of the patient's illness, and in some cases culminate in those of fatal enteritis or peritonitis. More commonly, however, about the end of this time they undergo some remission and then gradually subside. But while they are in progress, and even it may be in progress of amendment, other symptoms due to the migration of the parasite develop themselves and soon overshadow the rest. These consist mainly in gradually increasing pain and tenderness, swelling and stiffness of the voluntary muscles, together with œdema of the subcutaneous connective tissue, copious perspirations, and aggravation of debility and febrile disturbance. The pains have some resemblance to those of rheumatism, but they occupy the fleshy parts of the limbs and trunk and not the joints. The general stiffness, tenderness, and swelling lead to flexion and immobility of the limbs, and it may be to impediment to the due action of the muscles of the tongue and larynx and of those concerned in respiration. Dropsy, which is one of the earliest indications of the migration of the parasites, commences in the face, particularly in the eyelids, then attacks the extremities, and subsequently probably becomes general, involving even the serous cavities. Hoarseness, or loss of voice, and dyspnœa are not uncommon. The temperature presents great differences. It some cases it rarely, if ever, rises above the normal. In severe cases, however, it may reach 104°, 105°, or even 106°, but then varies greatly and irregularly from day to day, and always presents considerable morning remissions.

The total duration of the disease varies. In mild cases the patient recovers in the course of a month; in many cases recovery is delayed to the end of six weeks or two months; and occasionally the patient

continues ill for three or even four months. The trichinous disease varies very greatly in severity—its severity depending mainly on the number of living parasites which the patient receives into his bowels. In some instances there are few or no symptoms to attract attention; in some outbreaks where many persons have been attacked the mortality has been very light; while in others the death-rate has been twenty or twenty-five per cent. Death may result from enteritis, peritonitis, or pneumonia, or from the debility which the progress of the disease gradually induces; and may occur at any time between the fifth or sixth day and the end of the sixth week.

The presence of trichinosis in its acute stage may possibly be confirmed by the discovery of parasites in the intestinal discharges, or by the extraction by means of a suitable instrument (harpoon) of fragments of striped muscular tissue. The under part of the tongue has been specially recommended for exploration. No symptoms attend the presence of the encapsuled parasites in the muscles. The diseases with which trichinosis is most liable to be confounded are enteric fever, acute tuberculosis, and acute rheumatism; but the distinctions between it and them are obvious.

Treatment.—We have not, so far as is known, any power to destroy trichinæ, whether in the intestines or in the substance of the living frame. It is of course possible that remedies useful against other intestinal parasites may be useful against these, supposing their presence to be detected sufficiently early to justify us in attempting to dislodge them. As a general rule, therefore, we can only treat trichinosis on the same principles as we treat other affections made up of local inflammatory conditions and general fever. But we can also employ prophylactic measures; and these are, fortunately, sufficiently simple. They consist in the avoidance of pork which presents the characteristic appearances of the disease, and especially of pork which is not well and completely cooked. The mere toasting to which ham and bacon are frequently subjected is insufficient to destroy the vitality of the trichina. Smoked ham and German sausages are, unless they have been cooked, sources of considerable danger. It is mainly in Germany, where pork, raw, smoke-dried, or imperfectly cooked, is a common article of diet, that trichinosis is known to occur.

7. *Filaria Sanguinis Hominis.*[1]

In the year 1870, Dr. Lewis, of Calcutta, observed that certain minute nematoid worms were constantly present in the urine of persons suffering from chyluria; and two years later he published a

[1] Dr. T. R. Lewis, 'On a hæmatozoon inhabiting human blood: its relation to chyluria and other diseases'—Calcutta, 1872. 'The Indian Annals of Medical Science,' No. xxxiv., July 1875. And 'The Lancet,' vol. ii., 1877. Also Dr. Cobbold, 'The Lancet,' vol. ii., 1877.

short monograph, in which, whilst confirming his former statements, he showed that the blood of chyluric patients contained the same parasite in more or less abundance. Later, namely in 1875, he wrote a paper demonstrating the presence of the same animal in the blood and diseased tissues of persons suffering from that form of spurious elephantiasis of the scrotum, labia, and leg which we have already described under the name of elephantiasis lymphangiectodes. This parasite, to which he gave the name of 'filaria sanguinis hominis,' measures on the average $\frac{1}{3500}$ inch in diameter and $\frac{1}{75}$ inch in length; has a rounded anterior extremity, a pointed tail, a definite structureless envelope, with slightly granular contents, and no sexual organs. The total absence of these organs showed of course that the filariæ were immature; and it was important therefore to endeavour to ascertain the character and habitat of the parent worms, and the source of infection.

Filariæ much like the above have been many times observed in the blood of the lower animals, more especially dogs, in France, China, and America. In such cases both MM. Grube and Delafond and Professor Leidy have discovered in the right ventricle of the heart mature worms varying from 5 to 7 inches in length, with a diameter ranging from $\frac{1}{25}$ to $\frac{1}{16}$ inch; and in the last of Dr. Lewis's papers above referred to he gives an account of a series of original investigations with regard to the prevalence of a similar parasitic disease in the pariah dogs of India. He shows that a considerable number of them present in their blood hæmatozoa which are identical in appearance with those found in man; excepting that they are a little smaller, and do not appear to possess the same distinct structureless envelope. And on dissection he found: 1st, that along both the œsophagus and the thoracic aorta were firm fibrous-looking tumours, varying from the size of a pea to that of a walnut, within each one of which were from one to six worms of a pinkish tinge—the males varying between 1 and 2 inches in length, and between $\frac{1}{50}$ and $\frac{1}{40}$ inch in diameter, the females measuring from 2 to $3\frac{1}{2}$ inches long, and from $\frac{1}{30}$ to $\frac{1}{25}$ inch thick; and 2nd, that also studding the aorta were nodules, from the size of millet seed to that of a pea, containing immature but growing worms from $\frac{1}{10}$ inch in length upwards, and also, more or less abundant, scar-like depressions due either to the death of the parasite or to its migration and consequent retrogressive changes.

Since the above observations were made, Dr. Bancroft, of Queensland, late in 1876 and early in 1877, discovered almost by accident first in a lymphatic abscess in the arm and next in a hydrocele of the cord, mature female filariæ in length, appearance, and structure not unlike those found by Dr. Lewis in the pariah dog, but apparently a good deal thinner. A little later (August 1877) Dr. Lewis found in dissecting a 'nævoid' scrotal tumour removed from a patient whose blood contained filariæ two mature specimens of the worm. With these were found ova, thin-walled, oval in form, and measuring

from $\frac{1}{1800}$ to $\frac{1}{1000}$ inch in the long diameter. In this as well as in former dissections numerous embryos were found in the diseased tissues. He has also found them in great abundance in the renal arteries and veins and in the substance of the kidneys.

Whether the filariæ discovered severally in pariah dogs, Indians and Australians, are identical must at present be regarded as open to doubt. At any rate, while no pathognomonic symptoms attend their presence in the dog, and it is uncertain as regards the Australian filaria whether its immature representatives inhabit the blood; it is manifest that in India not only has the mature worm been found in the tissues, but the asexual embryos infest the blood, and characteristic consequences—mainly chyluria, and a spurious form of elephantiasis usually implicating the scrotum, labia, or lower extremities—are apt to ensue.

The above facts render it at any rate probable that the filaria sanguinis hominis, having found its way (most likely by the alimentary canal) into the interior of the organism, takes up its abode therein, and there breeding gives origin to the embryos which presently infest the blood. So long as the embryos remain in this fluid they undergo no further development; but whether the long duration of the disease is due to the longevity of these animalcules, or to the fact that successive families are born either from the original parents, or from embryos, which migrating from the blood-stream have undergone sexual development, is a question which cannot yet be solved.

As regards the consequences of the presence of filariæ, it is important to remark that in every case of chyluria and spurious elephantiasis which has come under his observation in India since his attention was first directed to the subject Dr. Lewis has found filariæ in the blood, veins, and discharges from the hypertrophic tissues, often in great abundance, but sometimes in such small numbers that they have only been discovered after prolonged and careful examination. There are many reasons for believing that chyluria and lymphangiectodes originating in this country are independent of parasitic disease; but the subject has not yet been investigated with sufficient completeness to justify us in asserting that it is so. We shall revert to this subject when we come to speak of chyluria.

XIII. DEGENERATIVE AFFECTIONS OF THE STOMACH AND BOWELS.

Degenerative changes of the mucous membrane play, no doubt, an important part in the various chronic disturbances of the stomach and bowels, to which the terms dyspepsia, diarrhœa, and the like are usually applied. They follow upon chronic inflammation and other

persistent lesions of the alimentary mucous membrane, and occasionally depend on the presence of certain forms of cachexiæ. They comprise mainly: fatty degeneration and wasting of the glands, associated either with general atrophy of the mucous membrane or with increased development of fibroid tissue; and lardaceous change. Lardaceous degeneration affects the small intestine much more frequently than the stomach or the larger bowel, and probably never occurs except in association with advanced lardaceous disease of the liver, spleen, or kidneys. The villi chiefly suffer.

The symptoms referrible to the different kinds of degeneration do not at present admit of identification.

XIV. OBSTRUCTION OF THE STOMACH.

Causation and morbid anatomy.—Many of the morbid conditions which have already been described involve more or less serious impediment to the due performance of the mechanical functions of the stomach, and consequently to the due transmission of its contents onwards; and indeed the symptoms arising from obstruction form an important part of their clinical history.

Obstruction occurs chiefly at the pyloric and cardiac orifices; it may, however, arise in some intermediate part. It may be due to mere fibroid thickening or cirrhosis; to malignant disease; to the cicatrisation of large ulcers; to the pressure of external tumours; to accumulation of hair, cocoa-nut fibres, or other solid matters which have from time to time been swallowed; to paralysis or spasm.

The consequences of obstruction at the cardiac orifice have already been considered under the head of œsophageal disease; they are dilatation and hypertrophy of the œsophagus, and contraction and atrophy of the stomach. In pyloric obstruction the food which is received into the stomach tends to accumulate within it, and thus to involve its dilatation and hypertrophy. The dilatation under such circumstances is sometimes enormous. If the impediment occupy some intermediate position, its influence over the form and functions of the stomach will, according to circumstances, approximate either to that of cardiac or to that of pyloric obstruction. In some cases habitual starvation causes more or less permanent general contraction of the stomach; in some cases habitual over-eating or accidental and ill-understood conditions involve extreme dilatation of the organ; and both of these states are apt to be attended with many of the phenomena of pyloric stricture.

Symptoms and progress.—The symptoms of cardiac obstruction are, mainly, ability to perform the act of deglutition, and in rapid succession to this act more or less uneasiness, referrible to the situation of

the cardiac orifice, and the rejection of the matters swallowed by a process which generally has more resemblance to eructation than to vomiting. The patient probably has a good appetite, but cannot gratify it, and suffers from all the usual symptoms of starvation. In most cases the obstruction is partial only, and more or less food finds its way into the stomach. In some the retention of food in the dilated œsophagus lasts for a considerable time, and the retained matters prior to their rejection undergo putrefaction or fermentation, and become mixed with mucus secreted from the surface of the tube. The epigastric region shrinks, owing to the necessary contraction of the starved stomach.

The symptoms referrible to obstructive disease of the pylorus are, in many important respects, different from the above. The patient can swallow with ease, and anything that is swallowed finds its way without difficulty into the stomach; whence (according to the degree of impediment present) it is in part transmitted more or less slowly onwards, in part, after a while (it may be half an hour, an hour, several hours, occasionally even several days), and after having caused more or less gastric uneasiness, rejected by vomiting. The characters of the vomited matters depend largely upon the length of time they have been retained. If they be discharged shortly after ingestion they consist mainly of partially digested food mingled with the normal secretions of the stomach; if after a long interval they have generally undergone putrefactive or fermentative changes, are more or less fetid, abnormally acid, and probably contain sarcinæ or the yeast-fungus, or both. Their quantity varies considerably, and sometimes amounts to several pints. The appetite is more likely to suffer in pyloric than in cardiac obstruction, but is not unfrequently retained. The dilatation of stomach which attends this affection reveals itself locally by protrusion of those parts of the abdominal surface with which the organ lies in contact, and probably by displacement of the diaphragm upwards. If it be moderate it causes fulness of the epigastric region only; if it be considerable, the body of the organ descends, forming a loop between the pyloric and cardiac orifices, and the chief distension then probably occupies the umbilical and hypochondriac regions —the epigastrium presenting a comparative depression. In some cases the dilated stomach occupies nearly the whole of the anterior part of the abdomen. That the distension is due to the stomach is shown: partly by its situation; partly (if it be considerable) by its looped form; partly by observing the peristaltic movements, which are generally easy of recognition and admit of being readily excited; and partly by the evidences which palpation and percussion give of a large cavity containing air and fluid.

The symptoms due to general contraction of the stomach are especially: inability to take food, excepting in small quantities; irritability of the organ; and tendency to vomit shortly after the ingestion of food.

Simple dilatation of the stomach differs little, if at all, in its symptoms from incomplete pyloric obstruction.

In all of the above cases, starvation, emaciation, asthenia, and the phenomena which attend these conditions supervene with more or less rapidity; further various complications are apt to arise in their course and to prove fatal, among which may be mentioned gastritis and peritonitis.

Treatment.—The treatment of obstruction must vary somewhat in different cases, in dependence partly on the site of obstruction, partly on its cause. If it be at the cardiac orifice, the careful passage of bougies may serve to maintain an available passage; and, failing this, the question of making an opening into the stomach at the epigastrium, and of feeding the patient through this opening, may be entertained. If it be at the pyloric orifice, or if the case be one of simple dilatation, it may become necessary under certain circumstances to empty the distended organ by means of the stomach pump, or to let off some of the gaseous accumulation by acupuncture through the abdominal walls. In all cases it is important to give food in small quantities at a time, and in the fluid or semifluid condition, in which form it most readily passes through a narrowed or strictured orifice. In cases of pyloric disease or passive dilatation, it is further important: that the stomach be not overburdened with food, and hence that this be administered in a concentrated form; that putrefaction and fermentation be obviated by the use of appropriate remedies, such as creasote and the hyposulphites; and that tendency to vomit and gastric uneasiness be met by the measures elsewhere recommended for these purposes. Lastly, it is often necessary to feed the patient per rectum.

XV. OBSTRUCTION OF THE BOWELS.

The affections which are here to be treated of present many features in common with enteritis, and their description is not unfrequently included in the description of that disease. Enteritis does indeed occur at some period or other in the course of most of them; but their special claim to form a group apart consists in the fact of the existence in all of them of some mechanical impediment to the transmission of the contents of the bowels, in connection with which enteritis is apt to supervene. They are: stricture, compression and traction, twisting, internal strangulation, impaction of foreign bodies, and intussusception. It will be convenient to preface our observations under these heads with some remarks upon their common factor, constipation.

A. *Constipation.*

Causation, morbid anatomy, and symptoms.—It may doubtless be accepted as a general rule that persons enjoying robust health, and undisturbed in the due performance of their various functions, have an alvine evacuation at least once daily. Yet many, who at any rate seem equally healthy, have their bowels habitually relieved every two or three days only, or even but once a week or fortnight. Cases are not altogether rare in which some degree of good health has been maintained for many years, although fæcal evacuations have during that time occurred only at intervals of six weeks or two months. Cases, indeed, are on record in which the interval between successive evacuations has been extended to a period of three months. In most cases, however, retention beyond the usual period is apt to produce, not only local uneasiness, such as fulness, heat, tendency to piles, and flatulence, but also some degree of general disturbance indicated by headache, foul breath, loss of appetite and dyspeptic symptoms, and not unfrequently ends with the occurrence of more or less tenesmus and even slight dysenteric diarrhœa. Habitual constipation is usually attended with chronic discomforts of the same kind; but it leads also to more or less permanent hypertrophy and dilatation of the rectum—conditions which render this tube less efficient for the performance of its expulsive duties. When constipation is of long duration, not only the rectum but the whole of the large intestine may become dilated by its contents and hypertrophied, the mucous surface may be fretted into ulceration, and perforation may ensue. The dilatation is sometimes so great that the colon measures from ten to twelve inches in circumference. The chief dilatation occurs in the rectum, sigmoid flexure, and cæcum. The hypertrophy under such circumstances is general, but it is usually greatest in the sigmoid flexure and upper part of the rectum, where the thickness of the muscular coat may be $\frac{1}{8}$ inch or more.

Constipation depends on various causes. It sometimes arises temporarily from change of diet, scene, or habits, among which may be included anything interfering with the regular performance of defæcation; it is common in many diseases; and it occurs in a chronic form in chlorotic or dyspeptic girls and young women, and in persons of sedentary habits or sluggish constitution. Among local conditions which may be supposed to operate in a greater or less degree in the above cases are: first, modifications in the character of the fæces, such as we see in diabetes, where they become preternaturally dry and proportionately diminished in bulk; second, sluggishness or debility on the part of the rectum itself; and, third, affections at or in the neighbourhood of the anus, rendering defæcation painful.

B. *Stricture.*

Causation and morbid anatomy.—By this term is meant a circumscribed diminution of the calibre of the bowel. Stricture is sometimes due to spasm; but although spasm undoubtedly forms a very important element in many cases of intestinal obstruction, it is rare as an independent affection, and in this form is practically limited to the rectum and anus. Indeed, spasmodic obstruction, even in these situations, is probably always secondary to ulceration, excoriation, or morbid sensitiveness of the mucous membrane. Stricture, in the vast majority of cases, is the consequence of some organic change—cicatrisation after ulcer, cirrhosis, or some adventitious growth (malignant or other) occupying the intestinal walls. It rarely follows ulceration unless this be of large extent, or encircle the bowel. It seldom, if ever, ensues on the cicatrisation of typhoid ulcers, and not very often on the healing of tubercular ulcers. It is a much more common consequence of dysenteric or syphilitic ulceration and of the separation of a portion of bowel in intussusception. But most frequently the ulcer to which it is traceable has, so far as we know, no specific or ascertainable origin. Cicatricial stricture may form a mere ring or occupy the bowel for several inches of its length. The surface is sometimes completely cicatrised; sometimes presents still unhealed spots of ulceration, with fungous excrescences or granulations; and is often separated from the subjacent muscular coat by a more or less abundant formation of dense fibroid tissue. Stricture again, especially in the lower part of the rectum, is sometimes connected with the progress of chronic inflammatory changes or the overgrowth of fibroid tissue, not only in the walls of the rectum, but in the surrounding connective tissue of the pelvis. But by far its most frequent cause is the development of carcinoma in the substance of the intestinal walls. When carcinoma causes stricture or serious obstruction, it is for the most part a primary growth at the seat of stricture, and, like simple ulcer, sometimes forms a mere ring round the gut, sometimes involves a comparatively large area. Congenital stricture or occlusion of the bowel is mainly an affection of the anus or rectum, and falls therefore especially under the cognisance of the surgeon. It has, however, been occasionally met with in the duodenum, in the neighbourhood of the orifice of the common bile-duct.

The presence of a stricture always leads, in a greater or less degree, to certain results. These are: first, undue accumulation of fæces in the bowel above, with proportionate dilatation of its tube; second, hypertrophy of the muscular walls of the dilated bowel; and, third, diminution in calibre, and even atrophy, of the bowel below. It is an interesting fact that, in stricture of the colon, the greatest degree of dilatation is often found, not in the portion of bowel immediately above

the stricture, but in the cæcum. The tighter or longer the stricture, the more marked, other things being equal, will be the several consequences just named; and the more danger will there be of the supervention of permanent obstruction. Yet it is remarkable that tight and long strictures are often found after death in cases in which during life there has been no suspicion of their presence—a statement more particularly true of stricture of the small intestine, in which part the contents are, as a rule, semi-fluid and easy of propulsion. The lodgment of fæces above a stricture is very apt not only to prevent the complete healing of the ulcer to which originally the stricture may have been due, but to cause erosion and ulceration in the dilated bowel above, and ultimately perforation.

Stricture may arise anywhere in the bowel, but is met with in different parts with different degrees of frequency. Its occurrence as a fatal disease in the small intestine is rare. It is mainly indeed a disease of the larger bowel. According to Dr. Brinton, out of 100 fatal cases of stricture of the large intestine, 4 occur in the cæcum, 10 in the ascending colon, 11 in the transverse colon, 14 in the descending colon, 30 in the sigmoid flexure, and 30 in the rectum. It is more frequent in men than in women; and the average age at which it proves fatal is a little over forty-four.

Symptoms and progress.—The symptoms due to stricture of the small intestine are rarely sufficiently definite to justify us in diagnosing its presence. They probably comprise nothing beyond occasional colicky pains, nausea, and sickness. Even in the case of the large intestine they may be for a long time vague and inconclusive, and even misleading. The patient perhaps suffers only from occasional attacks of colicky pain, associated it may be with more or less constipation; but not unfrequently, during the earlier period, diarrhœa is a prominent symptom. If the stricture be in the lower part of the rectum, solid motions generally soon assume a narrow tape-like or pipe-like form.

The symptoms of absolute obstruction occasionally come on quite suddenly, but are more frequently preceded by more or less long-continued tendency to constipation. It sometimes also happens that the patient, previous to his final attack, has experienced one or more similar attacks, which have yielded to treatment. The symptoms of impassable stricture are: insuperable constipation; painful peristalsis, coming on periodically, and often rendering itself audible by borborygmi, and visible through the abdominal walls; abdominal distension and uneasiness, followed by nausea, vomiting, and hiccough; and death at last from simple asthenia. The vomited matters become after a while stercoraceous. Febrile symptoms and abdominal tenderness may be absent from first to last; but sometimes inflammation supervenes, or perforation takes place, and then enteritic or peritonitic symptoms become superadded. When the case is free from these or

other complications, its progress is essentially chronic, and the patient, if not improperly treated, may live for a considerable time, occasionally for several weeks.

In determining the seat of stricture, it is well to recollect that at least three-quarters of the structures of the large intestine are situated to the left of the mesial plane of the body. We need not, however, rest content with a simple calculation of chances. It is natural to believe that the distension of the bowel above the stricture, and its collapse below, should reveal themselves to manual, if not to ocular examination; and in many cases undoubtedly the form and position of a struggling, or even of a quiescent, length of distended bowel may be thus readily identified. It must not be forgotten, however, that thickness or rigidity of the abdominal walls, tenderness, the presence of tumours, and the altered positions which greatly distended tracts of bowel are apt to assume, often prevent the easy recognition of even extreme differences of intestinal dilatation and fulness. Dr. Brinton maintains that the amount of fluid which may with care be injected per anum is a very valuable guide in estimating, so far as the large intestine is concerned, the seat of stricture. This method of investigation is, however, scarcely trustworthy, unless the stricture be at a comparatively small distance from the anus. But when in this latter situation, its presence may often be ascertained by the introduction of the finger, or even of the entire hand; and if it be beyond the reach of actual touch, yet in the rectum, the careful introduction of a bougie may possibly reveal its position.

C. *Compression and Traction.*

Causation and morbid anatomy.—Under these terms we include those cases in which the bowel is obstructed either by pressure exerted on it from without, or by being dragged out of its normal position by adhesions, without being at the same time strangulated.

Here also may be placed those cases in which the rectum is obstructed and defæcation rendered painful or difficult by the pressure of a displaced or enlarged uterus, or of a uterine or ovarian tumour. Any abdominal tumour may, conceivably, have a like effect on some part or other of the alimentary canal; and even the effused blood from a ruptured aneurysm may surround and compress the duodenum or some part of the colon. But the cases here more particularly referred to are those in which obstruction is due to the embarrassment of a greater or less length of bowel, caused by the presence on its outer surface of lymph or false membrane, which binds it more or less firmly to surrounding parts, and sometimes constricts it, sometimes leads to the formation of sharp angular bends. In some of these cases the bowel has been incarcerated in a hernia, and portions of it have become invested in adhesions which attach it to the neck, or some other part

of the sac, or to the omentum; in others, the transverse colon or sigmoid flexure, or some other tract of bowel, is hooked down, as it were, by bands of lymph to the uterus, ovary, or some other structure within the pelvis; in others, again, several contiguous coils of small intestine are tightly bound together, forming a kind of tangled mass. Fatal cases always furnish distinct evidence, in the contraction and emptiness of the bowel below, and in the fulness, dilatation, and hypertrophy of the bowel above, of more or less complete obstruction. But the part in which actual obstruction has taken place, though contracted and more or less empty, frequently admits with ease of the passage of the finger, or even of some larger body. The immediate cause of obstruction, indeed, is rarely a simple tight constriction. These lesions are of far more frequent occurrence in the small intestine than in the large, and, as Dr. Fagge points out, may, from the clinical point of view, be regarded as the strictures of the smaller bowel.

Symptoms and progress.—The symptoms of these affections are almost, if not quite, identical with those of stricture. It is impossible, indeed, to make any absolute clinical distinction between them. Obstruction of the small intestine, however, is as a rule more early followed by vomiting than obstruction of the large intestine; and it may occasionally be possible, by careful examination of the surface of the abdomen, to ascertain whether abnormal distension is due entirely to dilatation of the smaller bowel, or mainly to distension of the colon.

D. *Torsion or Twisting.*

Causation and morbid anatomy.—Cases are occasionally met with in which after death a loop of bowel is found to be twisted, enormously dilated and congested, and full of fetid pitchy stuff, consisting partly of fæcal matter, partly of the bloody secretion of the affected mucous membrane. We were formerly inclined to regard the twisting of the bowel in these cases as a mere unimportant consequence of localised enteritis. We are compelled, however, to retract this opinion, and to adopt the more commonly received interpretation of these conditions: namely, that the twist is the primary lesion to which the obstruction and enteritis are secondary. The portions of bowel which are most liable to torsion are the cæcum and sigmoid flexure. The causes of torsion are obscure; but, at any rate, the accident appears to arise suddenly: the affected loop becomes twisted, once or even more than that, upon its axis, is at once rendered more or less impervious, and what is still more important the trunk-vessels which supply it, more especially the veins, by being twisted on their axis, also get occluded, and consequently congestion, inflammation, gangrene, and paralysis of the bowel ensue. When the twist is of the sigmoid flexure the loop of bowel sometimes undergoes such enormous distension that it may measure a yard in length, and three or four inches in

diameter, and may occupy the whole of the front of the abdomen. The mucous membrane and sub-mucous tissue become thickened and black with congestion and extravasated blood; and the peritoneal aspect acquires a slaty hue, and gets studded with patches and streaks of congestion and inflammatory exudation. The contents are such as have been above described. The bowel below the lesion is usually contracted and empty, while that above it is for the most part more or less dilated, and may present patches of congestion and contain matters which have regurgitated from the diseased bowel.

Symptoms and progress.—This affection is sudden in its onset, and in most cases rapidly fatal—the patient sometimes dying in the course of a day or two, rarely surviving for a week. The symptoms are in the main those of strangulated hernia. The patient generally suffers at first from severe abdominal pain, attended with constipation, vomiting, and rapid flatulent distension of the belly. And although febrile phenomena may occur, he very rapidly falls into a condition of collapse, with failing temperature, clammy perspiration, feeble and irregular pulse, sighing respiration, great muscular debility and restlessness, and withal more or less drowsiness. Generally the urine is scanty or suppressed; the bowels completely obstructed; and the patient sensible to the last.

It is remarkable that in these cases vomiting and hiccough are often absent in a greater or less degree, at any rate are not prominent symptoms; and that notwithstanding the enormous distension of the abdomen there is frequently little or no absolute pain, excepting early in the disease, and little or no tenderness. Indeed, the patient often, as in colic, is relieved by pressure or friction. It is important to add that when the torsion involves the sigmoid flexure, the long tube may easily be introduced into the diseased bowel; and that the nature of the case may possibly be diagnosed partly by this fact, partly by withdrawing some of the contents.

E. *Internal Strangulation.*

Causation and morbid anatomy.—This arises from similar causes to those which produce ordinary strangulated hernia, namely, constriction or nipping of a portion of bowel by the edges of some natural or artificial orifice through which it protrudes, with consequent arrest of the circulation of blood through it, and impediment to the passage of fæcal matters along it. Such orifices are the foramen of Winslow, congenital or acquired perforations in the mesentery, meso-colon, great omentum, or other peritoneal duplicatures, or apertures formed with the aid of neighbouring parts by bands of fibroid tissue extending from one point of the peritoneal surface to another.

Hernial protrusion through the foramen of Winslow must be exceedingly rare. Perforation of the various peritoneal duplicatures,

with passage of bowel through the perforation, is much more common, and often the result of laceration from violence. This accident is most frequent in connection with the mesentery; but it occurs also in connection with the great omentum, the meso-colon, the fold belonging to the vermiform appendix, the suspensory ligament of the liver, and the broad ligament of the uterus. There is no part of the peritoneal surface to which bands capable of producing strangulation may not be attached; but there are certain structures and certain conditions of parts with which they are specially apt to be connected. Thus the vermiform appendix often adheres to neighbouring structures, such as the mesentery, small intestine, colon, and ovary, forming a kind of loop; diverticula of the ileum become attached, usually by the apex, to the mesentery or some other adjoining part, or are prolonged to the umbilicus by a cord—a remnant of fœtal life. Again, such bands are often connected with the mesentery, the parts concerned in old ruptures, or the pelvic organs—more particularly the uterus, Fallopian tubes, and ovaries. It may further be noted that strangulation occasionally results from the slipping of a loop of intestine under the lower edge of the unusually elongated mesentery of a portion of bowel hanging low into the pelvis, or under the pedicle of an ovarian or uterine tumour. Finally, there are rare cases of internal strangulation in which the bowel protrudes into a lacerated bladder, uterus, vagina, or bowel, or through an acquired or congenital communication between the peritoneum on the one hand, and the pericardium or one of the pleuræ on the other.

The small intestine is much more frequently strangulated than the large; and of the large intestine the parts most liable to this accident are those which are most freely movable, especially the sigmoid flexure and the cæcum. Internal strangulation occurs at any age, but generally above thirty. It seems, however, that strangulation from bands connected with the vermiform appendix and diverticula are most common at a comparatively early age.

The *symptoms* of internal strangulation are identical with those of ordinary strangulated hernia, and so like those which have already been described as the symptoms of torsion and the severer forms of enteritis, that there is no need to give any special account of them.

F. *Impaction of Foreign Bodies.*

Causation and morbid anatomy.—The ordinary intestinal contents, no matter how indigestible, unwholesome, or imperfectly comminuted the ingesta from which they are derived may be, very rarely cause by their accumulation permanent intestinal obstruction; yet it is doubtless the fact that undigested masses of food do sometimes in their passage along the small intestine move with difficulty or become temporarily impacted and so produce pain and sickness and even symptoms

of obstruction. Hard foreign bodies of comparatively small size—coins, bits of bone, teeth, marbles, plum-stones, and the like—generally traverse the intestine without causing inconvenience; and occasionally sharp bodies, such as pins, prove equally innocuous. They are all, however, a source of danger, especially in the presence of strictures, above which they are apt to become lodged; or from the fact that they may slip into diverticula or the vermiform appendix, or (if they be hard and pointed) may perforate the intestinal wall, and cause, according to the seat of perforation, fatal peritonitis, circumscribed abscess, or fistula. Further, an accumulation of such bodies, as for example a large number of cherry-stones, may become welded into a mass sufficiently bulky to obstruct fatally a perfectly healthy bowel. Insoluble matters in the form of powders or fibres, when habitually swallowed even in small quantities, often concrete into hard masses. These sometimes are round or oval, and may then be termed intestinal calculi, sometimes form hollow casts of the portion of the gut in which they lie. The former are probably always found in the large intestine; the latter rarely, if ever, occupy any other portion than the rectum. Among the substances here referred to are peroxide of iron, carbonate of magnesia, imperfectly cooked starch, and oat-hairs derived from articles of food made from oats. Among cases of exceptional rarity must be named those of persons who have been in the habit of swallowing knives, pins, string, hair, or cocoa-nut fibres. These things are generally found accumulated either in the stomach or in the upper part of the small intestine, and when fibrous usually become felted and form masses which take the shape of the cavities in which they lie.

But the usual cause of fatal impaction, and that with which we now have more especially to do, is the escape of biliary concretions from the gall-bladder into the small intestine. The concretions here referred to are single stones or masses of coherent stones of considerable bulk, varying at a rough estimate from three to four inches in circumference, and from one inch to two, three or even four in length. It is obvious that concretions of this magnitude can scarcely escape from the gall-bladder *per vias naturales*; and indeed there is little doubt that their discharge is in all cases effected through an ulcerated opening between the gall-bladder and duodenum. When such a body has got into the duodenum it is carried on with the other contents of the bowel by the ordinary peristaltic movements. But its mere bulk prevents it from moving rapidly; besides which it provokes by its shape, size, and hardness some irritation if not inflammation in the mucous surface over which it passes, and more or less spasmodic contraction of the muscular walls. It hence continues its progress fitfully onwards, until finally it becomes permanently arrested, sometimes in the jejunum, but more commonly in the ileum, especially in its narrowest part, just above the ileo-cæcal valve. Then all the effects of complete

obstruction, conjoined with those of enteritis, supervene: the bowel below becomes empty, that above distended and generally more or less inflamed; while at the seat of obstruction and in its immediate neighbourhood the inflammation becomes intense, speedily extends to the peritoneal surface, and not rarely ends in gangrene and perforation. Gall-stones seldom if ever lodge in any part of the large intestine; and when large ones are found there they have probably gained an entrance directly by ulceration between the gall-bladder and transverse colon. Gall-stones are usually a product of the later periods of life; and hence obstruction from them can scarcely be looked for excepting in advanced age. It occurs, indeed, rarely before the age of fifty, and much more frequently in women than in men.

Symptoms and progress.—The symptoms due to the impaction of gall-stones are as nearly as possible identical with those of internal strangulation or enteritis. These cases, however, are amongst the most violent in their symptoms and the most rapid in their course of all cases of intestinal obstruction. Dr. Brinton calculates their average duration at five days. A clue to the nature of the case may sometimes be furnished by the occurrence of precursory symptoms due to the passage of the calculus along the bowel, and by the detection of the hard mass itself *in transitu*. The age and sex of the patient are also suggestive. There is not necessarily or even generally in these cases any history of hepatic colic or other indication of hepatic affection.

G. *Intussusception.*

Causation and morbid anatomy.—By this term is meant the descent or prolapse of a portion of the bowel into that which immediately succeeds it and is continuous with it. As the result of this accident we find the normal course of the intestine interrupted by a kind of knot, in which three successive lengths of tube lie almost concentrically one within the other—the innermost being the portion of bowel which has descended, the outermost the portion into which the descent has occurred, the middle or intermediate length that which unites the lower extremity of the former with the upper extremity of the latter. The last is of course inverted, and has its mucous aspect facing outwards and in contact with that of the outermost layer. In the descent of the inner two lengths of bowel the mesentery belonging to them is necessarily dragged down with them into the pouch which they form, and by the traction which it exerts tilts the double tube or invaginated portion of bowel so that the lower orifice instead of lying in the axis of the containing bowel faces and rests upon some portion of its circumference. The several layers generally present more or less transverse corrugation, and this condition is always most marked in the middle tube. The immediate effects of intussusception are: first, more or less obstruction to the passage of the intestinal contents; and, second,

more or less impediment to the return of blood from the inner two cylinders of involved bowel, to which the stretched and compressed mesentery belongs. Nevertheless the obstacle which an intussusception opposes is often incomplete; for it is certain that in a good many cases fæcal matters pass pretty constantly through it. The obstruction to the venous circulation very soon renders the mucous and muscular coats of the inner two tubes black or nearly so with congestion and effusion of blood; and the serous surface assumes a more or less deep slate colour. At the same time these parts become much swollen, and sanguinolent serum or blood escapes from the mucous membrane into the interval between the opposed surfaces of the outer two tubes, into the central canal, and into the bowel below the seat of disease. At a somewhat later period coagulable lymph is secreted from the opposed serous surfaces of the middle and internal layers, and these may consequently become agglutinated in their whole length. In most cases an intussusception increases for a time more or less rapidly, owing to the active peristaltic movements of the several segments engaged. This increase is so effected that that portion of the bowel which formed the lowest point of the invaginated mass in the first instance continues to form its lowest point to the end of the chapter; in other words, the middle tube of an intussusception increases in length at its upper end only, and at the expense of the outer tube. The length of bowel engaged in an intussusception varies widely. Including in one measurement the inner two tubes only, it may be said to range usually from two or three inches up to three or four feet. Dr. Peacock records a case in which there were good grounds for believing it to have been no less than twelve feet.

Intussusception may arise at any part of the intestinal canal, but it occurs in different parts with different degrees of frequency. Jejunal and ileal intussusceptions are met with almost exclusively in adults, and form collectively about one-third of the total number of fatal cases. These are usually attended with rapid strangulation of the bowel, and run a rapid and for the most part rapidly fatal course. Ileo-cæcal invagination occurs largely among young children, including babes of a few months old. According to Dr. Brinton half the total number of these cases are in children under seven years of age. This is the most common form of the disease, accounting for more than half the total number of deaths. It commences with the descent into the cavity of the cæcum of the lips of the ileo-cæcal orifice, which henceforth form the lower extremity of the invagination. As this increases the descending ileo-cæcal orifice carries down with it more and more of the ileum to form the central tube, and inverts first the cæcum and then a gradually increasing quantity of the colon to form the inverted or middle layer; and, still descending, finally, in some cases, reaches the rectum, or even protrudes from the anus. It is in this variety that the greatest length of bowel may be engaged; in it

the transverse folding of the several layers of intestine is usually well shown, especially in the middle tube, which is often also much convoluted and twisted; and in it complete strangulation and complete obstruction to the passage of fæces are comparatively rare. Intussusception, commencing in the colon, is of somewhat unfrequent occurrence; and still more rare is intussusception of the rectum. The rarest form of all, probably, is that due to the descent of the ileum *through* the ileo-cæcal orifice.

If the patient survive sufficiently long, various consequences are apt to ensue. The inflammation, which by its products unites the opposed serous surfaces of the inner two layers, may spread beyond its primary seat, and cause general peritonitis. Or after these layers have become united, a further descent of bowel may take place into the portion already invaginated. Or the extremity of the invaginated bowel may fret the wall of its containing tube and cause its ulceration and even perforation. By far the most interesting and important event is the sloughing and separation of the included layers of bowel. This occurs almost exclusively in those cases in which the small intestine is alone engaged, and the strangulation of the contained bowel is most complete. This, first deeply congested, soon becomes gangrenous, and then, after a while, getting detached either bit by bit or in mass, gradually works its way downwards, and is expelled. The separation of the slough generally leaves the upper extremity of the outermost tube firmly united at the neck of the intussusception with the lower extremity of the healthy bowel above. But during the process of separation the adhesions are apt to get ruptured, and fæcal matter to be extravasated into the peritoneal cavity. The discharge of the invaginated bowel usually occurs between the twentieth and thirtieth day; but it may take place as early as the sixth or seventh day, or be delayed for a year or two. The results of separation seem to be favourable in about half the total number of cases. In the remainder death often results sooner or later from stricture.

Symptoms and progress.—The occurrence of intussusception is attended with sudden and severe abdominal pain of a griping or twisting character, usually referred to the neighbourhood of the umbilicus. This generally ceases in a short time, but, after an interval, recurs temporarily, and then perhaps continues to recur and remit alternately. There is not necessarily any abdominal tenderness, and indeed the patient frequently finds relief, as in colic, from pressure on the abdominal parietes. Sympathetic vomiting may be an early symptom, but in the beginning is often absent. Constipation generally follows upon the sudden attack of pain. Sometimes, on the other hand, there is actual diarrhœa, and generally more or less abundant discharge of blood furnished by the congested bowel. The symptoms which mark the subsequent progress of the case depend partly on the situation of the intussusception, partly on the degree of strangulation.

In ileo-cæcal invagination strangulation is rare, and the case tends to be protracted. In this event the symptoms are apt to be ill-defined; the paroxysms of pain are often slight, and recur at distant intervals; constipation may exist at the beginning only, or may recur from time to time, or may never be distinctly present; and there is generally more or less vomiting. As the case progresses, however, the pain often increases in severity; the vomiting becomes more or less incessant, and probably stercoraceous; the alvine evacuations either continue to pass or become re-established; blood and mucus are discharged in variable quantities; and even dysenteric diarrhœa may come on. And then, after a longer or shorter period, sometimes two, three, or four months, the patient, who has been gradually getting emaciated and feeble, dies of simple exhaustion.

When the small intestine is the seat of disease, strangulation usually takes place at once, and its occurrence adds the symptoms of enteritis to those of mere invagination. The case, therefore, speedily assumes a very threatening aspect. Febrile symptoms manifest themselves, the abdomen becomes tender, incessant vomiting comes on, the bowel gets occluded, but at the same time probably blood in some abundance is discharged per anum. With such symptoms the patient, as in simple enteritis or internal strangulation, may speedily succumb; but sometimes, at a moment when the disease appears still to be progressing unfavourably, the constipated bowel begins to act, offensive stools, mixed with blood and mucus, begin to be discharged, vomiting diminishes or ceases, febrile phenomena abate, and, after a longer or shorter time of dysenteric symptoms, a sequestrum is voided in the form of a dark fetid gangrenous mass.

A further indication of the presence of intussusception is the discovery of a tumour. No doubt this cannot always be detected; but it is most likely to be found in cases of ileo-cæcal or colic invagination. That the tumour is due to intussusception may be gathered: partly from its position; partly from its form; partly from the fact of its gradual enlargement and change of position; but above all from its hardening and enlarging and then subsiding under the influence of peristaltic movements. If the tumour descend into the rectum or protrude externally, its nature may of course be readily recognised. The distinctions which have been drawn between invaginations of the small intestine and those of the large—to wit that in the former case the symptoms are usually more sudden and severe, vomiting earlier and more persistent, constipation more complete, discharge of blood per anum more profuse, inflammation more intense, and death more rapid—are no doubt true of most cases, but they are not to be relied upon absolutely; for it occasionally happens that invaginations of the small intestine assume a chronic character, and still more frequently that those of the large take an acute course and even end in the detachment of the invaginated portion. The percentage mortality of

intussusception is very large. It must be observed, however, that in arriving at this conclusion we necessarily exclude all those cases in which intussusception is found accidentally after death from other diseases, and those (which we believe to occur now and then) in which intussusceptions form during life and disappear again after the temporary production of symptoms of more or less severity. The average duration of cases fatal from enteritis appears to be about five days.

H. *Concluding Remarks in reference to Symptoms.*

Before dismissing the subject of intestinal obstructions it may be convenient to consider some of the more important points upon which our discrimination of cases that come before us must depend.

1. *Pain* is a more or less general and prominent symptom in all cases of obstruction. It is sometimes due to peritonitis, sometimes to colic, sometimes to both of these causes. It varies in intensity in different cases, and may be almost entirely absent. Pain of peritonitic quality attends those cases of obstruction which are accompanied by enteritis, and is apt to subside as tympanites supervenes and the fatal event approaches. Colicky pains constitute one of the most characteristic and at the same time one of the most distressing symptoms of intestinal obstruction. They come on in paroxysms, and are attended with more or less violent peristaltic movements of the bowel above the seat of obstruction, which are often distinctly visible through the abdominal parietes, and may even from their course and point of apparent cessation furnish a clue to the seat of impediment. These pains may be present in a marked degree in all forms of obstruction, but are most severe and most constant in the cases of longest duration—in those, therefore, in which enteritis is either not present at all or comes on late.

2. *Vomiting* is rarely if ever entirely absent. At first it is merely sympathetic. But after a while it is due to mechanical causes. The bowels above the seat of the obstruction get distended by their contents, which are partly the ingesta, partly the secretions of the mucous surface. These, by the combined effects of simple overflow, peristaltic action, and pressure from without, regurgitate into the stomach, and then become voided, constituting what is called stercoraceous vomit. This may be peasoup-like and fetid from decomposition, but is never derived from the large intestine or truly fæcal. Vomiting is generally an early symptom in all cases of intestinal obstruction, and in those of acute progress may continue to the end without cessation. Yet even in some of these it intermits, and may be absent for a comparatively long period. In more chronic cases its occurrence is extremely variable, but even here it generally becomes more or less constant and stercoraceous towards the close of life. Vomiting is an earlier, more constant, and more severe symptom, in proportion to the nearness of the seat of obstruction to the stomach. In obstruction of the large

intestine it is usually long delayed, and may never be a prominent symptom.

3. *Constipation* is of course one of the most characteristic phenomena of obstruction; yet fæcal matters will often pass with little difficulty through even a tight stricture, especially of the small intestine. Nor must it be forgotten that generally at the time at which complete obstruction is established, the bowel below contains larger or smaller quantities of fæces, which may be removed naturally or by injections. Scybala are sometimes found post mortem in the large intestine below a complete obstruction of many weeks' standing. Nevertheless, insuperable constipation coming on suddenly is a striking feature of internal strangulation and of the lodgment of gall-stones; insuperable constipation coming on gradually or with premonitory stages, of stricture and compression. In intussusception also there is generally sudden constipation of varying duration; but the invaginated mass, especially when the large intestine is involved, is rarely quite impervious, so that before long, at all events in chronic cases, the transmission of fæcal matters is resumed. In intussusception, moreover, blood is apt to be passed at an early period by stool; and is generally passed in abundance when the small intestine is the part affected.

4. *Tumour and shape of belly.*—The belly usually becomes before long more or less tense and tympanitic in consequence of the accumulation of gas in the parts above the seat of obstruction; and the form of the stomach or of certain convolutions of the bowels may sometimes be distinctly mapped out. Careful attention to the form of the belly, to the visible movements of the organs beneath, and to the sounds elicited by percussion will often aid us in determining the seat of disease. Still too much reliance must not be placed upon these phenomena, for certain lengths of bowel become in some cases so enormously distended that they not only conceal all the other viscera, but a coil of small intestine may equal in diameter a distended colon, and either of them may simulate the stomach. The detection of a lump is an indication of capital importance. It may be due to the presence of a gall-stone or some other concreted mass lodged in the bowel; it may (in cases of stricture) be a mass of malignant disease; it may be the evidence of intussusception.

5. *The condition of the urine* is a matter of interest. In some cases of obstruction there is almost total suppression; in some there is an abundant limpid discharge. Dr. Barlow, who first observed this difference, attributed scantiness of urine to the obstruction being high up in the bowel, and to the consequently little available surface left for absorption; plentifulness of urine to the opposite conditions. Dr. Brinton, accepting Dr. Barlow's facts, referred the deficiency of urine to the abundant vomiting which attends the one class of cases, and the copious secretion of that fluid to the comparative absence of vomiting which is usual in the other class. Mr. W. Sedgwick, however,

argues that the diminution or suppression of the urinary secretion is related to the suddenness and intensity of the symptoms, and is due to the influence of the sympathetic system. On the whole, there is reason to believe that the diminished secretion, which is often only temporary, characterises mainly those cases in which the symptoms are sudden and acute; and almost necessarily, therefore, in larger proportion, cases involving the small intestine than cases involving the large.

6. *Duration of life.*—Complete obstruction occurring in the rectum or colon may not prove fatal for several weeks or even several months. Death as a rule supervenes earlier in proportion as the impediment is situated near the stomach. When, however, enteritis is associated with obstruction, then, wherever the obstruction may be, the progress of the case is always rapid, and, dating from the commencement of enteritic symptoms, rarely occupies more than a week, often only three or four days.

7. *Statistics.*—According to Dr. Brinton's figures, based on 500 deaths from obstruction, it appears that in every 100 cases, 43 are due to intussusception, 17 to stricture, 4·8 to impaction of gall-stones, 27·2 to internal strangulation (including, however, all those cases which have been ascribed above to compression or traction), and 8 to torsion or twisting.

I. *Treatment.*

1. The treatment of constipation turns, in different cases, upon very different considerations. A temporary attack may be put right by the use of a simple purge—a dose of castor-oil, a black draught, a colocynth pill, or a simple enema. In young babies the mere introduction of the nozzle of the enema tube, or the insertion into the anus of the point of a piece of soft paper rolled into the form of a pencil, is often amply efficacious. When constipation is of a more permanent character it may often be overcome by the mere persistent repetition of daily efforts at some particular time (preferably after breakfast) to evacuate the rectum; or by the habitual use of particular kinds of food, such as brown or bran bread, of a large proportion of fresh vegetables or fruit, or of dried fruit, such as plums and figs; or, again, by the daily use of small doses of mild laxatives, such as a few stewed prunes, a teaspoonful of confection of senna or of castor-oil, taken in the morning, on an empty stomach; or by the similar employment of a combination recommended by Trousseau of one-sixth or one-seventh of a grain of podophyllin combined with an equal quantity of extract of belladonna. In some of these cases, a course of iron, strychnia, belladonna or atropia in small doses, either alone, or combined with mild aloetic or other purgatives which act especially on the lower bowel, is serviceable; as also is the occasional employment of simple or purgative enemata. Galvanism applied to the surface of the abdomen, or

to that and to the anus, is occasionally efficacious. In some cases much more active purgation is needed; and in some it becomes absolutely necessary to dislodge the hard fæcal accumulation by the finger, spoon, or some such instrument, or by the use of repeated enemata, or, better still, by directing a forcible stream of warm water, conducted from a height by means of a tube, into the rectum, and allowing it to play upon the fæcal mass for half an hour or so at a time, and thus to cause its disintegration, and either effect or facilitate its removal.

2. The treatment of cases of obstructed bowel must be regulated partly in accordance with what we know or suspect of the nature of the cause of obstruction, partly in accordance with the character of the symptoms present. It may be laid down as a general rule, from which it is highly unsafe to depart, that, whenever the symptoms of obstruction are associated with those of enteritis—whenever, in fact, there is besides obstruction obvious inflammation—the treatment to be adopted is that already recommended for cases of enteritis:—namely, the local abstraction of blood, and application of fomentations, the use of opium in sufficiently large quantities, the avoidance of purgatives, the administration of food in small portions and in the liquid form, and all those subsidiary measures which are elsewhere considered in sufficient detail. This is the form of treatment that is especially applicable to cases of internal strangulation, impaction of foreign bodies, and jejunal or ileal intussusception.

In those cases, however, in which the symptoms of obstruction come on vaguely and without evidence of association with inflammatory mischief, it is generally advisable to commence the treatment with the administration, either by the mouth or by the rectum, of moderately powerful purgatives, and to persist in this treatment until, by their failure to act, and by their causing vomiting, and painful but fruitless peristaltic movements, their inefficacy is distinctly shown. It sometimes happens that, after drastic purgatives have failed, a large dose of some simple laxative, such as castor-oil, acts with singular efficacy. In aid of this treatment, hot baths, fomentations, ice or electricity to the surface of the belly, and voluminous enemata of gruel or water, administered by the long tube, may severally be employed. If these measures are without avail, it is generally advisable to give the bowels rest, and to relieve pain by the repeated use of adequate doses of opium or belladonna; the persistence in which treatment will, by relieving spasm or otherwise promoting the return of some length of bowel to a comparatively healthy condition, not unfrequently result, after a shorter or longer time, in an effectual and sufficient evacuation. If this treatment fail in its turn, it may be necessary again to solicit the action of the bowels by the employment of purgative medicines, enemata, and the like. Such is the routine that must generally be followed in cases of simple obstruction, in which the cause of obstruc-

tion is obscure; and in many cases also even when the cause is distinctly ascertained.

When, however, the obstruction depends on the presence of a stricture in the rectum or sigmoid flexure, the persistent use of powerful purgatives is scarcely judicious; copious and frequently repeated enemata are then of especial value. If the stricture be within reach it may admit of dilatation by the bougie. Again, when the obstruction is due to an ileo-cæcal or a colic intussusception, powerful purgation is likely to do more harm than good; large enemata, however, are occasionally efficacious in causing the reduction of the intussuscepted bowel. But the most powerful, and apparently the most efficacious, form of enema for this purpose appears to be the inflation of the large intestine with air. Lastly, when, in cases of obstruction, the bowel is working with visible violence and pain; or we have reason to believe that (as in the course of those cases in which the invaginated portion of bowel is discharged per anum, and in all cases where there is, and has been for some time, much tympanites) the bowel is enfeebled and in danger of rupture, purgatives must be religiously avoided.

In the course of many cases of obstruction, the question of relieving the patient by surgical means must necessarily arise. The time at which an operation should be performed, and the nature of the operation to be performed, are of course matters of grave importance. It may be accepted as a general rule that when once the desirability of this procedure has been recognised, the earlier recourse is had to it the better. Exploratory operations with the object of discovering the nature of the impediment, and then, if possible, of relieving it, are rarely successful. There are, however, some circumstances under which operations are not only justifiable, but imperatively demanded. No patient who has either a rupture (even if there be no evidence of strangulation in it) or a hernial sac, or any trace or hint of any affection of the sort, should be permitted to die with symptoms of obstruction without having the chance afforded him which an exploratory operation at the suspected site affords; nor should we, with the object of unfolding the involved bowel, hesitate to perform gastrotomy in cases of ileo-cæcal intussusception which have resisted other modes of treatment. Other operations which are often serviceable in prolonging life, and are sometimes curative, consist in opening the bowel and forming an artificial anus in some convenient spot above the seat of obstruction. Such operations are especially applicable when the large intestine is the seat of disease. If the obstruction be in the rectum or sigmoid flexure, the opening should be made in the left loin into the descending colon; if above these portions of the bowel, then in the right loin and into either the cæcum or the ascending colon. If the obstruction be in the cæcum itself, or in the small intestine, Littré's operation is alone available. This consists in laying open the perito-

neal cavity, and then opening the bowel (having first brought the part to be divided to the surface) above the seat of stricture. The lips of the wound in the intestine must be attached by sutures to those of the incision in the abdominal walls. The enormous gaseous distension of the bowel which often takes place in obstruction is a cause of great discomfort, and even adds to the patient's risks. It may be relieved by puncturing the distended gut through the abdominal parietes with a grooved needle or fine trocar and cannula. The operation is attended with little or no danger.

XVI. ASCITES. (*Abdominal Dropsy.*)

Causation and morbid anatomy.—The above terms are applied to the accumulation of serum within the peritoneal cavity. Ascites is an accompaniment or sequela of many different diseases; but depends immediately on some condition which modifies the action of the capillary vessels or lymphatics of the peritoneal membrane. This condition may be either some morbid process going on in the peritoneal tissue; some impediment to the flow of blood through the portal vessels; or some disease influencing the systemic circulation generally.

1. More or less effusion of serum attends ordinary cases of acute peritonitis. But such accumulations are rarely abundant, and generally soon disappear. Chronic peritonitis, on the other hand, is a common cause of persistent and progressive ascites, and especially perhaps those forms of chronic peritonitis which occur in women in connection with inflammatory conditions of the pelvic organs and the growth of ovarian cysts. In some of the latter cases the dropsy is due to the rupture of cysts and the discharge of their contents into the peritoneal cavity. Tuberculosis and malignant disease of the peritoneum are other frequent causes of ascites.

2. Impediment to the flow of blood through the portal system, and consequent ascites, may be referrible to direct compression of the portal trunk by cancerous, aneurysmal, or hydatid tumours arising external to the liver, or by tumours of various kinds originating in its substance, and especially by cancerous or fibroid growths occupying the lesser omentum and extending thence into the liver along the capsule of Glisson. Most commonly, however, they are caused by general diseases of the liver, involving the hepatic capillaries and the minute veins which open into and emerge from them. Of these cirrhosis is the most frequent and important. But the simple induration and congestion which constitute the 'nutmeg liver' may have the same effect, as also possibly may lardaceous degeneration. The compression of the liver by a fibroid capsule of inflammatory origin may act in the same way as cirrhosis.

3. Among the diseases by which dropsical effusion into the belly, as a part of general dropsy, may be caused, are heart-disease, chronic affections of the lungs, and certain forms of renal disease; to which may probably be added various cachexiæ and simple anæmia. In many of these cases the ascitic accumulation is proportionate only to the dropsy of other parts. In some cases, however, it becomes excessive, while the dropsy elsewhere undergoes but little increase. When this is the case there is generally some local complication (coming under the first or second group of causes which have been considered) to which this disproportion is attributable.

The amount of fluid present in ascites may vary between a few pints and four or five gallons. Its quality also may vary. It is usually slightly viscid, transparent, of a yellowish or greenish tinge, alkaline, and containing both albumen and fibrinogen, and often fibrinous clots. It is sometimes very viscid (especially in cases of ovarian tumours or colloid cancer), sometimes opaline from the presence of inflammatory or other products, or turbid and discoloured with blood in a more or less altered form.

Symptoms and progress.—The accumulation of fluid in the abdominal cavity causes its gradual distension, and sooner or later obstructs the intra-abdominal veins, especially those connected with the lower extremities, impedes the movements of the diaphragm, and interferes more or less injuriously with the healthy action of the abdominal viscera. It modifies also the patient's gait, making him walk like a pregnant woman, with his legs wide apart and his head and shoulders thrown back. The ascitic abdomen is large, uniformly rounded, with a tendency to spread or bulge in the flanks as the patient lies on his back, tense, and more or less smooth and shining, and often presents distended superficial veins and that linear atrophy of the skin so common in pregnancy. The stomach and intestines tend of course to float on the surface of the fluid; and hence generally the highest part of the abdomen is resonant, the more dependent parts dull—the line of demarcation between them being for the most part well defined and horizontal, but varying with the varying positions which the patient assumes. The liver, which is of higher specific gravity than dropsical fluid, often retreats distinctly from the anterior surface of the abdomen and from the diaphragm, a stratum of fluid with sometimes a loop of bowel occupying the interval. The presence of fluid is further indicated by the peculiar thrill which is experienced by the hand laid flat on the abdomen when a ripple or wave is produced in the ascitic fluid by a slight tap or fillip applied to some other part of the abdominal surface.

These signs are not always all present, or at least easy to recognise; and not unfrequently tumours and other conditions simulate or mask abdominal dropsy, and interfere with the formation of an accurate diagnosis. The fluid may be so small in quantity that it occupies the

pelvis only; it may then, however, often be detected by making the patient rest on his knees and elbows so as to allow it to gravitate to the neighbourhood of the umbilicus. It may be so abundant that the stomach and bowels fail to reach the surface; in which case the dulness may in all positions of the body continue general, excepting, perhaps, in the course of the ascending and descending colon; but here fluctuation will almost certainly be well marked. Or adhesions may limit the distribution and mobility of the ascitic fluid; or there may be adventitious growths in the abdominal cavity; or the parietes may be fat or œdematous.

In most cases ascites causes pretty uniform distension; but in some, where pouches exist, or the parietes are specially thin and yielding, this uniformity becomes interfered with. Thus hernial sacs, whether at the umbilicus or at the groin, get distended with fluid; and in females the recto-vaginal pouch sometimes become so much dilated that it protrudes through the vulva in the form of a tumour, carrying with it as a covering the posterior wall of the vagina.

Œdema of the lower extremities and genitals is a common and early accompaniment of ascites. It sometimes occurs so early that the patient observes it before his attention is particularly directed to the condition of his belly. It is doubtless due (when thus limited and unconnected with cardiac or other equivalent disease) to the pressure exerted by the ascitic fluid on the iliac veins, and is generally fairly equal in the two limbs. Shortness of breath is an early symptom. It depends on the mechanical impediment which the accumulated fluid opposes to the descent of the diaphragm, and increases, therefore, with the increase of accumulation. It is sometimes so slight that the patient only observes it when he exerts himself; sometimes it is exceedingly distressing; and it is usually increased when he lies down. The lower parts of the lungs are apt to become empty of air and collapsed. More or less abdominal discomfort or pain, mainly in the lumbar regions and about the umbilicus, generally arises in the course of the affection. This is often of an aching, flatulent, or colicky character, and is probably due in some degree to the pressure which the fluid exerts on the hollow viscera and other organs. Sometimes it is peritonitic, and indeed the supervention of acute or subacute peritonitis is not rare in the later stages of ascites. Although early in the affection there may be no visible morbid condition of tongue, and neither thirst nor loss of appetite, the tongue and the digestive functions become after a while variously and more or less seriously affected. Diarrhœa especially is a by no means uncommon complication; and is due, sometimes to the same impediment to the portal circulation as causes the ascites, sometimes to slight dysenteric disease. There is also generally some dryness of skin and diminution of the urinary secretion. Other symptoms more or less grave are usually presented by ascitic patients; but they are for the most part the symptoms of the morbid conditions

on which the ascites itself depends, and are sufficiently considered elsewhere.

Treatment.—The treatment of ascites merges, in a large proportion of cases, in the treatment of the diseases out of which it arises. Still there are many cases in which sooner or later special treatment directed against the ascites itself is demanded. To promote the absorption and removal of the dropsical accumulation there are good theoretical reasons for the employment of those remedial measures which increase the discharges from the skin, the kidneys, and the bowels. For diaphoretic purposes we must not forget the value of the hot bath, the vapour bath, and the Turkish bath. Among diuretics must be especially signalised iodide of potassium, copaiba, and the combination of crude mercury, fresh squills, and digitalis. Of purgatives, those which promote watery evacuations are obviously the most likely to prove efficacious. We are bound, however, to state that, while acquiescing in the importance of restoring, so far as may be, or of maintaining, the healthy action of the skin and kidneys, and of acting freely on the bowels, we have never been satisfied of the efficacy of such measures in causing the removal of the dropsical fluid. And indeed, as regards purgatives, we have frequently had to discard them because, while not distinctly benefiting the dropsy, they were in other ways obviously affecting the patient's health injuriously. Further, the diarrhœa which comes on spontaneously in the course of ascites is not only not curative, but is difficult to arrest, and very often of bad augury. In a large number of cases, no doubt, all medicines are alike inefficacious; but there are many in which the general improvement of the patient's health, no matter how brought about, is followed by the subsidence of the dropsy. Tonics, of which quinine, iron, and cod-liver oil are probably the best, are especially valuable in this respect. It is certain that they are often well borne by ascitic patients, and that, even when not well borne at first, a little judiciousness in their selection and mode of exhibition renders them tolerable; and it is certain that often, under their use, patients not only improve in general health, but lose in part or wholly their dropsical accumulations, and that occasionally their recovery is permanent, and permanent even after the performance of paracentesis. Local applications to the abdomen are only needed to relieve pain or uneasiness; but when the abdominal distension becomes so great as to cause the patient serious suffering or distress, the fluid must be removed by tapping. This operation is usually delayed as long as possible, and on the whole no doubt properly so. There is nevertheless reason to believe that the beneficial effect of remedies is sometimes exerted much more markedly immediately after it than while the abdomen is full of fluid. Paracentesis, though usually a harmless operation, is sometimes followed by peritonitis. It is rarely of even temporary benefit in the ascites which accompanies malignant disease.

XVII. HEMORRHAGE. HÆMATEMESIS. MELÆNA.

Definition.—When blood is vomited the affection is termed '*hæmatemesis*'; when blood is passed by stool and is of a black colour, as it usually then is, the term '*melæna*' is applied.

Causation.—Gastro-intestinal hemorrhage may be due: either to the influence of diseases, such as the infectious and malarious fevers, purpura, and scurvy, in which the quality of the blood is altered; or to mechanical impediments to the passage of blood through the portal system or any of its tributary branches; or to congestion, inflammation, breach of surface, or morbid growths, involving any part of the mucous membrane. It occasionally also occurs vicariously of menstruation.

Profuse hemorrhages arise mainly, from chronic ulcers of the stomach or duodenum, or from general hyperæmia of the gastro-intestinal mucous membrane, due either to cirrhosis of the liver or to obstruction of the portal trunk. Besides these causes must be especially named carcinomatous and villous growths of the stomach and bowels, and the rupture of aneurysms. Copious hemorrhage sometimes also takes place from typhoid or dysenteric ulcers. But it must not be forgotten that hemorrhage, which must be regarded clinically as hæmatemesis, often comes from the œsophagus, and may then be due to malignant disease causing perforation of the œsophageal, intercostal or other neighbouring vessels, or to the rupture of aortic or other aneurysms or of dilated veins; and, further, that blood vomited from the stomach may have been previously swallowed, as sometimes happens accidentally in epistaxis, or designedly and for the purpose of deception.

Symptoms and progress.—It may be taken as a rule to which there are few exceptions that blood, discharged from any part of the alimentary canal below the duodenum, is voided solely by the anus with the fæces. And although hemorrhage from the stomach, duodenum, or œsophagus is no doubt, in a large number of cases, attended with more or less obvious hæmatemesis, it must not be forgotten that in almost all cases a larger or smaller quantity of the blood which finds its way into the stomach is passed onwards into the bowels, and that in some the whole bulk of it is thus transmitted. These are facts of great importance, inasmuch as abundant gastro-intestinal hemorrhage may take place, and may continue for some length of time, causing progressive and extreme anæmia, without revealing its presence to the patient himself or to the medical man who fails to investigate the condition of the stools.

The recognition of blood in the vomit or fæces is not generally difficult. If it escape in small quantity, into either the stomach or the

intestine, it becomes mingled with the other contents of the viscus, which acquire a grumous, coffee-ground, sooty, or pitchy character. And, however abundant it may be, the longer its detention in the alimentary canal, or the longer the journey which it has to perform along it, the darker and blacker as a rule it appears at the time of its discharge. Under other circumstances it may be voided almost pure, sometimes fluid, sometimes coagulated, and, though generally of a dark hue, in some instances of a vivid arterial tint. If there be a doubt as to the presence of blood, the microscope will probably clear it up. When, however, the blood-corpuscles are wholly destroyed, and blood-pigment alone is left, some difficulty of identification may be experienced, and it may be necessary to have recourse to chemical investigation or to the spectroscope. It is of course important not to confound the discolouration of the vomit and fæces, due to articles of diet (port wine and the like), to drugs (iron, bismuth, and mercury), or to bile, with that dependent on the presence of blood.

Small hemorrhages are in themselves of little moment; their frequent repetition, however, necessarily tends to induce more or less marked anæmia, and the various symptoms which attend anæmia. Large hemorrhages, on the other hand, are alarming in their immediate symptoms, and of extreme danger to life.

The occurrence of copious bleeding into the gastro-intestinal canal is usually attended with sudden faintness; sometimes indeed the patient falls down insensible and convulsed. The phenomena are those of the rapid abstraction of a large quantity of blood, but are also not unlike such as may attend the sudden effusion of blood into the substance of the brain, or the commencement of an epileptic seizure. From this attack of faintness, the patient usually soon recovers somewhat; and then, if the hemorrhage have taken place into the stomach, he probably ere long vomits a more or less considerable quantity of blood—sometimes as much as a quart or two at one time or within a short period—and later on passes a greater or less bulk of pitchy matter by stool. In some cases, as has been pointed out, no vomiting of blood takes place, but melæna alone supervenes. The recognition of the initial symptoms as due to gastro-intestinal hemorrhage rests on the supervention of hæmatemis, or melæna, or both.

The further progress of such cases depends largely upon their cause. In some instances the patient dies from sudden faintness, or falls into a condition of collapse from which he never recovers. In some he is suffocated by the entrance of blood into the air-passages. In some the hemorrhage is repeated over and over again, the patient becomes excessively anæmic, the usual symptoms due to recurrent losses of blood ensue, and at length death occurs. In some cases of course he makes a good recovery, and possibly never has a return of his malady. A curious occasional phenomenon, in connection with profuse hæmatemesis, to which Lenke calls special attention, and which we have

witnessed, is the sudden occurrence of double amaurosis; in which the ophthalmoscope reveals only unnatural whiteness of the optic nerve and diminution of the retinal arteries, and which appears to be for the most part incurable.

Treatment.—An accurate diagnosis of the cause and seat of bleeding in hæmatemesis and melæna is very important in reference to treatment, and must rest partly on close observation of the phenomena which the case presents, partly on a careful enquiry into its history, but is in many cases, at least for a time, absolutely impossible. There are certain measures, however, which under any circumstances should be taken. The patient should be placed and kept in the supine position, forbidden to make muscular exertion, and guarded from all causes of mental excitement; the stomach and bowels should be kept as far as possible at rest, and hence generally purgatives and emetics, solid food and stimulants, should be eschewed, and fluid food should be given in small quantities; and the force of the circulation should be restrained, a result which is in some degree attainable by perfect quiescence of mind and body, by keeping the outer surface only moderately warm, and by the use of certain medicines, of which digitalis and lead are among the most valuable. Simple styptics are not generally of much use in restraining hemorrhage, unless they can be directly and well applied to the bleeding surface; and hence their value is not generally very great in restraining gastro-intestinal hemorrhage. It can, however, at least do no harm to employ them. Among such remedies may be named perchloride of iron, sulphuric acid, tannic acid, and turpentine. Ice and ice-cold drinks are serviceable, as well for their astringent as for their sedative influence. Ice may also be applied with benefit to the surface of the chest or abdomen. When we have reason to believe that the hemorrhage is due to an overloaded state of the portal system, it is commonly regarded as injudicious, if not useless, to attempt to restrain it. And indeed it is often recommended that the bowels should then be acted on by repeated and tolerably strong purgatives, with the object of relieving the distended vessels. It must be remarked, however, that this variety of gastro-intestinal hemorrhage is probably the most frequently fatal of all varieties; and that death, when it occurs, is generally due simply to loss of blood. It seems scarcely reasonable, therefore, in such cases to promote, by stimulating the bowels, a kind of relief which is so dangerous to the patient's life, and which, even without such stimulation, is only too often fatal. The blood, indeed, which comes away is probably derived mainly, if not entirely, not from the overloaded portal system, but from the systemic arteries which feed that system, and directly from the congested capillaries distributed to the mucous surface. We should therefore recommend the employment not only of cold and of astringents to the alimentary tract, but of all those measures which have been noticed as tending to soothe and regulate the circulation. The further treatment

of gastro-intestinal hemorrhage must depend on the nature of the primary disease from which the patient is suffering, and of the special features which his case from time to time exhibits.

XVIII. DYSPEPSIA. (*Indigestion.*)

Definition.—No account of the diseases of the alimentary canal and its appendages would be deemed complete unless it comprised some separate consideration of dyspepsia or indigestion, that most common and fashionable of all complaints. It is difficult, however, to know how to deal with it; for, on the one hand, it includes within itself all those functional derangements of the stomach which attend and help to characterise the various diseases of that viscus, and many of those of the rest of the alimentary canal, and of the glandular organs opening upon its mucous surface, together with such derangements as are connected with general morbid states of the system, and such as depend upon the quality, quantity, and condition of the alimentary matters taken into the stomach; while, on the other hand, it is often regarded as the collective name for groups of morbid symptoms, referrible to the stomach, which are independent of any discoverable local or constitutional disease. In the former point of view, dyspepsia ranges throughout the whole domain of clinical pathology; in the latter, the advance of pathological knowledge tends day by day to restrict more and more the limits of its applicability. To discuss dyspepsia in the former sense would be utterly beyond the scope and purport of the present work: to consider it strictly in the latter sense would be at once difficult and unsatisfactory. The most convenient course will probably be to consider briefly: the causes of dyspeptic symptoms; the several local phenomena which constitute dyspepsia; the sympathetic and other consequences to which dyspepsia may give rise; and, lastly, its medical treatment.

Causation.—The causes of dyspepsia may be conveniently divided into three groups:—namely, those connected with the ingestion of food; those connected with morbid conditions of the stomach; and those connected with derangements or diseases of other organs or of the general system. 1. In the first group are comprised many pregnant causes of indigestion—causes, some of them, none the less important because they involve the habitual and conscious transgression of obvious sanitary laws. Among them may be included the following:—*Imperfect mastication*, or the bolting of food, usually arising from undue haste in eating, or from defect or absence of teeth, or from soreness or paralytic conditions of the mouth; *Active bodily or mental exertion* immediately before or after a meal; *Over-eating*, whether this consist in a

single surfeit, or in that habitual indulgence to excess of which so many of us are guilty, and which is especially injurious if it go along with sluggish sedentary habits; *Insufficiency of food*; *Improper arrangement of meals*, such, for example, as the taking of one meal only during the twenty-four hours, or the crowding of all one's meals within a period of eight or ten hours, leaving the remainder of the four-and-twenty without any, or the practice (included to some extent under the last head) of interpolating meals between the more important meals, and thus refilling the stomach ere it has had time to rid itself of its previous load; *Injudicious admixture of foods*—of the frequently injurious influence of the admixture of many different kinds of even wholesome articles of diet there can be no doubt; it is difficult, however, to lay down any exact rule in regard to this matter, for, within limits of moderation, variety is conducive to health, and the too strict limitation to one or two kinds of food not unfrequently proves as detrimental as excessive heterogeneous indulgence; The *use of indigestible or unwholesome aliments*—this might serve as the text for a very wide discussion; it is sufficient, however, to point out here that, in addition to substances which may be regarded as generally more or less injurious, there are many which become injurious only from the circumstances or conditions under which they are taken, or from temporary or permanent peculiarities in the constitution of the sufferer, or in the condition of his digestive organs; thus sometimes mutton, pork, veal, game, or shellfish disagrees, sometimes pastry, milk, or eggs, sometimes different forms of vegetables or fruit, sometimes tea or coffee. To these causes may be added the abuse of alcoholic stimulants, or of tobacco, and the excessive indulgence in condiments, and perhaps also the habitual abstention from certain kinds of food which are essential to the due maintenance of the integrity of the organism. 2. The second group of causes—that which embraces the morbid conditions of the stomach itself—is necessarily also a very extensive group. It includes, moreover, all those morbid conditions which have already been described, and the presence of any one of which removes the case from among the dyspepsiæ in the restricted sense of that term. The following is a list of the more obvious of the conditions here adverted to:—*Catarrhal inflammation and congestion* of the mucous membrane, which are amongst the most persistent causes of dyspeptic symptoms, and are often the immediate cause of such symptoms attending the various alimentary abnormalities just enumerated; *Gastric ulcer*; *Carcinomatous and other morbid growths*; *Abnormal dilatation of the stomach*, whether this be of primary origin, depending upon inherent feebleness of the walls or habitual overloading of the organ, or whether it be secondary to pyloric or other obstructions to the onward passage of alimentary matters; *Diminution in size*, whether arising from the gradual contraction of infiltrating growths of the gastric walls, or from long-continued abstinence, or from spasmodic action of the muscular coat referrible to irritability of

the mucous surface or other sources of reflex action; *Degenerative changes* of the mucous membrane, such as may result from chronic catarrhal inflammation, or the abuse of alcohol, or may arise in the course of chronic wasting diseases; And, lastly, functional derangements, including irritability, and excess, diminution, or derangement of the gastric secretions. 3. The third group of causes again is one of very great extent. It includes all those conditions of the alimentary canal—constipation and the like—which react on the functions of the stomach; all those morbid states of surrounding organs which lead to pressure on the stomach and interference with the performance of its duties; all those lesions of the portal system, lungs, heart, and kidneys, which, by impeding the circulation, induce congestion or other abnormal conditions of the stomach; all those disturbances of the nervous system (among others powerful mental impressions or emotions, and the reflex phenomena of early pregnancy) which influence the actions of the stomach; and, further, all those general diseases —anæmia, pulmonary phthisis, fevers, and innumerable others—of which difficult, painful, or faulty digestion forms an appreciable, if not a prominent, symptomatic feature.

Symptoms. 1. *Referrible to the stomach.*—The symptoms which attend and indicate dyspepsia are to a large extent those which also accompany in a greater or less degree the various organic lesions of the stomach. They comprise derangements of appetite, derangements of sensation, flatulence and eructation, nausea and vomiting.

The *appetite* in dyspeptic patients is very variable. In some cases it remains but little affected, or there is simply a distaste for certain articles of diet; or without there being any actual distaste, experience shows that certain alimentary matters formerly taken with impunity now induce various discomforts. In many cases there is more or less loss of appetite, and occasionally this amounts to absolute repugnance to all forms and varieties of food. In many cases, again, a persistent sense of uneasiness or emptiness, with constant craving for food, is a marked phenomenon; it occasionally happens that the appetite is absolutely increased; more frequently, however, the craving is changed by the ingestion of even small quantities of food into some other sensation of discomfort, which brings the meal to a speedy close. Now and then, and especially in hysterical females, the appetite becomes depraved—the patient not merely craving for aliments which are of an unwholesome character, but swallowing earth, coals, chalk, or other substances which are either wholly devoid of alimentary virtues, or disgusting, or absolutely injurious. Thirst may or may not be present.

The *abnormal sensations* which attend dyspepsia are of different kinds. There is generally more or less uneasiness or pain. A sense of weight, sinking, fulness, shooting, aching or burning, referred to the pit of the stomach or some neighbouring part, or to the inter-scapular

region, is rarely absent. In some cases this comes on mainly when the stomach is empty, and disappears under the influence of a meal; in some it comes on wholly after food, and lasts during the whole period of gastric digestion; in some it is more or less constant, being present when the stomach is empty, and getting aggravated or modified after a meal. In other cases pain comes on some little time after food has been received into the stomach, it may be in the course of a quarter or half an hour, or after the lapse of two, three, or four hours. Another form of gastric pain is described as connected with dyspepsia, namely, a pain of great intensity, frequently likened to that of cramp, which comes on at irregular and often rare intervals, which lasts a variable time, and is usually attended with marked symptoms of faintness or collapse, and often in women with hysterical phenomena. This pain, which is not uncommon in gouty persons, occupies the usual position of gastric pains, but shoots in various directions, upwards into the chest and downwards into the abdomen. There is no doubt that this variety of gastrodynia is largely confounded with that due to the passage of gall-stones, and with pains originating in various other than gastric sources. Epigastric tenderness is not usual.

Flatulence and eructation are generally complained of by dyspeptics in a greater or less degree. Flatulence usually goes along with sense of fulness or distension of the stomach, and other of the uneasy or painful feelings which have been considered. The accumulation of gas is indicated also by actual distension of the epigastric region, and the occurrence of gurgling and other noises within the stomach; it moreover gives rise to eructation. Eructation, which is generally attended with more or less relief to the patient, is often noisy, and effected with powerful and uncontrollable spasmodic action of the muscles. The amount of wind thus discharged is sometimes enormous: and at the same time it is so sudden in its evolution that it has been assumed to be secreted by the mucous membrane of the stomach and bowels. Of this, however, there is no proof; and indeed there can be no doubt that it is really derived from decomposition of the food. The gases consist of carbonic acid, hydro-carbons, and in some cases of sulphuretted hydrogen. Together with these, small quantities of the contents of the stomach are not unfrequently brought up. In some cases the quantity of matter thus discharged without sensation of sickness or material effort is very considerable; and the process by which it is returned is sometimes termed rumination.

Nausea and sickness, again, are frequent symptoms of dyspepsia, and are sometimes exceedingly distressing. In many cases of functional dyspepsia, as in that of pregnancy, nausea often goes along with increased appetite. Sickness is usually preceded by nausea, and occurs at different times and with various degrees of severity. In some cases it comes on when the stomach is empty; more frequently it occurs shortly after ingestion; sometimes it does not happen until an hour

or two after a meal; and occasionally it takes place at irregular and long intervals. The material vomited presents considerable variety: in some cases it is simply the food but little altered; in others it is an alkaline ropy mucus; in others it consists mainly of the ordinary acid juices of the stomach; in others it is a neutral watery fluid having many of the characters of the salivary secretion. In other instances (and especially when the vomiting does not take place until long after the ingestion of food) the vomited matters have undergone fermentation: they are acid from the development of acetic, lactic, and butyric acids, and present, on standing, a brownish frothy scum and a more or less abundant sediment; or else they have undergone putrefactive changes and have an offensive, occasionally rotten-egg-like, occasionally almost fæcal, odour. The vomit presents as much variety in quantity as in quality; sometimes it is scanty, and little more abundant than occurs in eructation; at other times it is discharged in enormous quantities. The latter occurrence is most frequent when the vomiting comes on some hours after a meal, or at irregular and comparatively long intervals, and therefore in cases of obstructive disease of the pylorus, or when the stomach, from whatever cause, is abnormally dilated and sluggish or enfeebled. It may be observed that vomiting immediately after food is generally indicative of irritability of the stomach; that the discharge of abundant ropy mucus usually implies the presence of inflammation; that fermentative and putrefactive changes point to long retention of alimentary matters in the stomach, and possibly also to some defect of relation between the quantity of food ingested and the quantity of gastric fluid secreted; and that always after long-continued vomiting the contents of the duodenum, inclusive of bile, regurgitate into the stomach, and thus mingle with the vomit. When fermentation takes place, the torula cerevisiæ may always be discovered in great abundance in the vomited matters, and it is usually under similar circumstances that the sarcina ventriculi also may be recognised.

The term *pyrosis* is generally used of those cases in which a clear fluid is vomited or eructated, for the most part in connection with more or less severe epigastric pain, and at times when the stomach is empty, or nearly empty, of food. The quantity of fluid brought up at one time may vary from a few teaspoonfuls to several pints. It is usually neutral, but may be alkaline or acid. Both by Budd and by Frerichs this fluid, when of neutral reaction, is looked upon as being saliva which has been swallowed. Pyrosis is not unfrequently connected with organic disease of the stomach; but in its most typical form is either functional or due to the constant use of certain irritating articles of diet. It is said to be especially common among the lower classes in Scotland and Lapland, and to be dependent on the quality of their food.

2. *Referrible to other organs.*—Among the many secondary pheno-

mena of dyspepsia, those connected with the remaining regions of the alimentary canal first claim attention. The tongue varies in character; it is sometimes clean and healthy, sometimes pale and flabby, sometimes more or less thickly coated, and sometimes cracked or fissured. The bowels are for the most part constipated, but there may be persistent diarrhœa, and not unfrequently there is considerable irregularity of action. In some cases of indigestion, attended with looseness of bowels, undigested food in considerable abundance is found in the stools. It is obvious that in these cases the passage of the contents along the bowel is exceedingly rapid; and in many of them, according to Trousseau, whose experience is confirmed by that of Dr. Wilson Fox, there is at the same time large appetite, with rapid escape of food from the stomach, and rapid consequent renewal of appetite. Trousseau assumes that there is excessive irritability of the muscular walls of the stomach and bowels, and that it is on this account that the food is carried too swiftly onwards. The urine is frequently affected, and may contain an excess of phosphates, oxalates, or urates, the last not unfrequently being deposited as a lateritious sediment. The action of the heart is commonly quickened, but is sometimes slower than natural, and often variable. Dyspeptic patients are liable to palpitation and irregularity of action, coming on especially after meals or in the night. Dyspnœa is apt to attend the attacks of palpitation; and a variety of asthma has been referred to the presence of indigestion. Dyspeptic patients are liable in a peculiar degree to certain forms of skin disease, such as urticaria, erythema, lichen, and eczema, but above all, perhaps, to acne rosacea, and other allied conditions manifesting themselves upon the nose and cheeks. Elevation of temperature and other distinct febrile symptoms are no necessary features of dyspepsia; but they may appear if the dyspepsia be connected with inflammatory affections of the stomach. The influence of dyspepsia and of other morbid conditions of the stomach on the functions of the nervous system is very remarkable. Vertigo, headache, intolerance of light or sound, depression of spirits, irritability, hypochondriasis, sleeplessness, and various forms of neuralgia, are all of common occurrence. The severer forms of dyspepsia, and especially those in which there is much sickness, are usually attended with more or less debility and emaciation. Indeed, purely functional affections of the stomach, attended either with total loss of appetite, or with constant vomiting after food, occasionally induce a degree of emaciation and debility rivalling that which one meets with in the last stages of carcinoma of the cardia or pylorus, or of pulmonary phthisis with intestinal ulceration. On the other hand, it is often curious to observe how, notwithstanding incessant vomiting, patients retain a fair amount of plumpness.

Treatment.—The treatment of dyspepsia is a subject of considerable importance and no little difficulty, and demands a good deal of firmness, *savoir faire*, sound judgment and readiness of resource on the

part of the physician, and often at the same time no little trust and resolution on the part of the patient. The first thing to be done is to ascertain as far as may be the circumstances to which the dyspepsia owes its origin, or those which determine its continuance, and, if possible, to cure or obviate them. With this object, it may be of essential importance: to insist on the proper comminution of food, to see that the teeth are in good order, and if not that they are supplemented or replaced by false ones, or that artificial mastication is employed, and that the patient gives ample time to his eating; to regulate the distribution of the meals, so that, if they be full meals, they shall be separated by intervals of four or five hours at least, or if, from any circumstance, the patient is compelled to take only small proportions of food at any one time, the intervals between them shall be correspondingly reduced; to regulate the quantity of food taken at each meal and daily, in the sense of neither letting it fall below what is required, nor of permitting any great excess; to insist that the food taken shall be wholesome and readily digestible, and that especially any article of diet which experience has shown to be injurious shall be strictly abjured. There are considerable differences in regard to the articles of diet which are most suitable for different dyspeptics; and, in order to treat successfully, it is often important to study each patient's peculiarities in this respect. It may be stated generally, however, that all rich and greasy compounds and fat are likely to disagree; that fish, flesh, and fowl (whichever be selected) should be well cooked; that raw vegetables should be eschewed; that in a large number of cases (and especially those in which the stomach is irritable or inflamed) milk and farinaceous foods and eggs are of especial value; that ripe fruits are admissible and often beneficial; and that alcoholic beverages should be only moderately indulged in. In many cases total abstinence from alcohol is imperatively demanded. Again, it is always important: to ascertain the morbid condition, if there be any, under which the stomach is labouring—if inflammation, ulcer, or growth of any kind be present, if there be obstruction at the pylorus or at the cardia, if the stomach be dilated or contracted, and so on; and to determine the treatment in accordance with the nature of the lesion which is present. It is of equal importance to ascertain whether the dyspeptic symptoms are secondary to any constitutional disturbance, such as anæmia, phthisis, or gout, which happens to be associated with them, in order that we may direct our treatment to the relief or cure of the essential disease.

The above remarks are not intended to distract attention in any degree from the actual symptoms which cause the patient's sufferings. These generally need special treatment; but, guided by the principles which have been laid down, we may in most cases so select or so combine our remedies as, on the one hand to relieve local symptoms,

on the other to remedy the conditions out of which the dyspepsia has arisen.

Loss of appetite is often very difficult of treatment. It may, however, in some cases be overcome by the use of vegetable tonics, especially gentian, quassia, calumba, or the liquid extract of cinchona, in combination with small quantities of rhubarb, aromatics, and an alkaline carbonate; or by the employment of quinine, strychnia or iron, or (if there be constipation) of aperients, especially rhubarb and aloes, in combination with aromatic bitters. But food has often to be administered when the patient has not only no desire, but possibly even a loathing, for it. It is then necessary either to study the patient's fancies by making frequent variations in the food which is placed before him, or to administer food of a wholesome and suitable character in small quantities and at short intervals. It is sometimes necessary indeed to employ nutrient enemata, and for a time partially or altogether to discontinue the use of food by the mouth.

Gastric uneasiness or pain needs different treatment according to the circumstances under which it arises or the conditions to which it is immediately due. When it occurs mainly during the period in which the stomach is empty, the obvious remedy is the ingestion of food; it may then be necessary to take meals at more frequent intervals than in health, or to relieve the uneasiness in these intervals by taking a biscuit or some other light and easily digested refreshment. It not unfrequently happens that persons otherwise healthy who have nothing after their dinner at five, six, or seven o'clock, wake in the middle of the night with more or less gastralgia, or complain of similar pain with nausea and perhaps sickness in the morning. The proper treatment for such cases is the taking either of a light supper before going to bed or of a light meal before rising in the morning. When pain occurs immediately after the ingestion of food it implies the presence of some morbid irritability, inflammation, or organic mischief in the walls of the stomach, and may be treated partly by regulation and selection of diet, and partly by the use of drugs, such as nitrate of silver, hydrocyanic acid, or bismuth, given before food. If the pain be dependent on flatulent distension, peppermint, ginger, and other carminatives are generally useful. Mineral acids, and the earthy or alkaline carbonates, are often valuable in relieving pain, as they are in relieving other dyspeptic symptoms. It is not always easy to determine *a priori* which remedies are best suited for any particular case. It may, however, be assumed as a general rule that, when the secretions of the stomach are alkaline or neutral, as they are apt to be in inflammatory conditions, acids are indicated; that when they are acid, alkalies, if not specially indicated, are at all events more suitable. Opium is of great value in the relief of gastric pain, and may frequently be advantageously combined with other remedial agents, especially perhaps with bismuth or kino. When the gastralgia is severe, and especially if it be of a

spasmodic character, and associated with faintness or collapse, opium may be regarded as our sheet-anchor. It should be given in large, and, if necessary, repeated doses. Blisters and other counter-irritants, or fomentations to the epigastric region, are often useful.

For *flatulence and eructation*, carminatives, and more especially the essential oils, some of the oleo- or gum-resins, ammonia, or brandy in small quantities, are generally beneficial; but they are beneficial rather by assuaging present uneasiness and dispersing wind by eructation than by any direct curative influence. For relieving these conditions, however, as well as for checking vomiting, careful attention to the quality, quantity, and times of administration of food must always be paid.

Nausea and *vomiting* may be benefited by various agents: by ice in small quantities; by the alkaline carbonates, which may often be advantageously given in an effervescing form with lemon-juice or citric or tartaric acids; by oxalate of cerium, carbonate of magnesia, lime-water, bismuth, nitrate or oxide of silver, hydrocyanic acid, or creasote. When flatulence, eructation, and vomiting are dependent on, or associated with, fermentation or putrefaction of the contents of the stomach, special treatment may be called for; fermentation may be checked by the use of creasote, sulphite of soda, or sulphurous acid; putrefaction by the exhibition of the mineral acids and more especially hydrochloric acid, with which pepsine may be combined.

In pyrosis or water-brash the above forms of treatment may be serviceable, but generally bismuth alone or combined with opium, or the vegetable astringents conjoined with a narcotic—the compound kino powder, for example—appear to have a special value. In cases in which the stomach is excessively dilated it has been recommended to empty the organ from time to time by means of the stomach-pump, and then to wash it out.

Lastly, it must never be forgotten that in all cases of chronic dyspepsia hygienic treatment, inclusive of moderate exercise, regulated hours, well-ventilated rooms, and change of air and scene, is of considerable importance.

XIX. DIARRHŒA.

The term diarrhœa, like the term dyspepsia, is applied to a symptom or group of symptoms which is common to a wide range of morbid conditions, of which the majority are discussed with more or less completeness in various parts of this volume. It is needless, therefore, as well as inappropriate, to enter upon the subject here at any great length.

Causation.—Diarrhœa is of common occurrence at some period or other in the course of many febrile or other constitutional maladies. It not unfrequently complicates hepatic and splenic diseases, and other affections which induce undue congestion of the portal vessels and their tributaries. It is one of the ordinary consequences of organic lesions, of whatever kind, of the mucous membrane of the bowels. It is frequently induced by inflammatory conditions of the same tract, by the ingestion of unwholesome or irritating articles of food, and by over-eating. It is certain also: that it is sometimes caused by nervous influences, and especially by anxiety, fear, and allied mental emotions; and that excessive or perverted secretion from the alimentary canal, or from the glands which open upon it, has a large share in its production. Among circumstances which exert an important influence in causing diarrhœa, are age, habits, and season, with other climatic conditions. Thus it is peculiarly frequent amongst young children, especially at or about the times of weaning and teething; again it, or its converse—constipation—is very apt to follow upon dietetic and other changes of habit; and, further, the influence of hot weather, and especially in this country of the later summer months, and of alternations of temperature, in its causation is a well-known fact.

In considering the pathology of diarrhœa, we shall first discuss the influence of the contents of the gastro-intestinal canal in its causation. It is the presence of alimentary matters which, in conjunction with that of the normal secretions, excites those peristaltic movements which terminate with defæcation. The bowels as well as the stomach are no doubt in many cases very long-suffering; yet, notwithstanding this, they are frequently stimulated to unwonted action by the matters which gain entrance into them. Excess of even wholesome food, the ingestion of difficultly digestible or unwholesome matters, the use of polluted water, even the transmission from the stomach of imperfectly reduced contents, or of such as are undergoing fermentation or putrefaction, are all likely to cause more or less intestinal disturbance, with consecutive diarrhœa. Again, excessive discharges from the liver or intestinal surface, especially if they assume an inflammatory character, do, even when themselves determined by the influence of irritating alimentary matters, materially promote the abnormal action of the bowels. Amongst causes of intestinal irritation must also be included prolonged constipation, or excessive accumulation of fæces.

Of the important part which the mucous surface of the bowels plays in relation to diarrhœa there is no room for doubt. It is, in fact, by the influence of the contents on this surface that they are themselves influential in causing it. The conditions of the mucous membrane which promote diarrhœa are (omitting morbid growths, degenerative changes, and other destructive lesions) irritability, irritation, and catarrhal inflammation. In the first case, the over-sensitive sur-

face resents the contact of the normal intestinal contents, and excites the muscular walls to propel them rapidly onwards; in the second, the healthy intestinal walls are excited to unwonted action and over-secretion by the irritating matters which are in contact with them; in the last there is actual inflammation present, with more or less important change in the character and quantity of the secreted juices.

Without the action of the intestinal muscular walls, diarrhœa could not exist; it is owing indeed to their powerful and frequently-recurring peristaltic movements, for the most part reflectorially excited from the mucous surface, that the contents of the bowels are carried onwards with unwonted energy. But their action is under the immediate direction of the sympathetic nerves, and it is quite possible (as has been proved experimentally) for energetic peristalsis to be excited by the direct irritation of these nerves, and hence for similar movements to be induced through their agency by causes originating in the central nervous organs or other remote sources of irritation. Trousseau, indeed, refers one form of diarrhœa, as well as one form of dyspepsia which is commonly associated with it, to increased tonicity of the intestinal and gastric muscles—a condition which, if it exist, is evidently dependent on nervous agency. The influence of depressing passions in causing diarrhœa is exerted obviously through the nervous system; but whether this operates by simply augmenting peristaltic movement or in the first instance promoting excessive flow of mucus and other fluids into the intestinal canal, is a question which it would be somewhat difficult to decide.

Symptoms and progress.—By diarrhœa we mean strictly the actual discharge from the anus of unformed or fluid motions in greater quantity or more frequently than natural. But owing to the remarkable length of the alimentary canal, and to the variations in its structure and functions in different parts of its course, we may have conditions which correspond essentially to diarrhœa developed at different parts and leading to different results. Thus if the affection involve the large intestine, diarrhœa (dysenteric in character) will certainly ensue; if, however, it attack the upper part of the jejunum, the diarrhœa (so to speak) may only occur between the jejunum and the ileum, or between these and the cæcum: the patient will suffer from colic or griping, but instead of frequent loose evacuations there may be actual constipation.

As regards the characters of the alvine discharges, there will necessarily be much variety, dependent partly on the nature of the ingesta, partly on the amount and quality of the secretions of the different glandular organs, partly on the fermentative and other changes which take place in the bowel, and partly on the rapidity with which the contents of the stomach are carried onwards to the anal orifice. We may discover in the evacuations solid masses of animal or vegetable matter, fat which has not been saponified, comparatively large quantities of

only slightly modified starch, and, in young infants, coagulated but otherwise scarcely altered milk. They may contain large quantities of mucus, unmixed if it be secreted by the large intestine, incorporated and imparting pallor and fluidity if it be furnished by the remoter portions of the bowel. Or the discharges may be exceedingly copious and almost watery in character, and may contain either large quantities or merely traces of biliary colouring matter. Fluidity of the evacuations may be due in large measure to simple hurry in the transmission of the contents of the bowels, and to the consequent escape with the fæces of those natural secretions which under normal circumstances would have been reabsorbed; there is no doubt, however, that in a large number of cases it is dependent in a greater or less degree on excessive secretion. When fermentation or decomposition occurs, there is, attending the diarrhœa, much discharge of flatus, which is often exceedingly offensive; and the evacuations, which are more or less watery and fetid, present a frothy or yeast-like character. Under these circumstances the yeast-fungus or the sarcina ventriculi may generally be discovered in them. When the contents are propelled along the intestinal canal with great rapidity, there is insufficient time for digestion, at all events for intestinal digestion, to be efficiently performed; and it is under such circumstances that the condition termed '*lientery*,' or the passage of undigested food, frequently takes place. We may here call attention to the fact that ovarian and other cysts, hydatid tumours and abscesses, may open into the bowel and give rise to diarrhœal stools, of which their contents form an important and more or less obvious constituent.

The essential symptoms of diarrhœa are pain and the occurrence of loose stools; but with these are usually associated others of more or less severity and importance. Pain of an aching, griping, or colicky character is generally present, coming on at intervals, attended with borborygmi and more or less manifest movements of the bowel, and varying in its seat. It differs in severity, and is sometimes so intense that the patient rolls about or writhes in agony, and a state of partial collapse, with coldness of surface, perspirations, and feeble pulse, is induced. If it be developed high up in the course of the bowel, vomiting not unfrequently takes place; if it occur in the lower part of the large intestine, spasmodic expulsive actions of the abdominal muscles are excited. There is not usually abdominal tenderness; the pain, indeed, is often relieved by pressure or friction. In some cases of diarrhœa, copious evacuations take place with little or no uneasiness or pain. The different characters of the stools have already been detailed. It remains to say: that the quantities discharged vary within wide limits, and are sometimes as enormous as they are in cases of epidemic cholera; and that the frequency of the evacuations presents equal variety. Among the associated symptoms, which may or may not be present, are dryness or coating of the tongue, soreness of the mouth and fauces, anorexia and thirst, nausea, vomiting, and eructation, giddi-

ness or headache, and, as has been already stated, symptoms of faintness or collapse, sometimes alternating with flushes of heat and slight febrile symptoms. When diarrhœa is profuse, and at the same time acquires a chronic character, innutrition with more or less rapid emaciation and loss of strength ensues: and death may ultimately result either from simple exhaustion or from the supervention of complications.

It is scarcely necessary to specify in detail the different characteristic features of the many various forms of diarrhœa which are met with in practice, or to insist on the extreme difficulty and frequent impossibility of distinguishing functional diarrhœa, which is now under consideration, from the diarrhœa of intestinal lesions. There are two forms of diarrhœa, however, which call for particular remark, namely, '*infantile diarrhœa*' and '*summer* (*English*) *cholera*.' Although receiving different names it would be difficult to draw any clear line of distinction between the morbid conditions here associated. We shall, therefore, combine their description. Infants, especially at or about the time of weaning, are remarkably apt to be attacked with diarrhœa, and to fall victims to it; and this tendency is greatly increased during the summer months when diarrhœal complaints are common not only in children but in adults. The attack, whether in the infant or the adult, sometimes comes on suddenly, sometimes supervenes in the course of some slight gastro-intestinal disturbance. It usually commences with copious and repeated vomiting, first of the normal contents of the stomach, then of watery fluid, containing bile. The diarrhœa is at first characterised by the expulsion of the contents of the lower bowel but little altered; but gradually the evacuations become more and more thin and watery, although still tinged more or less strongly and not unfrequently green with biliary colouring matter. With these phenomena are associated intense thirst, much pain and griping in the belly, which is usually retracted, and cramps in the limbs, together with more or less marked collapse, indicated by coldness of surface, rapidity and feebleness of pulse, pinched features, sunken eyes surrounded by dark circles, bluish finger-nails, sighing respiration, altered voice, and restlessness. The symptoms have, in fact, a close resemblance to those of Asiatic cholera, but differ from them clinically in the circumstances: that the evacuations rarely if ever assume the rice-water character, or are devoid of bile; that the urine is not generally suppressed; and that the collapse is neither so sudden nor so extreme as that of the epidemic disease. Nevertheless the affection is very dangerous, carrying off a very large proportion of the children whom it attacks, and not unfrequently proving fatal to adults. If recovery take place from the stage of collapse, a febrile stage ensues in which the temperature rises, the surface assumes a normal or febrile aspect, the tongue gets red and dry, and the evacuations (which probably remain diarrhœal) acquire something of a dysenteric character. The patient becomes dull and lethargic, and, if a child, falls into a con-

dition of stupor, with moaning, plaintive cries, and jactitation, which may readily be mistaken for symptoms of cerebral disease. The period of collapse lasts from a few hours to twenty-four or thirty-six hours; and it is especially during this period that death is likely to occur. The later stage may be continued for several days or for a week or two.

Treatment.—The treatment of diarrhœa must depend mainly upon the causes to which it is due and the symptoms with which it is attended. When it is distinctly the consequence of alimentary errors, it is usually best at the commencement to aid the removal of offending matters either by emetics, such as mustard and water or a full dose of ipecacuanha, or by purgative medicines such as hyd. c̄ cretâ, blue pill, Gregory's powder, compound rhubarb pill, an ordinary black draught, or castor-oil. Such measures may effect a cure; but if the diarrhœa still persist, carminatives and astringents may be requisite. Of these compound kino powder, aromatic chalk and opium, chalk mixture, or lime-water or bismuth combined with vegetable astringents, opium or rhubarb, may be efficacious. If these fail recourse may be had, according to circumstances, to tannic acid, lead and opium, copper, perchloride of iron, nitrate of silver, or sulphuric acid. An essential element in the treatment, however, and one which is alone often sufficient for the purpose, is partial or complete abstinence from food for a time, and subsequent limitation of the patient's dietary to such matters as are bland and easily digestible. Milk, arrow-root, and such-like substances, broths, toast, and simple well-baked biscuits are especially suitable. In the case of young children no purgative medicines probably are better than chalk and mercury, Gregory's powder, and castor-oil; and no combination of astringents and aromatics better than aromatic chalk and opium, or small quantities of catechu, opium, aromatic chalk, and syrup of ginger in solution. In this case, too, especial attention must be paid to diet. If the child has been weaned it may be necessary to supply it again from the breast; or to provide it with asses' or goats' milk; or to feed it with skimmed cow's milk to which lime-water may be added, or with well-baked flour or suitable biscuit-powder diffused or suspended in water or milk. If the diarrhœa be of distinctly inflammatory origin, very much the same kind of treatment is needed; purgatives may still be requisite in the early stages, but castor-oil or salines are probably to be preferred. Dietetic treatment also in these cases is of great importance. If there be much abdominal uneasiness or griping, warm fomentations or mustard plaisters to the parietes, or the warm bath, may be beneficial.

In the choleraic form of diarrhœa, which attacks young children and adults, mainly in the summer time, little or nothing can be done at first to arrest the diarrhœal phenomena. Trousseau regards the mustard bath (made by enclosing a cold paste of mustard in a muslin bag and squeezing this in the water of the warm bath until the latter is

sufficiently impregnated) as the most powerful and efficacious remedy; and directs that it should be employed for about a quarter of an hour, or until the mustard causes some tingling of the surface, and that it should be repeated if necessary. For internal treatment the exhibition of iced water, rice-water, decoction of barley, skimmed milk, or the *eau albumineuse* of Trousseau (made by diluting the whites of four eggs with about 1¾ pints of water, sweetened with sugar and flavoured with orange-flower water) may be resorted to with advantage. During the same period emetic doses of ipecacuanha, and purgative doses of the hyd. c̄ cretâ are of common use and strongly recommended; but if the collapse be serious, diffusible stimulants, such as ether and ammonia, or some form of alcoholic beverage, are demanded. At this period opium is a remedy of questionable efficacy, and in the case of young children should be carefully avoided. With the cessation of diarrhœa and vomiting and the supervention of febrile symptoms the diet above recommended must still be continued, but the medicinal treatment must now be that which is beneficial in catarrhal inflammation of the bowels, and may include such drugs as bismuth, chalk, and lime-water, with opium.

Chronic diarrhœa is often very intractable, and requires much judicious management for its successful treatment. Hence attention to diet is of supreme importance. It is impossible, however, to lay down any definite rules in reference thereto. In many cases fluid nutriment is most suitable; in some food is best administered in the solid form; in some the farinacea agree best; in some alimentary matters derived from the animal kingdom. We must be guided in each case, partly by the patient's own feelings and experience, partly by the special symptoms present and the character of the evacuations, and partly, of course, by the opinion which we form of the nature and origin of the attack. In such cases it not unfrequently happens that the diarrhœa is kept up by the habitual use of some unsuitable article of diet, or by the constant presence of some hygienic condition inimical to the patient's health, or by the continued indulgence in habits which are injurious to him. It is in chronic diarrhœa (especially in children) that the use of raw meat to the exclusion of all other food has been so strongly advocated. The lean of beef or mutton should be selected, minced, pounded in a mortar, squeezed through a sieve, and given either in the form of the simple pulp, or mingled with sugar, red-currant jelly, or other similar substances. It should be administered at first in small doses, and then gradually increased. Trousseau has thus given as much as a pound a day to a child of less than three years old. We must not forget the danger which in taking raw meat our patient incurs of becoming affected with tænia—a danger which both Trousseau and Goodeve have shown not to be fanciful. For medicinal treatment we may have recourse to the various vegetable astringents and bitters, or to bismuth, silver, copper, or iron, or to the mineral

acids, or to rhubarb or ipecacuanha, or to opium, nor must we forget the benefit which may result from the occasional administration of saline or stomachic purgatives.

Section IV.—DISEASES OF THE LIVER AND PANCREAS.

I. INTRODUCTORY REMARKS.

A. *Anatomical Relations.*

In investigating hepatic diseases, a careful examination of the hepatic region should not be neglected. The healthy liver occupies the right hypochondrium, extending across the scrobiculus cordis into the left hypochondrium; and throughout this extent is accurately adapted to the vault of the diaphragm. In the recumbent posture the lower edge is usually concealed by the lower margin of the right side of the chest, except in the upper part of the epigastric region, where small portions of the right and left lobes lie uncovered. The position of this edge varies, however, during respiration—descending somewhat in inspiration, ascending again in expiration; it descends also to a slight extent when the sitting or upright posture is assumed. Moreover, in women who lace tightly, and occasionally in other healthy persons, it may be found as much as two or three inches below the margins of the ribs. In some cases, on the other hand, it occupies normally a higher position than usual. The upper limit of the liver necessarily corresponds to the position of the diaphragm with which it is in contact, and is higher, therefore, on the right than on the left side. The upper margin, however, of that area of its upper surface which has only the diaphragm and thoracic walls in front of it occupies a lower level, corresponds to the lower and outer margin of the right lung, and varies with the varying positions of that margin. Adopting Frerichs's estimates, it may be assumed that (liable to more or less variation): in the nipple line, the true upper boundary of the liver corresponds to the fifth interspace, the line of separation between the edge of the lung and the liver to the sixth rib; in the axillary line, the former to the seventh interspace, the latter to the eighth rib; and near the vertebral column, the former to the tenth interspace, the latter to the eleventh rib. It must be added: that all that region to which the liver is immediately subjacent is dull or nearly so on percussion; and that in front and to the left the upper part of that region merges in the cardiac area, and below and behind in that of the right kidney. General increase in the bulk of the liver is attended both with the ascent of the upper margin of the hepatic area into the chest, and with the descent of its lower margin into the abdomen—the latter being necessarily the

more considerable. The lower margin then can generally be well distinguished, with all its characteristic peculiarities of outline. In some cases it descends into the iliac and hypogastric regions. When, however, the enlargement of the liver is irregular, or due to the presence of tumours, in some cases its extension is wholly at the expense of the thoracic cavity, and the walls of the lower part of the right side of the chest may be distinctly protruded over it; in other cases its extension takes place mainly downwards, and the irregularity of form of the affected organ may then be readily distinguished through the abdominal walls. When the liver diminishes in size its area of dulness shrinks correspondingly, and sometimes wholly disappears. Occasionally, moreover, under these and other circumstances, the intestines rise up and intervene between the liver and parietes.

B. *Physiological Considerations.*

In entering upon the subject of the diseases of the liver it is important that we should have some preliminary acquaintance with the nature of the functions which this organ has to perform, and on the disturbance or modification of which many of the more important or striking phenomena of hepatic disease necessarily depend. In the brief review of this subject, which we are now about to place before the reader, we shall avail ourselves largely of the masterly summary given by Dr. Murchison in his work on the functional derangements of the liver.

The liver appears to have at least three important and more or less distinct offices to fulfil. First, starchy and saccharine matters, brought to it by the portal vessels from the alimentary canal, are converted by it into glycogen ($C_6H_{10}O_5$), a substance resembling dextrine, and convertible, like it, into sugar by the action of albuminoid ferments. Glycogen is formed and stored in the hepatic cells, whence (especially during the intervals of fasting) it is removed in the form of sugar by the hepatic veins, and then distributed: partly, for the maintenance of heat, to be converted by the respired oxygen into carbonic acid and water; partly to take an important share in the growth, development, and functional activity of cells, and probably even in the development of the white corpuscles of the blood. Further, glycogen is probably convertible into fat, and, under certain circumstances, the source of accumulation of oil in the hepatic cells, or of adipose deposition in other parts of the body. Second, albuminous matters, whether derived directly from the food, or constituting an essential part of the blood, and especially fibrine, appear to become reduced, through the agency of the liver, into various simpler compounds. These include glycogen, the destination of which has already been considered, and effete matters, such as leucine ($C_6H_{13}NO_2$) and tyrosine ($C_9H_{11}NO_3$), which are ultimately resolved into uric acid ($C_5H_4N_4O_3$), and more

particularly into urea (CH_4N_2O), and then discharged with the urine. Urea itself appears, at all events to some extent, to be manufactured in the liver. Third, the liver secretes bile. This is a thin, transparent, golden-yellow fluid, which gets viscid and assumes a darker colour in the gall-bladder in consequence of its admixture with mucus. Roughly speaking, about two pints of bile are secreted daily by a healthy adult, of which from 9 to 17 parts per cent. consist of solid matters. These comprise small though varying proportions of mucus, fat, salts, and compounds due to the disintegration of albuminous substances, but mainly certain ingredients of special interest and importance, namely resinous acids in combination with soda, colouring matter, and cholesterine. The resinous acids, which are two in number, are the glycocholic and the taurocholic; they are both conjugate acids, the former being formed by the union of glycocoll ($C_2H_5NO_2$) with cholic acid ($C_{24}H_{40}O_5$), the latter by the union of glycocoll with taurine ($C_2H_7NO_3S$). The latter acid contains all the sulphur of the bile, and to it the bitterness of this fluid is due. The peculiar colour of bile is owing to the presence of a pigment now termed bilirubine ($C_{16}H_{18}N_2O_3$). This readily undergoes oxidation even in the gall-bladder, becoming successively yellow, green, brown, and black. Bilirubine crystallises in ruby-coloured, rhomboidal crystals, which are scarcely, if at all, distinguishable from hæmatoidine crystals, but chemically are said to contain one atom more of carbon. Cholesterine forms a small but constant part of the solid constituents of the bile, and is usually the main constituent of biliary calculi. As regards the sources of the essential ingredients of the bile, it has been maintained by some that they are, like urea, formed in the blood, and simply separated from it by the liver; by others that they are a product of that disintegrating power which the liver itself has over the albuminous and other matters which are brought within its influence. The latter view is now generally accepted. Taurocholic and glycocholic acids appear, therefore, to be products of that disintegration of albuminous substances to which reference has already been made; and bilirubine to be a derivative of the colouring matter furnished by disintegrating blood-corpuscles. It has been suggested by Dr. Austin Flint, junr. that the cholesterine of the bile is to be traced to the disintegration of nervous tissue, and that one of the chief functions of the liver is the separation of this fatty matter from the blood. The purpose and destination of the bile have been equally a matter of dispute. There is little doubt, however, that the bile is an important agent in the saponification and absorption of fats, and even in the assimilation of albuminous matters, and further, that it promotes peristaltic action, and arrests decomposition. It is certain that it is only in some small degree excrementitious, the great bulk of it, like saliva and gastric juice, being reabsorbed, in a more or less modified condition, into the system. The excrementitious parts comprise portions of the colouring

matter and of cholic acid, and certain derivatives of cholesterine. The parts which are reabsorbed comprise the taurine, the glycocoll, the greater part of the cholic acid, and a considerable proportion of the colouring matter; which last, there is reason to believe, becomes converted into the pigment of the urine.

C. *Pathological Considerations.*

It will be readily understood from the above observations how numerous and various are the ways in which diseases of the liver may affect the nutritive and other processes of the body, and how numerous and various are the symptoms to which they may give rise. Diabetes has long been regarded as a functional affection of the liver; and Dr. Murchison attributes to functional disturbance of this organ not only gout, renal calculi, and biliary calculi, but a large proportion of the functional and structural derangements of nearly all the organs and tissues of the body. The most striking, if not the most important, results, however, of hepatic diseases are those which are connected with derangement or suppression of the biliary secretion—namely, jaundice and various associated phenomena, which will presently be fully considered.

A further consequence of structural disease of the liver, or of any disease implicating the trunk of the portal vein, is impediment to the ready flow of blood through this vessel or through its branches of distribution to the liver, and hyperæmia of the tributary vessels connected with the other chylo-poietic viscera. This hyperæmia leads to various mechanical consequences, especially to permanent dilatation of the vessels, which when occurring in the vicinity of the anus constitutes hæmorrhoids; to more or less profuse hemorrhage from the mucous surface of the alimentary canal; and to abdominal dropsy.

Jaundice.—This is due to the circulation with the blood, the deposition in various tissues, and the separation, through the agency of certain unwonted emunctories, of the colouring matter of the bile, and its various modifications. But, as we have pointed out, the bile contains other ingredients besides colouring matter, and the hepatic cells have other functions to perform besides the mere manufacture of bile. It is obvious, therefore, that the existence of jaundice—the circulation of biliary colouring matter—almost necessarily involves the circulation of other ingredients of the bile which are less readily detected, and probably also the presence in the blood in greater or less abundance of various effete derivatives of albuminous matters.

But what is the explanation of the accumulation of biliary colouring matter in the blood? By those who hold that the liver excretes bilirubine and the other constituents of the bile exactly as the kidney excretes urea, jaundice is attributed to loss or diminution on the part of the liver of its dialysing power. There is ample evidence, however,

from the results of removal of the liver in the lower animals, that bile is not formed in the blood, and that as a general rule the presence of the liver is essential to the production of jaundice. It is certain also that when jaundice follows the experimental obstruction of the hepatic ducts, it first manifests itself in the hepatic cells and then in the lymphatic vessels which take their origin in the liver—facts which clearly demonstrate that in this case at all events the jaundice is due to the passage into the general circulation of colouring matters manufactured in the liver. It has hence been assumed, and doubtless with truth, that in those cases in which jaundice is due to obstruction, the colouring matter of the bile formed in the cells is absorbed both from the cells, and from the hepatic ducts behind the seat of obstruction, by the hepatic venous capillaries and lymphatics, and thus becomes distributed throughout the system. It has also been assumed, but on a far less substantial basis, both by Frerichs and by Murchison, that jaundice may arise, in cases of prolonged constipation and in cases of excessive secretion of bile, from absorption taking place at the mucous surface of the bowel.

But if the hepatic cells are in a condition to manufacture bilirubine they are doubtless also in a condition to manufacture the biliary acids. What, then, becomes of these? They are absorbed, together with biliary colouring matter, and mingle with the circulation, but what becomes of them further is still a matter of dispute. Dr. G. Harley and others assert that they accumulate in the blood, and are discharged with the urine, in which fluid they may be detected by appropriate means. Frerichs, on the other hand, and Dr. Murchison agrees with him, believes not only that they are never found in the urine, but that in the blood they speedily undergo chemical changes, and their identity becomes lost. Frerichs, it may be added, holds that the reabsorbed biliary acids may be converted into biliary pigment, and that hence their absorption may increase jaundice if it do not absolutely create it.

But, even if it be admitted that the above explanation holds good of all those cases in which jaundice is due to obstruction of the hepatic ducts, and that it may be extended to cases of jaundice (if there be such) referrible to intestinal obstruction, or to excessive production of bile, it is clearly inapplicable to some, at all events, of those cases in which (as in pyæmia and certain infectious fevers) jaundice is an item of a general disease, and to the cases in which (as probably in malignant jaundice) the icteric tinge is associated with the destruction of the secreting cells of the liver, and their consequent inability to discharge their specific functions. The close relationship of the colouring matter of the blood to that of the bile, and the facts that the one is derived from the other; and that both yield parallel series of almost identical coloured derivatives, make the view which Virchow strongly advocates—namely, that in many of the latter cases, jaundice

is due, not to the agency of the liver, but to changes in the hæmatine effected in the general circulation—both highly probable and easy of acceptance. Frerichs, however, even in reference to some of these cases, prefers to believe that the jaundice is attributable to the absorption of the elements of bile at the mucous surface of the bowel and to interference with the due course of those changes which the reabsorbed bile should undergo in the blood. It is important to add: that when, as in malignant jaundice, the secreting structure of the liver is destroyed, the jaundice is not usually intense, and the biliary acids are certainly never detected in the urine, while on the other hand, the retrograde metamorphosis of albuminous matters remains incomplete, and leucine and tyrosine, which accumulate in the blood, replace more or less completely urea in the urine; and that, according to Dr. G. Harley, Kühne and others, when jaundice is the result of obstruction, the biliary acids may be recognised in the urine, and that their recognition there may be taken as a proof of the obstructive origin of the jaundice.

The phenomena which attend and indicate jaundice, and the consequences which flow from it are very various, but the more important of them may be readily enumerated.

1. There is usually deficiency of bile in the alvine discharges, and more or less consequent tendency to constipation, flatulence, fetor of the evacuations, faulty assimilation, especially of fat, and distaste for fat. If the bile be wholly absent, these phenomena are more strongly marked, and the fæces acquire a chalky, grey or slaty colour, or assume some tint referrible to the prevailing character of the patient's diet. Diarrhœa sometimes comes on; and fatty matters, but little altered, are apt to pass away with the evacuations.

2. Omitting for the present all reference to the changes taking place in the liver itself, the colouring matter of the bile first accumulates in the blood, then escapes with the urine, and subsequently gradually tinges the conjunctivæ and skin, passing off at the same time in some small degree with the sweat. Other parts which become bile-stained are the serous membranes and all effusions which take place in connection with them, the connective and fibrous tissues, fat, muscles, and bones. The mucous membranes as a rule are scarcely affected; and the secretions from their surfaces and from the glands which open upon them are usually entirely free. The brain and nerves remain for the most part uncoloured. The only secretions besides those of the kidneys and sudoriparous glands, which have certainly been found to contain bile, are that of the mammary gland and those furnished by *inflamed* mucous surfaces. Superficial jaundice first shows itself in the conjunctivæ, but soon becomes generally diffused throughout the whole cutaneous surface. It is at first a mere condition of sallowness, but soon assumes a saffron or golden yellow hue, and if long continued, a brownish, olive, or bronze-like tint. The seat of discolouration is

mainly the rete mucosum and the sudoriparous glands. The secretion of the latter, indeed, sometimes becomes so largely charged with bile-pigment as to stain the linen. The usual characteristics of bilious urine, and the tests for the recognition of bile-pigment in that fluid, are elsewhere considered. It may, however, be pointed out here that the urine varies in colour from a saffron-yellow to a greenish or brownish black, that its froth always presents a peculiarly yellow hue, that it stains white paper and linen, and, further, that it is generally free from sediment, transparent, and acid. It is apt, however, to present other peculiarities, of which some have been already adverted to; it commonly yields uratic or other deposits; it may possibly, when the jaundice is obstructive, contain bile acids; it certainly displays, in the presence of extensive destruction of the hepatic cells, a remarkable diminution of urea and of phosphates, and in their place a great abundance of leucine and tyrosine, which then sometimes fall as a greenish-yellow sediment; and, lastly, there is often, especially towards the fatal close, either glycosuria or albuminuria, or both. Albuminuria is probably connected with the irritation caused by the long-continued passage of bile-pigment. All the tissues of the kidneys, and more especially the cells of the convoluted and straight tubes, gradually get deeply stained; and the canals of the tubes are not unfrequently occupied by granular or amorphous pigmented casts, which become shed and may be found in the urine.

3. There are a number of other phenomena occasionally associated with jaundice, of which some are interesting, others are of grave importance. It is asserted that sometimes all objects appear yellow to jaundiced patients. But this occurrence is rare, and the explanations which have been given of it are conflicting. There is often troublesome, and sometimes unbearable, itching of the skin. This is not generally attended with obvious eruption; but occasionally we find lichen, urticaria, or some one of the different varieties of erythema multiforme. Vitiligoidea or xanthoma is well known to be frequently associated with chronic jaundice. The action of the heart is usually much enfeebled, and often reduced in frequency; there is also a marked tendency to the occurrence of hemorrhage, revealing itself by the appearance of petechiæ, or by epistaxis or gastro-intestinal or other fluxes, which may be so copious, or so frequently repeated, as to prove fatal. Together with these symptoms the patient usually becomes emaciated and feeble, irritable or low-spirited, and little capable of resisting the influence of either mental or bodily fatigue or changes of weather.

It is not surprising that patients suffering from jaundice should sooner or later present impairment of nutrition and other indications of profound ill-health. It is surprising rather that they should live as long as they occasionally do, and yet present so few symptoms and undergo so little suffering; and that bile itself should, as has been

shown by experiment, have so little injurious influence over the blood and the various corporeal functions. Occasionally, however, symptoms of so-called 'bilious toxæmia' arise. They seem, however, to occur mainly, if not solely, in those cases in which jaundice is connected with destruction of the hepatic cells, in which urea tends to disappear from the urine, and leucine, tyrosine, and other products of albuminous decomposition circulate with the blood and find their way into the urine. And, indeed, the toxæmic effects seem to be due, not to the influence of the proper elements of bile, but to that of various excrementitious matters, of which leucine and tyrosine are probably the most important. The symptoms here referred to comprise: in the first instance, headache, restlessness, mental depression or excitement, and sense of illness; then busy or violent delirium, or convulsions, varying from mere rigors to general epileptiform attacks or tetanic spasms, or delirium and convulsions intermingled; and finally stupor, passing into coma and death.

II. INFLAMMATION OF THE HEPATIC DUCTS.

Causation.—Inflammation of the biliary ducts and of the gall-bladder may be due to the presence of gravel or calculi, or to the extension of inflammation from the parenchyma of the liver. These subjects will be considered hereafter. But inflammation affecting mainly the mucous surface, and catarrhal in character, frequently arises from exposure to vicissitudes of weather, or gastro-intestinal disturbance, and perhaps also in connection with pneumonic and other acute inflammations, and various infectious fevers. It is most frequently preceded by similar affection of the duodenum.

Morbid anatomy.—Catarrhal inflammation is generally indicated by an excessive discharge of ropy mucus, and by a swollen condition of the mucous membrane; and these phenomena not unfrequently lead to more or less complete obstruction. This usually occurs in the common duct, and especially in that part of it which is embraced by the intestinal walls. It is generally temporary, subsiding in the course of two or three weeks or less, but sometimes results in organic stricture, sometimes in permanent closure, or in closure which is relieved only by the supervention of more or less extensive ulcerative destruction. In some instances a false membrane forms upon the mucous surface, and occasionally polypi or papillary growths are developed. The most remarkable consequences of catarrhal inflammation are those which are dependent immediately on mechanical impediment to the escape of bile, and which therefore are associated from the begin-

ning with mechanical distension of the ducts. These are suppuration, hemorrhage, ulceration, and more or less extensive destruction of the mucous membrane.

Symptoms and progress.—The early symptoms of catarrhal inflammation of the gall-ducts, which are usually undistinguishable from those of the gastro-intestinal catarrh with which they are associated, are mainly flatulence, distension, weight and pain in the region of the stomach, nausea and vomiting, and for the most part constipation, together with slight febrile disturbance. The proof of implication of the hepatic ducts is furnished after the affection has lasted for severa days, possibly a week or two, by the supervention of jaundice with some degree of tenderness and enlargement of the liver. In many cases the symptoms preliminary to jaundice are so vague and slightly developed that they escape observation; and not unfrequently they subside shortly after the supervention of jaundice. The jaundice itself, however, with constipation and other results of retention of bile, usually continues for a week or two, or a little longer. Catarrhal inflammation almost always ends in resolution within the period above assigned. Sometimes, however, it becomes chronic, and may then continue for months with the combined symptoms of gastro-intestinal irritation and retention of bile, the patient probably becoming feeble and emaciated. The consequences of permanent stricture, or of complete impediment to the escape of bile, will be considered under the head of 'Obstruction of the hepatic ducts.'

Treatment.—In treating the affection under consideration, febrifuge or alkaline medicines, and saline purgatives, or mild laxatives of other kinds are generally indicated. Food of an unstimulating character, for the most part farinaceous substances and milk, should be administered. And local pains or uneasiness should be counteracted by hot fomentations or counter-irritants. Leeches or cupping is rarely if ever necessary. It is often well to have recourse to those remedies which have a special influence over the morbid conditions of the gastro-intestinal mucous membrane, and especially to stomachic combinations, such as mixtures of soda, potash, or bismuth with rhubarb, ginger, and some bitter infusion. Emetics also have been strongly recommended in the early stages of the disease, with the object mainly of promoting the flow of bile along the obstructed tubes, or of effecting the dislodgment of the plugs of mucus which it is assumed may be impacted in them.

III. ACUTE HEPATITIS. ABSCESS OF THE LIVER.

Causation.—Acute inflammation arises under various circumstances. It may be due to injury; to the irritation of adventitious growths or

hydatids; to extension of inflammation from the ducts or veins or from without; to pyæmia, whether taking its origin in ulceration of the bowel or from some more remote source; to pre-existing congestion of the organ, especially in connection with heart-disease; to the influence of acute inflammations, such as erysipelas and pneumonia, or of the specific fevers, such as yellow fever, small-pox, and scarlatina; to the toxic effects of phosphorus and certain metallic poisons; and lastly, it is not unfrequently of idiopathic origin, mainly, however, in tropical climates and in connection with dysentery. The nature of the relation between dysentery and hepatic abscess has already been discussed under the head of 'Dysentery.'

Morbid anatomy.—Inflammation of the liver, as of other organs, affects mainly the connective tissue and the small vessels in the meshes of which the proper elements of the gland are contained. The vessels become dilated and full of blood with a superabundance of leucocytes; the tissues get infiltrated with inflammatory exudation; and a development of embryonic tissue takes place in the walls of the small vessels and ducts, and their immediate vicinity, in Glisson's capsule, and generally in the connective tissue. At the same time the proper cells of the liver become swollen and cloudy and even the seat of fatty or pigmental deposition, and are sometimes destroyed. The appearances presented by the inflamed tissues differ widely in different cases, in dependence mainly on the causes to which the inflammation is due and on its intensity. In some cases the most marked features are uniform opacity and lightness of tint, with doughiness of consistence, and enlargement of the lobules. These peculiarities are due to the fact that the hepatic cells have become cloudy and swollen; while, in some measure owing to this very circumstance, there is but little inflammatory exudation present, and the vessels contain little blood. When a limited portion of liver is thus affected, the pallid and swollen patch is usually surrounded by a more or less diffused area of congestion; when the whole organ is implicated, the pallor may be universal, or it may be marbled with patches of congestion. As will hereafter be shown, there is reason to believe that the condition known as yellow atrophy of the liver is probably an inflammation of the kind here referred to.

Inflammation ending in abscess is probably always circumscribed and at the same time necessarily intense. The early stage of suppuration is usually indicated by pallor, opacity, and swelling of a definite patch of liver substance, the tissues immediately surrounding which and for some distance beyond are generally more or less deeply congested. Soon the affected patch softens, and then breaks down. The process is identical with that of the formation of abscesses elsewhere: embryonic cells make their appearance in large numbers, the hepatic cells become swollen, granular, fatty, and fall into detritus, and the web of connective tissue in which they are imbedded liquefies and

disappears under the influence of the inflammatory exudation and growth. If the abscess extend, these processes gradually involve the surrounding structures, partly by simple extension, partly by the development of new foci in the immediate vicinity. And, under such circumstances, it not unfrequently happens that abundant shreds and filaments of a pinkish grey hue and soft consistence hang from the parietes of the abscess into its cavity, and that these if traced outwards are found to merge into a pulpy flocculent network, infiltrated more or less abundantly with pus, and from the meshes of which the disintegrated liver-cells have disappeared. The purulent contents vary in character, and are not unfrequently tinged with bile; they are often glairy and of a greenish hue. Hepatic abscesses vary in their seat, size, and number. They may be found in any part of the liver. They may range from the size of a pin's head up to that of a cocoa-nut, and, indeed, are sometimes much larger than this. In the Netley Museum (according to Dr. Maclean) is an hepatic abscess which contained no less than seventeen pints of pus. They may be solitary, or may amount numerically to 20, 30, or even 100. Idiopathic abscesses are for the most part solitary, or at any rate occur in small numbers. When they are numerous there is reason to suspect a pyæmic origin. It is important, however, not to overlook the remarkable influence which the various tubes permeating the liver exert over the distribution and multiplication of abscesses. Pus or inflammatory lymph gaining entrance into the portal veins may be conveyed in the form of emboli until they become arrested in vessels too small for their further transit; when as a result congestion, followed by softening and suppuration, of the areæ to which the obstructed vessels lead takes place. This condition not unfrequently ensues upon a spreading hepatic abscess: a branch of the portal vein in the substance of the liver becomes involved; pus in more or less abundance enters the affected vessel and is thence distributed amongst its ramifications, causing, sometimes the formation of scattered abscesses in some particular district, sometimes a series of branching abscesses due to the conversion of the veins themselves into suppurating channels. The same thing may occur in connection with the hepatic vein; some large branch of which may get perforated by an advancing abscess; when, communication with the cava having been cut off by the formation of a plug, the pus may flow backwards into the tributary branches, and a ramifying abscess result. Again, one of the hepatic ducts may be the seat of a like mischance. It may become perforated by an hepatic abscess which may then discharge itself into the bowel; or, the duct becoming obstructed below the seat of perforation, the pus may be driven back into the smaller branches. Moreover, inflammation, commencing in the biliary ducts, sometimes leads to abundant suppuration, and occasionally to the almost complete destruction of their parietes and the development of irregular branching abscesses. Some of the terminations of hepatic abscesses

have been indicated in the foregoing statement. It remains to add that they may discharge themselves in various directions: as, for example, through the external abdominal parietes; through the diaphragm into the pericardium, pleura, or lung; or into the peritoneum, stomach, duodenum, or colon. Sometimes the abscess burrows, and it may then take almost any route: either infiltrating the tissue of the great omentum; or running downwards in the meso-colon or behind the peritoneum and thus finding its way into the cæcum or rectum, bladder or vagina, or taking either of the courses which an ordinary psoas abscess is apt to take. In some instances the abscess ceases to spread, the tissues around get thickened and indurated, and the matter becomes encysted, and undergoes fatty, caseous, or calcareous change.

Symptoms and progress.—The symptoms which attend inflammation of the liver are exceedingly various and often so slight as to elude observation. They include enlargement of the organ, which may be detected by inspection, palpation, and percussion; weight, and uneasiness or pain in the hepatic region, which last is often increased, and perhaps only developed, by pressure, change of position, or the respiratory acts; occasional sympathetic pains in the right shoulder, and possibly down the right arm; disturbance of the digestive organs, indicated by fulness, flatulence, nausea, sickness, and loss of appetite; and slight febrile disturbance. Jaundice not unfrequently supervenes, but is rarely intense. It must be added that the pain is always most severe when the surface of the liver is involved; that it is then of a pricking or cutting character, resembling that of ordinary acute peritonitis; and that it is in this case chiefly that the movements of respiration become affected, as in diaphragmatic pleurisy, that the sympathetic pain in the shoulder manifests itself, and that a dry hacking cough is induced.

The symptoms of hepatic suppuration are in many cases vague and misleading. This is no doubt due in part to the fact that hepatic abscess so often supervenes in the course of dysentery or pyæmia—affections which by the severity of their proper symptoms tend to overshadow those of the hepatic complication. It is not, however, due entirely to this cause; for idiopathic suppuration, independent of dysentery, and the suppuration which complicates hydatid tumours, not unfrequently (for a time at least) fail of recognition. The local indications of abscess are pain and tenderness in the region of the liver, tumour in the same situation, displacement of neighbouring organs, and interference with their functions. Pain and tenderness may be almost entirely absent; and when present they vary largely in intensity and extent. They are generally most severe when the abscess approaches the surface and this becomes implicated. The pain is then, as in peritonitis, of a pricking or cutting character. When the abscess is deep-seated, it is usually dull and aching. Pain referred to the right shoulder is not unfrequent during the progress of suppu-

ration. Whether or not there be any obvious enlargement of the liver depends partly on the number, partly on the size, partly on the situation of the abscesses. An abscess, even of large dimensions, situated at the back, or deeply imbedded, may easily escape observation. It may, indeed, lead to the descent of the anterior edge of the liver, and so induce a belief in the uniform enlargement of the organ, which is of course no sufficient indication of the presence of an abscess. When, however, the abscess is situated more anteriorly, it tends gradually to form a rounded mass, which increases more or less rapidly in size, protrudes the parietes, and sooner or later probably yields a distinct sense of fluctuation. This protrusion sometimes occupies the scrobiculus, and may then involve also more or less of the umbilical and hypochondriac regions; sometimes it takes place principally upwards, displacing the lungs and heart; not unfrequently it occurs mainly towards the right side of the chest, in which case the base of the lung may be displaced upwards considerably above the level of the nipple, and the corresponding part of the thoracic walls, with more or less of the adjoining hypochondrium, may form a smooth round swelling. It is always important in these cases to determine the exact limits of the hepatic mass and of the area of dulness. As regards neighbouring organs, the diaphragm is not only frequently displaced, but from the implication of the convex surface of the liver is often embarrassed in its action, and respiration becomes thoracic, shallow, and painful, and a dry hacking cough and hiccough arise; the stomach, again, is often displaced, and nausea, vomiting, and other dyspeptic symptoms may consequently ensue. The general symptoms due to hepatic abscess are various. The most important probably are those of fever. Fever, however, is sometimes wholly absent, and may indeed be absent during the entire progress of cases attended with extensive suppuration. More commonly, however, there is some elevation of temperature, either at the commencement of suppuration, or at some intermediate period of its progress, or at a late stage when the surface of the liver becomes involved, or during the whole course of the case. The temperature does not commonly rise above 102° or 103°, presents for the most part morning remissions and evening exacerbations, and is sometimes attended with chills or even severe rigors, which in their severity and periodicity may simulate those of ague. The fever, if persistent, assumes a hectic character, and is attended with profuse perspiration, especially at night time. Jaundice is by no means a necessary accompaniment of hepatic abscess. It is occasionally present, however, and is then usually slight. The condition of the digestive organs varies considerably. Sometimes they are but little affected; sometimes, on the other hand, the tongue may be coated or dry, and thirst, loss of appetite, flatulence, nausea, vomiting, diarrhœa, and other indications of gastro-intestinal irritation or catarrh, may be developed.

Hepatic abscess is always an affection of great danger, and frequently

proves fatal. In some cases death is due simply to the impairment of nutrition and the extreme debility which extensive suppuration entails. Sometimes these conditions are associated with the persistence of a febrile temperature, or with the retention of effete matters in the blood; and the patient, previous to death, lapses into a typhoid state, with dry brown or black tongue, subsultus tendinum, and muttering delirium. In a large number of cases, however, complications ensue, dependent mainly on the bursting of the abscess into, or the extension of inflammation to, some neighbouring organ. The nature of these has already been sufficiently considered under the head of morbid anatomy. For their symptoms we must refer to the accounts of the diseases of the several organs which may be thus implicated. When restoration to health occurs it may be due either to the abscess becoming encysted and undergoing degeneration, or to the discharge of its contents through the abdominal walls, or lung, or into the bowel.

Treatment.—The treatment of hepatic inflammation must depend on the nature and severity of the symptoms by which it is attended. Febrifuge or alkaline medicines, and saline purgatives or mild laxatives of other kinds, are generally indicated. Food of unstimulating character, for the most part farinaceous substances and milk, should be administered. And local pains or uneasiness should be counteracted by hot fomentations or cold applications, or by counter-irritants, such as mustard plaisters and the like; or if there be much feverishness, and the local phenomena be at the same time severe, by the use of leeches or the cupping-glasses. Amongst drugs which are frequently had recourse to in the treatment of these affections (especially when they are severe or chronic in character), and which are for the most part highly esteemed, are iodide of potassium, chloride of ammonium, taraxacum, and nitro-muriatic acid. During convalescence good diet, change of air, and tonics are always valuable.

In the earlier stages of hepatic suppuration, nothing probably arises to call for special treatment. When, however, the abscess is so far developed as to render its presence pretty certain, the question of evacuating its contents arises. It is a moot point whether it should be allowed to take its own course, to open when and where it pleases, or whether it should be punctured at the earliest possible opportunity. Dr. G. Budd is a strong advocate of the former plan. Many, however, prefer the latter procedure, and we are of their number. The risks, indeed, which attend the progress of an abscess are, partly from the amount of disorganisation it produces in the liver itself, partly from the uncertainty as to the route it may take, so serious, that they can scarcely be aggravated by operative procedure and they may be largely diminished by it. We believe the best plan is to evacuate the contents as soon as opportunity offers, by means of a fine trocar and cannula with or without the aid of the aspirator. This operation may generally be safely effected even if no adhesions have formed between the

liver and the abdominal walls. It is well to avoid the admission of air, and to employ an evenly and firmly applied bandage afterwards. The operation may be repeated from time to time. If the discharge become offensive, and adhesions have formed, a free opening should be made and maintained; and the cavity of the abscess should be occasionally washed out with weak solutions of some antiseptic, such as chlorinated soda, nitric acid, carbolic acid, or quinine. During the progress of these cases the patient's strength should be supported by tonics, stimulants, and nutritious diet. Opiates are always of great value. It may of course be necessary to treat gastric and other complicating disorders.

IV. CIRRHOSIS OF THE LIVER.

A. *Atrophic Cirrhosis.* (*Hobnailed or Drunkard's Liver.*)

Causation.—Atrophic cirrhosis is almost always a consequence of the persistent excessive use of alcoholic beverages. This, however, is not the sole cause of the disease. For it is occasionally met with in persons who have been undoubtedly temperate in their habits, and in children who have never taken stimulants. It is probably more common in men than in women; and is generally first recognised in persons above 35.

Morbid anatomy.—In typical cases the liver is much reduced in size, rounded, studded with hemispherical elevations for about $\frac{1}{4}$ to $\frac{1}{3}$ inch in diameter, and obtusely marginated. Moreover generally its capsule is thickened and old adhesions bind it to the diaphragm and neighbouring organs. On section it is found to be remarkably indurated; and the same nodulated condition which marks the surface is manifest throughout its substance. This nodulation is due to the permeation of the liver by a network of dense greyish fibroid tissue, within the meshes of which the proper parenchyma of the organ is contained in the form of roundish bodies, varying roughly from the size of a tare to that of a large pea. The fibroid growth occupies mainly the capsule of Glisson surrounding the small branches of the portal vein, and the vaginal veins, which give rise to the interlobular plexuses, and extends thence in a greater or less degree into the interlobular spaces. On close inspection it will generally be seen: that the larger hepatic nodules are made up of smaller ones—the latter comprising several hepatic lobules; and, moreover, that there is always a more or less definite line of demarcation between the annular bands of fibroid tissue and the groups of hepatic lobules which they circumscribe. The proper hepatic tissue of the hobnailed liver may present its normal hue; but not unfrequently it is fatty and jaundiced and has a

light or orange yellow tint—whence the name 'cirrhosis.' There is reason to believe that in the early stage of the disease the liver is somewhat enlarged, and at the same time smooth on the surface. Atrophic cirrhosis appears to originate in a chronic inflammatory condition of the branches of the portal vein above indicated, attended with the abundant appearance in the tissues immediately surrounding them of embryonic cells, which undergo slow conversion into cicatricial fibroid tissue, and then gradually contract. The inflammatory process rarely implicates the substance of the hepatic lobules even at their periphery; but by the compression to which they are subjected the liver cells become flattened and atrophied, and after a while disappear to a greater or less extent. It is an interesting fact, in connection with the morbid anatomy of the disease, that the adventitious fibroid growth (notwithstanding that by involving the smaller branches of the portal vein it interposes a serious obstacle to the portal circulation) is itself, unlike ordinary cicatricial tissue, richly permeated with large tortuous vessels of capillary character. These, though still communicating in some degree with the portal system, seem specially to serve as a route for the passage of the blood of the hepatic artery to the intralobular vessels; and it is probably in consequence of their presence or development, that the nutrition of the liver and the formation of bile are usually maintained to the last. Further, as the smaller biliary ducts are not necessarily implicated, bile is not as a rule retained, and jaundice rarely occurs. The obstruction of the portal veins leads to hyperæmia and distension of the tributary vessels, and consequently to dropsy, hemorrhage, enlargement of the spleen, development of hæmorrhoids, and vicarious dilatation of veins—more especially of those about the umbilicus and in the abdominal walls above the umbilicus.

Symptoms and progress.—Atrophic cirrhosis is for the most part very insidious in its progress. In many cases no symptoms manifest themselves, sufficient at any rate to attract attention, until the affection is far advanced; in many, the patient suffers only from the usual symptoms of dyspepsia or chronic gastric catarrh—symptoms which may equally occur in the absence of hepatic disease; in many, he has vague indications of ill-health with progressive loss of strength and emaciation, and these phenomena may be associated with distinct evidence of similar disease going on in the kidneys; in some, no doubt, slight signs of hepatic derangement show themselves from time to time, and, in association with the habits or history of the patient, reveal to the careful observer the momentous changes which are going on within. The hobnailed liver, excepting perhaps in its earlier stages, is usually atrophic, and the normal hepatic dulness is consequently diminished in area or suppressed; but the presence of an enlarged liver by no means forbids the diagnosis of this disease. Its chief indications are the supervention of abdominal dropsy, and the occurrence of hemorrhage (often profuse) from the stomach and bowels.

The more frequent of these is no doubt ascites, but it does not necessarily become developed even in fatal cases; and even when once it has appeared, will sometimes subside under appropriate treatment, and never recur. Hæmatemesis and melæna are sometimes the first indications of the presence of the hepatic disease; and the first attack may prove fatal. When once such hemorrhage has occurred, it has a marked tendency to recur; and it is a phenomenon of very fatal augury. Jaundice supervenes in a minority of cases, and is very rarely intense. Besides the symptoms just enumerated, others of more or less importance are commonly present:—There is usually progressive and finally extreme emaciation, with a sallow or earthy cachectic aspect. There is generally more or less obvious disturbance of the digestive functions; the tongue becomes coated or dryish, and there may be thirst, loss of appetite, sense of flatulent distension, nausea and vomiting; the bowels may be constipated or relaxed, and, indeed, diarrhœa, which is apt to assume a dysenteric character, is not an unfrequent precursor of death; piles are of common occurrence. The urine is often scanty and loaded with lithates. And there is a liability for hemorrhage to take place from the various mucous membranes and beneath the skin. The tendency of cirrhosis is always to a fatal issue, but the duration of the disease is almost impossible to ascertain. It may certainly last for many years; but when once distinctive symptoms have shown themselves, the patient's days are numbered. He may, however, even then survive for a year or two. The immediate causes of death are various. The natural termination is by gradual asthenia. But the patient is often carried off by the consequences of the ascitic accumulation, by gastro-intestinal hemorrhage, by profuse alvine discharges, or by the supervention of pneumonia or other pulmonary complications. Not unfrequently also the hepatic affection is only one of a series of lesions of an allied character involving, it may be, heart, lungs, spleen, kidneys, and other organs.

B. *Hypertrophic Cirrhosis.*[1]

Causation and morbid anatomy.—This is a form of cirrhosis which has long been recognised and, so far as its coarser features are concerned, described by pathologists. But it has only recently been distinguished from the ordinary hobnailed liver as a condition having a different pathology and different consequences. The causes of hypertrophic cirrhosis have not been clearly established; but at any rate there is no distinct evidence that it has any connection with alcoholic intemperance. In this condition the liver is enlarged, sometimes to twice or even thrice its normal size; is tolerably smooth on the sur-

[1] Hanot, 'Etude sur une forme de cirrhose hypertrophique du foie.' Thèse de Paris, 1876. Charcot. 'Leçons sur les Maladies du Foie, &c.' Paris, 1877.

face; and retains its normal form and its sharp well-defined anterior edge. It is extremely dense in texture, and on section is found to be largely infiltrated with dense greyish slightly translucent connective tissue. This does not form a distinct network as in the hobnailed liver, circumscribing definite groups of hepatic lobules, but is rather a diffused growth in which the remnants of the hepatic parenchyma are scattered irregularly in yellowish masses from the size of a poppy seed to that of a pea. In some parts the hepatic tissue predominates and there is an approach to the hobnailed condition; in some almost every remnant of the natural structure has disappeared, and dense fibroid tissue alone remains. The remnants of hepatic parenchyma vary in colour from orange-yellow to green. Microscopically the fibroid growth shows the same characters as those of ordinary cirrhosis; and in its early stage and when it is in progress of extension it consists mainly of embryonic cells. But it commences, not from the terminal branches of the portal vein, but from the interlobular branches of the bile-ducts, and from those branches of them which occupy the periphery of the lobules. The morbid process begins in fact with the development of embryonic tissue immediately around these channels, whence it spreads—implicating the portal vessels at an advanced period only. It tends also to involve the lobules themselves, which as a rule escape in atrophic cirrhosis. The affected ducts become largely dilated and their epithelium increases in quantity. The smaller branches, indeed, get blocked up with their accumulated contents. The liver cells are atrophied, contain biliary pigment, and are more or less fatty. It is important to note that in this disease the obstruction which the fibroid growth causes is chiefly of the ducts, and that the portal circulation is not necessarily implicated.

Symptoms and progress.—Hypertrophic cirrhosis is a disease mainly of adult life, and like the atrophic variety is of slow progress. The ordinary symptoms of derangement of the gastro-intestinal organs, gradual emaciation, and failing strength belong to both affections. But whereas the main consequences of hobnailed liver are ascites and gastro-intestinal hemorrhage dependent on obstruction to the portal system; in this case these phenomena are for the most part conspicuous by their absence, and dropsy if it occurs at all comes on late, and then probably only in a slight degree. On the other hand, jaundice, which is generally absent, and never well developed in atrophic cirrhosis, always appears early in this disease, acquires considerable intensity, and persists during its whole course. It is liable, however, to fluctuations. It need scarcely be added that with the jaundice we have all the characteristic phenomena which attend and follow the presence of bile in the blood; and that consequently such patients are specially liable to cerebral and other toxæmic symptoms. The detection by palpation of an hypertrophied, hard, and sharp-edged liver is an important element in the diagnosis of these cases. The spleen is usually

hypertrophied. But the superficial abdominal veins are probably not enlarged.

C. *Other Conditions allied to Cirrhosis.*

Causation and morbid anatomy.—Among these we may enumerate:—the diffused form of cirrhosis observed mainly in syphilitic children; chronic inflammatory conditions involving especially the capsule of Glisson surrounding the larger vessels and ducts; and chronic perihepatitis attended with compression and atrophy of the liver.

1. The cirrhotic condition of liver, which occasionally arises in the course of syphilis, whether in the adult or in infants, and which is not unfrequently associated with the formation of gummata, is characterised (see Cornil and Ranvier), as is hypertrophic cirrhosis, by uniform enlargement of the organ; but the enlargement is due to the general development of embryonic tissue—not simply in the interlobular spaces, but in the lobules themselves, between the columnar groups of cells, and even separating the individual cells from one another. In this affection the liver is at first readily lacerable, but as the embryonic tissue becomes converted into fibrous tissue the organ becomes indurated; and its general condition presents a close resemblance to that of hypertrophic cirrhosis. 2. It not unfrequently happens, sometimes in the course of syphilis, sometimes in connection with obstructive affections of the ducts, but often under conditions which are not understood, that growth of fibroid tissue takes place along the vessels and ducts which enter at the transverse fissure. This condition is sometimes general, sometimes limited to certain regions; and may have the effect of causing more or less obstruction of the ducts or portal veins or both; and frequently leads to the formation of deep fissures on the surface of the liver, and to a lobulated condition of that organ. 3. Perihepatitis may occur as a part of general peritonitis; and generally takes place in a greater or less degree in connection with the different varieties of cirrhosis. Sometimes the fibroid investment is exceedingly thick and dense; and by its contraction compresses the liver into a roundish mass with obtuse ill-defined edges. This sometimes occurs when the general texture of the liver is healthy, but necessarily causes more or less compression, atrophy, and obstruction of vessels and ducts.

Symptoms and progress.—It would be difficult to define the symptoms and consequences of these several conditions. It is sufficient to say that besides inducing, like hobnailed liver, general symptoms of ill-health, they are likely in various degrees to be attended sooner or later with the specific consequences of obstructive hepatic disease, namely, ascites, gastro-intestinal hemorrhage, and jaundice.

D. *Treatment of Cirrhosis.*

As regards hobnailed liver, more important than its treatment by medicine is the avoidance of those habits of indulgence in alcoholic drinks on which it seems mainly to depend—and it should be borne in mind that it is not so much the occasionally getting drunk which is dangerous in this respect, as the habit of constant tippling. Even if the disease be in progress, the discontinuance of this habit must be of real benefit to the patient, inasmuch as that condition which keeps the morbid process active then ceases. It is important that this fact should not be forgotten; for the victim of atrophic cirrhosis for the most part so craves for his accustomed stimulant that the physician is apt unwisely to indulge him in his cravings. But besides abstinence from alcohol, the patient should attend carefully to hygienic measures; his diet should be light, nutritious, and not too stimulating or too abundant; he should keep good hours, be warmly clad, and take moderate exercise. Further, he should be put under a course of vegetable bitters, with (especially if there be gastro-intestinal catarrh) the addition of some stomachic. Or he may take one of the drugs often recommended for cases in which hepatic inflammation is assuming a chronic character:—namely, iodide of potassium, chloride of ammonium, taraxacum, or nitro-muriatic acid. The bowels should be kept freely open, but violent purging should be avoided. When the various late complications or results, such as ascites, hæmatemesis, and melæna, dysentery, or jaundice, supervene, they will of course require special treatment. But for details we must refer to the articles devoted to these several subjects.

The remarks just made in regard to the treatment of atrophic cirrhosis are applicable in the main to the other forms of chronic hepatitis. If, however, there be reason to suspect a syphilitic origin it is obvious that antisyphilitic remedies should be prescribed.

V. CONGESTION OF THE LIVER. (*Nutmeg Liver.*)

Causation.—Congestion of the liver necessarily attends inflammation of the organ; it occurs also in connection with over-eating, excessive use of alcohol, sedentary habits, and exposure to atmospheric influences, especially great heat; and it is a common consequence of ague or exposure to malaria, and of various febrile and inflammatory disorders. The most interesting form, however, of congestion is probably the mechanical congestion which arises in the course of obstructive lung and heart diseases, more especially diseases of the mitral valve, and

valves of the right side of the heart. It is this form of congestion alone which we shall now consider.

Morbid anatomy.—Congestion of the liver is attended with more or less enlargement of the organ due to distension of its vessels, and mainly its veins and capillaries, with blood. When general the liver structure presents a more or less uniform deep-red hue, and blood escapes in abundance from the cut surface. In cardiac or mechanical congestion it is the hepatic veins and their minute branches occupying the centres of the lobules which are chiefly if not exclusively involved. These become dilated and full of blood, and the liver consequently undergoes more or less considerable increase in bulk. If sections of the organ be made at this time it will probably be found that the centres of the lobules are deeply congested while their peripheral parts are more or less markedly pale. With the progress of the disease the hepatic texture undergoes important changes; in consequence of the increasing dilatation of the intra-lobular hepatic veins the cells which lie in their meshes undergo atrophy and perhaps finally disappear; the cells immediately bounding this region often become deeply jaundiced and the seat of granular biliary pigment, and hæmatoidine crystals are occasionally deposited; and the peripheral cells of the lobules get more or less distended with oil. In this stage the liver is often larger than natural, though probably smaller than it was at first; it is apt to be somewhat granular on the surface with a more or less thickened capsule; it presents some degree of induration; and on section the surface is found to be thickly studded with small circles or festoons of an opaque buff or bright yellow colour, interwoven with discs or small lobulated patches of intense perhaps black congestion. The appearance has been not unaptly compared to that of the sectional surface of a nutmeg. It may be added that, in connection with atrophy of the cells of the central parts of the lobules, a development of fibroid tissue takes place at length, and that thus a condition approaching to that of cirrhosis supervenes.

Symptoms and progress.—The symptoms of congestion of the liver or of the congestive hepatitis which takes place in the course of obstructive cardiac or pulmonary disease are: uniform enlargement of the organ, which probably descends an inch or two below the lower margin of the right ribs, and encroaches to an abnormal extent on the right half of the thoracic cavity; pain and fulness in the hepatic region, with considerable tenderness on pressure or percussion; pain or tenderness on lying on the right side; pain also in lying on the left side from the tendency of the liver to drag; and pain on drawing a deep breath or coughing. Slight jaundice is apt to supervene and to persist after all other symptoms of hepatic affection, excepting enlargement of the organ, have subsided. It is in these cases that hepatic pulsation is occasionally observed. Hepatic engorgement comes on as a rule late in the progress of cardiac and pulmonary diseases; and

although it often subsides under treatment it is very apt to recur, and then to become more or less permanent. When the congestion has become chronic and the liver shrunken and indurated, ascites and other consequences of portal obstruction, as in ordinary cirrhosis, are apt to ensue.

The *treatment* of hepatic congestion is mainly of course that of the pulmonary or heart-disease which causes it. In other respects it may be regarded as identical with what has already been prescribed for inflammation of the ducts of the liver or the earlier stages of acute hepatitis.

VI. MORBID GROWTHS.

A. *Tubercle.*

This affection is much more common in the liver, in connection with tuberculosis of other organs, than is generally supposed, but has no clinical importance whatever. Miliary tubercles are most frequently met with, and are often present in considerable numbers; but, owing to their close approximation in colour to the hepatic lobules and their extreme minuteness, are apt, excepting they be at the surface, to elude detection. Occasionally tubercles of the average size of a pea or bean are observed. These always present a central cavity full of broken-down tissue and biliary colouring matter, with a capsule of yellowish or greyish tubercular growth.

B. *Syphilis.*

Morbid anatomy.—Syphilitic disease is recognised post mortem chiefly by the presence of gummata, which have already undergone retrogressive changes. These are opaque, buff-coloured, dense, tough masses, rounded or irregular in form, and varying from about the size of a pin's head to that of a chestnut. They are rarely solitary, and are often grouped in clusters of considerable bulk. They are incapable of enucleation, and are imbedded in dense fibroid or cicatricial tissue, which is continuous, on the one hand, with the bodies just described, on the other, with the surrounding hepatic texture. They are mostly solid; but occasionally, when one is permeated by a duct, the latter is broken down into a cavity within it. Wherever these masses with the surrounding cicatricial tissue are present, the hepatic surface which corresponds to them is thickened, drawn in, and sometimes very deeply indented—facts which prove the chronic nature of the affection, and that much contraction of tissue has attended its progress. Not unfrequently, in cases where many of these tumours are present, we also find dense masses of cicatrix-like tissue, which are either free from

obvious tumours in their interior, or which, in place of them, present merely a few opaque or gritty particles.

The conditions above described are, however, only the last phases of a more acute syphilitic affection. The influence of the syphilitic virus on the liver is in the first instance to cause interstitial inflammation, which, as we have shown, has considerable resemblance to that of the early stage of ordinary cirrhosis. This affection may be general throughout the liver, or confined to certain areæ; and it is in connection with it that, sooner or later, gummata make their appearance. These are due to the active proliferation of certain of the cell-elements of the newly formed fibroid tissue, which increase in number and diminish in size, and collectively form tumours which have a close resemblance to granulation tissue or tubercle in the early stage. These growths then rapidly degenerate in their central parts, while they increase peripherally, so that at an early period they present caseous masses surrounded by a thin rim of living cell-growth. After a while they cease to enlarge, and the whole mass undergoes caseous degeneration. Gummata may occur in any region; they are common on the convex surface of the liver, and especially, as Virchow points out, in parts exposed to injury. They are common, also, in the neighbourhood of the transverse fissure, and may there seriously interfere with the permeability of the ducts and vessels. They usually vary in size between that of a pea and that of a walnut; but they may be larger or smaller, and are often aggregated. Although interstitial hepatitis is a common result of congenital or hereditary syphilis, the firm cheesy masses just described are rarely discovered in that variety of the disease.

Symptoms.—The symptoms which may be looked for in hepatic syphilis are those of cirrhosis in its various stages, especially, therefore, ascites, intestinal hemorrhage, and jaundice. But it must be admitted that syphilitic disease is, from first to last, often unattended with symptoms, and that it is not unfrequently discovered post mortem in cases where its presence during life had never been suspected. When, however, the gummatous growths obstruct the vena portæ or the hepatic duct, the symptoms due to such lesions will necessarily manifest themselves with considerable, and perhaps sudden, intensity. The detection of some irregularity of form, or manifest but sluggish tumour, in the liver may aid our diagnosis. The chief grounds, however, for suspecting the presence of syphilitic disease in this organ would be the association of symptoms of hepatic disorder with a history of syphilis and visible indications of its presence in a constitutional form.

Treatment.—In addition to the treatment suitable for cirrhosis and its consequences, the use of antisyphilitic remedies is obviously indicated in the treatment of hepatic syphilis.

C. *Non-malignant Growths.*

Under this head we may make a brief reference to two varieties of morbid formations which have little more than a pathological interest. These are *simple cysts* and *cavernous tumours*. The latter are small, blackish, spongy masses, rarely exceeding the size of a filbert; replacing definite portions of hepatic substance; and consisting of irregular intercommunicating vascular spaces, separated from one another by trabeculæ of fibrous tissue covered with pavement epithelium. Simple cysts vary from scarcely visible points up to the size of an orange. They are sometimes solitary, and are then usually situated about the middle of the anterior edge of the liver. Occasionally they are present in enormous numbers, when they display all grades of size and varieties of grouping. They are generally thin-walled, and in some cases give evidence of their enlargement by the coalescence of neighbouring cysts; they are lined with pavement epithelium, and usually filled with clear serous fluid. The smaller cysts sometimes contain yellowish or brownish colloidal masses like those found in renal cysts. Cysts, however numerous they may be, rarely, if ever, induce hepatic symptoms; it is possible, of course, that the presence of one of large size in relation with the anterior edge might be detected by manual examination; as a matter of fact, however, they are rarely, if ever, recognised during life. The most interesting point in connection with them is the fact of their comparatively frequent association with cystic developments in other organs, more especially in the kidneys and spleen. They must not be confounded with hydatid cysts.

B. *Malignant Growths.*

Morbid anatomy.—Malignant tumours of the liver are usually secondary to similar growths originating elsewhere in the body, and especially, perhaps, to such as are developed in the other chylo-poietic viscera. Not unfrequently, however, they are primary. No age is exempt from liability to the disease; yet it rarely occurs before adult age, and is most common in persons of middle and advanced life. It has been met with in young children, and in them is probably always a secondary manifestation. The influence of sex is unappreciable.

Malignant disease appears in the liver in two forms: either as isolated tumours or as a more or less general infiltration. In the former case the tumours vary in size from that of a good-sized orange, or even a cocoa-nut, down to minute granules, which the naked eye may fail to recognise. Their general form is globular, unless the coalescence of neighbouring masses, or accidental circumstances, have led to their irregular development. When they involve the surface of the liver, those areæ of disease which are immediately subjacent to the

capsule, and which are generally circular, assume a peculiar cupped appearance, due to the presence of a more or less prominent tumid peripheral ring, circumscribing a central concave depression. This cupping is very characteristic, and may frequently be recognised in tumours not more than a line or two in diameter as readily as in such as have attained the bulk of a chestnut or orange. There is usually more or less well marked vascularity of the superficial aspect of these tumours, and especially of their peripheral portions and of the liver-structure immediately surrounding them. The tumours grow at their margins, partly by progressively invading the healthy tissues bounding them, partly by the formation in their immediate neighbourhood of new foci of disease, with which they gradually coalesce. But while the marginal growth is in progress, the central portions fall into more or less rapid degeneration. This may be fatty, caseous, or even calcareous, or connected with hemorrhagic extravasation. Occasionally the central portions undergo liquefaction, and become converted into cysts containing a milky or watery fluid. These several forms of degeneration do not, as a rule, occur indiscriminately; each one, in fact, indicates to some extent an inherent peculiarity in the tumour in which it occurs, and which is shared more or less by all the other tumours which are in genetic relation with it. It is to the combination of active peripheral growth with central retrogression and necrosis that the superficial cupping to which reference has been made is mainly referrible. Malignant tumours may occur in any part of the hepatic substance; and may vary numerically from one or two to an innumerable multitude. In the former case they are usually primary, and it is here that probably the greatest size of growth is attained. In the latter they are generally secondary to growths elsewhere.

The diffused or infiltrating form of malignant disease is much more rare than that which has just been described. In this case we find the liver generally, or large portions of it, greatly enlarged, but retaining their normal shape: the enlargement being due to the abundant dissemination of small growths, more or less indistinctly defined from the liver-tissue, and tending to run together, so as to give to both the outer and the sectional surface of the affected organ a more or less spotty, reticulate, or uniformly morbid character. Sometimes, indeed, the naked eye fails to detect in the enlarged liver any traces of normal hepatic tissue. The presence of distinct rounded tumours may be associated with the condition here described.

Of the several forms of malignant disease which attack the liver, the carcinomata are the most common. The variety of cancer most frequently met with is the encephaloid, of which several sub-varieties, not, however, calling for description, exist; scirrhus is more rare; and still rarer than scirrhus are melanotic cancer and colloid cancer. Most of these appear under the form of isolated scattered masses. Sarcomatous malignant growths are comparatively unfrequent. The most

common and interesting of them is the melanotic variety, which is usually secondary to similar disease of the choroid coat of the eye, or of pigmentary nævi. Melanosis is usually very widely distributed throughout the organ; the tumours are small and tend to coalesce; and the condition above described as 'infiltrating' is apt to be produced; the liver often becomes enormously enlarged, and assumes, from the intermingling of melanotic spots with spots of colourless growth and remnants of hepatic texture, an appearance which has been aptly likened to that of granite. Melanotic masses of considerable bulk, however, are not uncommon. Other forms of sarcoma—spindle-celled sarcoma, for example, and the closely related myxoma—have been discovered in the liver, secondary to similar growths in remote organs. True epithelioma of the liver is scarcely more than a pathological curiosity. Cylindrical-celled epithelioma, however, or adenoma, secondary, for the most part, to gastro-intestinal disease of the same kind, is of much more frequent occurrence. Its tumours are scarcely distinguishable, excepting microscopically, from those of ordinary carcinoma. Lastly, lympho-sarcoma, or lymphadenoma, is often developed in the liver. This may form independent tumours, like carcinoma, but seems specially to affect the capsule of Glisson and the interlobular tracts; so that, in some cases, it involves the liver by ramifying through it with the portal vessels, in some it follows the ordinary distribution of the fibrous growth of atrophic cirrhosis, but in either case is apt to develop here and there into manifest tumours. Other forms of malignant disease besides lymphadenoma are liable to invade the liver from the transverse fissure. In cases of gastric or peritoneal cancer especially, the small omentum is very commonly infiltrated with cancerous growth, which thence propagates itself along Glisson's capsule, surrounding and compressing, or otherwise involving the veins and ducts. Again, the lymphatic glands in this situation are often affected secondarily to hepatic or other neighbouring malignant disease, and may then by their enlargement more or less seriously implicate the same channels.

Symptoms and progress.—The symptoms which attend malignant disease of the liver are in the main identical with those of cirrhosis and other structural diseases of the same organ. They comprise: alterations in the form and size of the organ, with local pain or uneasiness; impediment, mechanical or other, to the due performance of the hepatic functions; mechanical interference with the functions of neighbouring organs; and general impairment of nutrition. Increase of size and alteration of shape furnish very important indications of the presence of hepatic malignant tumours. The increase may be either uniform, or, as is more commonly the case, dependent on the formation of rounded projecting lumps, which may often be readily distinguished by the hand. Mere increase of size, however, is not so indicative of the morbid conditions in question as is rapid progressive

increase; nor is the simple fact of the presence of irregularity from outgrowths so suggestive as the progressive enlargement and development of such excrescences, and the existence of a certain degree of hardness and resistance which is not usually observed in mere cystic formations. It must be borne in mind, however, that malignant disease is often present in a liver which is not noticeably altered in form or size: the growths may be few and small, or they may occupy the posterior part of the organ, or the liver itself may be concealed by the overlapping of distended and adherent bowel, or by other conditions. And, further, tumours of the stomach, or of the retro-peritoneal glands, and even of the abdominal walls, may seem from their position to be of hepatic origin. Pain is, no doubt, a frequent attendant on hepatic malignant disease. Sometimes it is excruciating, and apt to come on in paroxysms; but it is often absent, and may be totally absent from first to last. Jaundice, usually due to obstruction of some of the hepatic ducts, makes its appearance sooner or later in a considerable number of cases. It is rarely intense, unless the main duct be involved; and hence it is chiefly in those cases in which the disease attacks the lesser omentum, and extends thence into the transverse fissure, that deep jaundice becomes developed. Jaundice, however, is by no means a necessary result, and is not unfrequently absent from the most extreme cases—cases in which the whole hepatic texture seems to be replaced by the morbid growth. Ascites is, perhaps even more rarely than jaundice, a direct consequence of malignant disease of the liver. It is often, no doubt, developed during the progress of the case, and may be due, as in cirrhosis, to impediment to the flow of blood through the portal vessels; but it is usually comparatively small in amount, and dependent either on peritoneal inflammation or on other abdominal complications. When, however, the portal vein is distinctly obstructed, the ascites may be considerable, and other consequences of portal obstruction, such as melæna, may ensue. In most cases ascites is absent. As regards neighbouring organs, the pressure of the enlarged and possibly painful liver is apt to induce functional disturbance of the stomach on the one hand, and pain and difficulty of breathing, and perhaps cough, on the other. General impairment of nutrition, debility, and emaciation are usually marked phenomena of the progress of the disease. Scanty secretion of urine, with abundant deposit of vermilion- or carmine-coloured urates, is commonly observed.

In most cases, malignant disease of the liver is associated with similar disease of other organs; and the symptoms which the patient presents are, therefore, of complicated origin. This fact, while it may be of the greatest value in enabling us to form a correct diagnosis of the malady under which he is labouring, often renders it difficult to determine how much and which of his sufferings are due to the hepatic lesion. As of malignant disease generally, so no doubt of that affecting

only the liver, it may be regarded as generally true: that the symptoms are insidious and progressive; that the disease has usually made more or less considerable progress before the suspicion arises that the patient is ill; that this suspicion is first aroused, either by the gradual creeping on of emaciation, debility, and cachexia, or by the slow supervention of gastric symptoms, or by a sense of fulness, heat, or pain—continuous or paroxysmal—in the hepatic region, or lastly by the discovery of obvious tumours. During the further progress of the case all the symptoms of this period of invasion are apt to become commingled, and the special phenomena which we have attributed to the declared disease to supervene. It must not, however, be forgotten that malignant disease of the liver may prove fatal without having ever been attended with some of those symptoms which would seem to be most typical of it: not only, as we have pointed out, may there never be obvious tumour, hepatic pain, jaundice, ascites, or distinct impairment of the digestive functions, but the so-called 'cancerous cachexia' may never be distinguishable, and the patient, instead of becoming emaciated, may remain in good flesh, or even become fat.

From the difficulty of determining the date at which it commences, it is impossible to determine, even approximately, the duration of hepatic malignant disease. Nor is it important to do so. It is sufficient for practical purposes to know: that when once the disease has given clear evidence of its presence, the patient rarely survives beyond twelve months; and that generally his death occurs within six or eight months. The natural cause of death is gradually increasing asthenia; but the fatal event is apt to be accelerated by the occurrence of peritonitis or other complications.

Treatment.—Medical skill is powerless to arrest the progress of the morbid growths under consideration. All that the physician can do is to relieve pain and uneasiness by opium or other sedatives, or by local measures; to check vomiting; to obviate constipation; and generally to aim at relieving the various symptoms which distress the patient; and by hygienic and other measures to maintain, as far as possible, his general health and strength.

VII. HYDATIDS OF THE LIVER.

Morbid anatomy.—These parasites affect the liver more frequently than any other organ; they are not uncommonly developed, however, in various parts of the sub-peritoneal connective tissue, more especially that of the pelvis. In the liver hydatid tumours are usually solitary; but sometimes two or more are developed there simultaneously; and occasionally also such tumours in the liver are associated with other

similar tumours elsewhere in the abdominal cavity. Their size varies: they are not unfrequently met with as large as a child's head, and containing several pints of fluid; but they are slow in attaining these dimensions; and although the exact period during which they live and grow is uncertain, there is no doubt that it occasionally extends to at least ten or fifteen years, possibly even to twenty or thirty. They are for the most part globular in form, unless bands or ligatures, or other accidental conditions, have interfered with their development. In the liver they most frequently involve the right lobe, a fact which is probably due simply to its comparatively large size. Hydatids appear to originate in the hepatic substance, which becomes displaced by them in the course of their development, and at the same time the seat of fibroid growth and induration in the layer which immediately surrounds them. By this means a kind of fibrous capsule is formed. In most cases there is no communication between the hydatid tumour and the hepatic ducts; sometimes, however, a large, and even a primary, duct may be found leading directly into the cavity, and its open continuation and that of some of its branches, studded with the orifices of their numerous tributaries, may then be seen ramifying upon its walls. The normal event of hydatid tumours, and one which is fortunately far from uncommon, is the death of the parasite, and the degeneration and contraction of the tumour. This has already been sufficiently described; it may, however, be added that hæmatoidine crystals, derived from the biliary colouring matter, are not unfrequently met with in such degenerated cysts. Other events of not uncommon occurrence are the rupture of the cyst by accidental violence and its suppuration.

Symptoms and progress.—Hydatid tumours are rarely attended with pain, or even uneasiness, excepting by reason of their bulk, and the pressure they exert on neighbouring parts, or in consequence of the supervention of inflammation. It generally happens, indeed, that the patient's attention, or that of friends, is first attracted by the discovery of gradual and at the same time more or less unsymmetrical abdominal swelling. So that when the case first comes under medical observation there is generally an obvious tumour in some part of the abdomen, and the question is consequently not so much whether or not a tumour is present, as what the nature of the existing tumour is.

Uninflamed hydatid tumours, which abut upon the surface, usually appear as rounded, tense, elastic swellings, free from pain or tenderness. They often fluctuate distinctly, and are not unfrequently attended with the peculiar hydatid thrill first described by Briançon and Piorry. This, which is best recognised by placing the left hand flat upon the tumour, and then percussing sharply with the fingers of the right hand, consists in a peculiarly long-sustained tremor, reminding one of that experienced on an iron railway bridge during the passage of a train over it. The nature of the swelling, however, may

generally be placed beyond the possibility of doubt by tapping. The fluid which comes away from the living hydatid cyst is transparent and colourless like water, limpid, containing an excessive quantity of chloride of sodium, and as a rule neither albumen nor fibrinogen. Its specific gravity varies from about 1008 to 1013; and its reaction is neutral or slightly alkaline. Further, it may contain echinococci or microscopic hydatids. The position of the tumour will necessarily vary with its seat of development. If in the liver, it perhaps most commonly projects forwards—occupying the scrobiculus, or this with more or less of the adjoining abdominal regions; but it may also protrude directly upwards, pushing the heart before it upwards and to the left; or it may displace the right half of the diaphragm, together with the base of the lung, at the same time distending the lowermost zone of the right side of the chest; or again it may be developed in the posterior region of the liver, and so elude detection. It is impossible to lay down any rules with regard to the situation of the tumour when it originates in other parts of the abdominal cavity. Suffice it to say: that it may, according to circumstances, assume the position of a renal, omental, ovarian, uterine, aneurysmal, or other growth; and that it is with these mainly (especially if they be cystic) and with hepatic swellings, more especially abscesses and dilated gall-bladders, that hydatid tumours may be confounded. When displacing the right lung upwards, and distending the corresponding part of the chest, they may simulate pleuritic effusion. Further, an hydatid cyst may be separated from the surface by a considerable thickness of the tissue in which it originates, or by an exceedingly thick and dense capsule, and hence may be mistaken for a solid tumour; or owing to the simultaneous development of several cysts, or to various other accidental circumstances, it may appear nodulated or multiple, and may present different degrees of consistence and elasticity at different points, and so may easily be taken for a lobulated malignant growth, or for a compound ovarian or other cystic tumour. The diagnosis of a contracted and degenerated cyst, even if occupying a situation readily accessible to examination, would, without the guidance of a clear history, be exceedingly difficult, if not impossible.

Hydatid tumours are not always unattended with symptoms; they may, from their bulk or situation, interfere seriously with respiration; they may cause vomiting and other dyspeptic phenomena; they may compress the hepatic ducts and so induce jaundice, or the portal vein, causing ascites, or the inferior cava, leading to anasarca of the lower extremities and probably congestion of the kidneys; and hence by the gradual supervention of asphyxia, asthenia, or other conditions, death may after a while ensue. The sudden rupture of hydatid tumours, with the escape of their contents into the peritoneal cavity, is usually followed by rapidly fatal peritonitis. The symptoms due to suppuration are sometimes obscure, sometimes very well-marked; they are

those, however, which usually attend extensive suppurative inflammation. The hydatid cyst in fact becomes converted into an abscess, and comports itself in its further progress exactly as any other large hepatic abscess. It increases more or less rapidly in size, and after a while discharges its contents either at the external abdominal surface, into the pleura, through the lung, into the pericardium, into the stomach, intestine, or abdominal cavity, or into the hepatic ducts and thence into the duodenum. Other rare terminations have been met with, such as by perforation of the vena cava, or right auricle of the heart. The proof that an hepatic or abdominal abscess is of hydatid origin rests on the discovery of hydatid membranes, echinococci or their débris in the pus which escapes. The hooklets, which are peculiarly indestructible, should especially be looked for.

Treatment.—No medicinal treatment avails either to cause the death of hydatids or to arrest their growth. For the cure of the disease we must look to local measures only; and these consist mainly in the evacuation of the contents of the cysts. The puncture of the cyst with a bistoury, or a trocar and cannula sufficiently large to admit of the escape of the cystic progeny of the parent hydatid, is a procedure which has been largely adopted. It is obvious, however, that it can only be employed with safety when the cyst is adherent to the abdominal parietes, and the escape of the contents into the peritoneal cavity thus prevented. It can only be justifiably had recourse to, therefore, when the cyst has undergone inflammation or suppuration and has consequently got united with the surface over it; or after measures have been taken to ensure the formation of adhesions. Among methods which may be adopted to effect this object are: first, incision through the abdominal parietes until the cyst is exposed; second, the gradual destruction by caustics of a limited area of the abdominal walls down to the parietal peritoneum over the intended seat of operation; and, third, Trousseau's method of multiple acupuncture. In all such cases it is essential that the patient should be kept at rest, and the abdominal walls in close apposition with the subjacent cyst-walls by means of pressure, in order to ensure the formation of adhesions and their maintenance when formed.

A far better plan, however, for evacuating the contents in all those cases in which suppuration has not yet occurred, is that which was strongly recommended some years since by Moissenet, and has since been successfully employed in this country, and especially in the Middlesex Hospital by Drs. Greenhow and Murchison. It consists in the employment of an exceedingly fine trocar and cannula. The minute puncture made by this instrument rarely permits, even if no adhesions be present, of the escape of any appreciable quantity of the hydatid fluid into the peritoneal cavity, and is rarely, therefore, followed by grave peritoneal complications. In order, however, to guard against such accidents it is well to select some prominent and central portion of the hydatid

protuberance for puncture, to refrain from removing the whole of the contents at one operation, and after the operation to keep the patient at perfect rest and the punctured parts in close apposition by means of a compress and bandage. It is further desirable to preclude the entrance of atmospheric air, and for this reason also, if the aspirator be not employed, to be content with the partial evacuation of the cyst. In consequence of the operation the hydatid collapses, and falls away from the walls of the adventitious cyst in which it is contained. The space thus formed becomes filled to a greater or less extent with serous exudation which soon gets turbid: and the hydatid bathed in the unwonted fluid generally soon perishes. The cyst, shortly after paracentesis, may become nearly as tense as it was originally; but generally it begins to shrink again before long, and then gradually undergoes cure. The operation does not generally need to be repeated. Another method of treatment has been recommended by Dr. Althaus, and successfully practised by Dr. Fagge and Mr. Durham. The details are furnished by Dr. Fagge in the following words:—'Two electrolytic needles are passed into the tumour one or two inches apart, they are then attached to two metallic wires, both connected with the negative pole of a battery of ten cells. A moistened sponge forms the termination of the positive pole, and is placed on the patient's skin at a little distance from the point of entrance of the needles. Its position is changed from time to time during the operation. The current is allowed to pass for about ten minutes. At the end of this time the needles are gently withdrawn and the seats of puncture covered with adhesive plaister.' The above operation is often attended with some escape of fluid into the abdominal cavity, and some rise of temperature with other febrile symptoms. And as with simple paracentesis, so here, the immediate effects are not always obvious. The operation may need to be repeated. It has been recommended by some that after the evacuation of more or less of the contents of the cyst, a solution of iodine, perchloride of iron, bile or some other antiseptic or parasiticidal fluid should be injected; and this practice has in some cases been successful. It is obvious, however, that the injection of irritating fluids is apt to induce inflammation and suppuration, which are in themselves very undesirable; and it is at least doubtful whether the death of the parasite is more surely attained by this procedure than it is by the simple evacuation of the fluid contents. If unfortunately peritoneal inflammation ensue, it must be combated by appropriate treatment. If suppuration of the cyst take place (and this is an accident for which we must be prepared), it will also be necessary to accommodate our treatment to the altered condition of things. But especially the local treatment will need some modification. It will then at all events be desirable, so soon as we are satisfied that the cyst is adherent, that a free opening be made, and the contents, inclusive of the hydatid cysts, freely evacuated. Whether, however, that opening should be

made with the trocar and cannula or the knife; or whether it should be allowed to close or be kept open; and in the latter case whether the contents should be allowed to escape by means of a drainage tube or not; or whether the cavity should be washed out with some disinfectant solution; are points on which it is difficult to express oneself absolutely. The exigencies of cases as they arise necessarily call for modifications in the details of treatment. It is needless to discuss the treatment of the numerous other accidents and complications which are apt to manifest themselves during the course of hydatid disease.

VIII. FATTY LIVER.

Causation.—The deposition of fat globules in the hepatic cells is not necessarily an indication of disease. It is frequently observed to a small extent in health; and sometimes indeed to a large extent in healthy persons who lead sedentary lives, or feed largely, especially those whose diet comprises an excess of fatty matter, or who have a tendency to obesity. That abundant deposition of fat, however, which constitutes what is meant by 'fatty liver,' is usually associated with various morbid states either of the system or of the liver itself. Among the former of these we may enumerate chronic alcoholism, heart disease, malignant cachexia, and especially pulmonary phthisis; among the latter cirrhosis, lardaceous degeneration, and the indurated condition which supervenes on chronic cardiac or pulmonary affections.

Morbid anatomy.—In the early stage fat globules of small size are found scattered in the substance of the hepatic cells; at a later period many of the globules have enlarged, partly by coalescence, partly by fresh deposition, and may then considerably exceed in size the nuclei around which they cluster; at a still later period complete coalescence takes place, and the cells distended with their oily contents assume very much the appearance of the cells of adipose tissue. The deposition of fat always commences at the periphery of the hepatic lobules, and is very often limited to that part; and even when the change becomes universal it is still this outer zone which chiefly suffers. The presence of fat in any abundance renders the affected portion of the liver coarse, soft, dull, and opaque—the yellowness due to bile and the redness due to blood alike disappearing in a greater or less degree. Further, the tissue often becomes distinctly greasy, the fat adhering to the knife and fingers. It often happens in cirrhosis that the isolated nodules of hepatic substance are more or less loaded with oil. In lardaceous change scattered patches of hepatic tissue are not unfrequently similarly affected. In cardiac and chronic lung disease the deposition is mostly limited to the peripheral parts of lobules; and indeed it is owing mainly to the contrast between the outer fatty and anæmic

zones and the central deeply congested areæ, that the term 'nutmeg' has been applied to this form of hepatic affection. It is not uncommon to find the fatty and the congested regions of the lobules separated from one another by a line of deep jaundice. In the fatty liver of phthisis and other wasting diseases, the fatty accumulation may still be mainly peripheral, and the liver may consequently present something of the nutmeg character; but not unfrequently the organ is pretty generally involved. Under these circumstances it presents a nearly uniform pallor, dulness of aspect, and softness, and its bulk is generally very largely increased. The enlargement of fatty liver is as nearly as possible uniform. The fat consists mainly of olein and margarin, with traces of cholesterine. Its amount varies; in extreme cases from 43 to 45 per cent. of the hepatic substance has been found to consist of fat, and indeed after removal of the water Frerichs has found no less than 78 per cent. of the residue to be fat.

Symptoms.—It is natural to believe that excessive accumulation of fat in the liver would seriously affect the functions of that organ; and many different symptoms have been ascribed to it. We are bound, however, to confess that we have never met with a case in which hepatic or other derangement has been clearly attributable to it. And, indeed, it must not be forgotten that fatty accumulation is frequently associated with structural changes in the liver; and that when under such circumstances hepatic symptoms are present, they are probably referrible to these associated lesions. The enlargement due to fatty deposition in the liver may often be recognised during life, and occasionally the augmented bulk of the organ produces fulness, weight, and uneasiness in the side.

Treatment.—When fatty liver depends on actual disease, it is essentially by treating the disease that we must hope to remove the hepatic accumulation. When we have reason to believe that enlargement of the liver, in persons who are fairly healthy, is due to fatty deposit, our treatment must be guided by our knowledge of their habits and tendencies, and must necessarily be mainly hygienic. It is very seldom, however, that we shall be called upon to make fatty liver a distinct object of medical treatment.

IX. LARDACEOUS LIVER.

Causation.—This affection is secondary to those morbid conditions of the system in which general lardaceous disease takes its origin: especially chronic phthisis, tertiary syphilis, caries of bone, and other conditions attended with prolonged suppuration.

Morbid anatomy.—The lardaceous change takes place first, according to Rindfleisch, in what he terms the arterial zone of the hepatic

lobules, that is, midway between the centre and periphery, implicating both the minute arteries and capillaries of the part, and the hepatic cells. But soon the morbid process extends to the central portions of the lobules, and after a time the periphery becomes equally involved. The change is attended: with great thickening of the affected vessels, and the acquisition by them of a peculiar homogeneous pellucid character; and with considerable enlargement of the hepatic cells, which lose all trace of granules, bile-pigment and nucleus, and become irregular or botryoidal vitreous-looking lumps which after a while break down into irregular fragments. The lardaceous liver, like the fatty, undergoes uniform enlargement in all its dimensions. It becomes smooth, heavy, and of somewhat doughy consistence; and if uniformly affected, presents a remarkably homogeneous sectional surface, of a greyish tint, with a peculiar glistening, or rather, perhaps, semi-translucent aspect, which has some resemblance to that of bees'-wax. It is equally free from biliary and vascular congestion, and from moisture. The lardaceous change is not unfrequently associated with more or less fatty deposit, sometimes with cirrhosis, sometimes with syphilitic disease.

The size which the lardaceous liver may attain is almost unlimited. It has been met with weighing between ten and fifteen pounds. This increase of bulk is, however, a slow process, and often extends over some years.

Symptoms.—The circumstances which in combination justify the diagnosis of this affection are the slow but continuous uniform enlargement of the liver, without pain or obvious hepatic symptoms; the long continuance of some one of those morbid conditions which we know to be conducive to lardaceous degeneration; and the coetaneous enlargement of the spleen, and involvement of the kidneys. There is no doubt that patients with lardaceous liver manifest, as a rule, marked cachectic symptoms; but there is little evidence to show that these are dependent in any peculiar degree upon the hepatic disease. It is true that a slight icteroid tinge occasionally manifests itself after a while, and that the bile in the gall-bladder and ducts is usually pale and watery; but, on the other hand, there is never obvious pain in the region of the liver, never deep jaundice, rarely if ever ascites, and (beyond the occasional presence of bile-pigment in the urine) nothing in that secretion distinctly to indicate impairment of hepatic function. The greater number of cases in which lardaceous disease manifests itself no doubt end fatally; but there is reason to believe with Frerichs that, if the change be not far advanced, the arrest of the morbid process upon which it is dependent may be followed by the restoration of the lardaceous organs to the condition of health.

The *treatment* of lardaceous degeneration merges in the treatment of the disease which produces it.

X. GALL-STONES.

Very little of practical importance is as yet known with respect to the variations in quality and quantity of the bile, and the influence of these variations on the action of the bowels, the assimilation of alimentary matters, and the general health. We know, no doubt, that when the bile which enters the duodenum is deficient in quantity, fatty matters are imperfectly assimilated, the evacuations are fetid, and the bowels usually constipated; and we have reason to believe that when there is an excessive discharge of bile, bilious diarrhœa and vomiting may be excited; but, on the other hand, we know that in many diseases, whether of the liver itself or of the general organism, the bile is found post mortem deviating widely from its normal condition, and yet there have been no symptoms during life which could be distinctly referred to this deviation. There is one abnormal condition of the bile, however, of great practical interest, which reveals itself to us, not directly by any of the consequences just enumerated, but by the formation of concretions which bring with them special symptoms and special dangers.

Causation.—The origin of gall-stones is obscure. It is easy, of course, to understand their increase of size by the accretion of additional solid matter; but it is not generally easy to determine the cause of the first step in their development, namely, the formation of a nucleus. In some rare cases this has been found to be a fragment of a needle, a dead entozoon, a small blood-clot, or (according to Dr. Thudichum) portions of the epithelial lining of the gall-ducts. In the majority of cases, however, it consists of a mass of concreted biliary colouring matter. Concentration and stagnation of bile have doubtless some influence over the production of gall-stones, as is shown by their much more frequent formation in the gall-bladder than in the hepatic ducts, and probably also by their comparative frequency in cases of carcinoma, and other organic diseases of the liver. It is not clear that the tendency to biliary calculi is inherited, or that it is ever traceable to any dyscrasia, notwithstanding the statements which are made to the effect that it is generally associated with gout, renal calculi, or other maladies. On the other hand, we know that gall-stones occur much more frequently in women than in men, and rarely in either sex below the age of thirty. They are occasionally met with, however, at earlier periods of life, and even in infancy. There is reason also to believe that they specially affect persons of sedentary habits. The influence of diet is unknown.

Morbid anatomy.—Gall-stones vary in size from mere granules up to masses moulded to the form of the gall-bladder, and measuring three or four inches in length, from one to one and a half inches in thickness,

and weighing between one and two ounces. When they are minute (less in size, say, than a poppy-seed), they are usually spoken of as biliary gravel. Gall-stones may be solitary; but they are much more frequently multiple, and, indeed, many have been found at one and the same time scattered throughout the biliary ducts, and several hundreds in the gall-bladder. When occupying the latter cavity their size has necessarily some relation to their number; at all events, when they are very numerous, they cannot possibly be large; whereas solitary calculi, and calculi occurring in groups of two or three, often attain considerable dimensions. The forms which they assume depend mainly on their relations, during growth, to the surrounding parts. In the commencement they may be rounded or amorphous accumulations of biliary colouring matter, or even rhomboidal tablets of cholesterine. But with increase of size some modification takes place. They may acquire a branched or coral-like form in the smaller bile-ducts; in the larger ducts or in the gall-bladder they may either form roundish masses, or accommodate their general shape to that of the cavity which contains them; but when, in the gall-bladder, the simultaneous development of many calculi takes place, they mutually interfere with each other's growth, and instead of assuming a globular form, become polyhedral or faceted, or flattened one against the other. In this manner the bladder may get uniformly distended with a pyriform mass of closely-packed, mutually-fitting gall-stones; and, indeed, it generally happens that, when its cavity appears to be occupied by a single large calculus, this consists of at least two or three, and generally more, well-articulated but distinct masses.

Gall-stones are usually smooth, but sometimes granular or tuberculated, and vary in colour from milk-white, through yellow or brown, to deep reddish or greenish-black. Their specific gravity ranges between ·8 and 1·15; they are as a rule, however, heavier than water, and sink in it, excepting when they have undergone desiccation. In some cases they are so soft and friable as readily to fall to powder between the finger and thumb; and generally they are sufficiently soft to admit of being readily crushed into irregular fragments, or of being cut with a knife. They are usually soapy or greasy to the touch. As to their general structure, they sometimes consist of a simple tuberculated accumulation of pigmentary matter, sometimes of a nearly homogeneous waxy mass. In most cases, however, three regions may be more or less obviously recognised: namely, a central nucleus, which, as has already been stated, is mostly pigmented and often irregular in form and shrunken; a zone of variable thickness around this, which is more or less homogeneous in texture, but marked with radial lines; and a cortical lamina, also of variable thickness, which is usually concentrically striated. These several regions are further characterised by differences of colour.

The chief constituent of gall-stones is cholesterine, and this forms

on the average from 70 to 80 per cent. of the entire mass; but in addition to this, biliary colouring matter, biliary acids, and lime are found in various proportions. Other ingredients are so rare or so small in quantity as scarcely, from a clinical point of view, to be worth consideration. They are chiefly the fatty acids, uric acid, earthy phosphates, alkaline salts, and mucus. Calculi consist sometimes almost entirely of pigmentary matter, sometimes mainly of carbonate of lime with some admixture of phosphate, and sometimes of pure cholesterine. Moreover, the different laminæ often differ in composition, the outer shell of large calculi frequently presenting an excess of earthy salts.

The consequences of biliary calculi are various. In many cases they form in the gall-bladder, and slowly grow there until, moulded to its shape, they entirely fill it; the gall-bladder contracts upon them, ceases to perform its proper functions, and becomes merely the capsule of what then probably proves to be an inert mass. Sometimes the presence of these bodies irritates the mucous membrane of the bladder into inflammation, and, it may be, into suppuration and ulceration. Slight attacks of inflammation doubtless arise occasionally and subside again without further result. But when the inflammation is of a more intense character, the cavity of the bladder may be converted into an abscess which discharges itself either *per vias naturales*, or by some abnormal channel; or the mucous surface of the bladder may at some point or other be fretted by its contained calculi into an ulcer which, gradually eating its way through the parietes (then probably glued to some neighbouring part) forms a sinus or diverticulum which, like the abscess, may open in one of several directions. The most common routes are externally through the abdominal walls, into the duodenum, and into the transverse colon. But the opening may also take place into the stomach, peritoneum, pleura, or lung. In many cases a gall-stone becomes dislodged, and slips into the cystic duct, whence it may pass slowly onwards until it reaches the duodenum. The duration of this process is very variable; in some cases it is over in a few hours, more frequently it occupies several days. The stone generally travels by fits and starts, and may be either temporarily or permanently arrested in any part of the channel along which it passes. If arrested in the cystic duct, it probably leads to its complete closure and to the enforced disuse of the gall-bladder, which may then either shrivel away or dilate into a mucous or serous cyst; if arrested in the common duct, it probably sooner or later obstructs the flow of bile, which then accumulates in the gall-bladder and ducts ramifying in the liver, and distends them. Further, in either of these situations, the presence of the stone may fret the surface against which it lies, and cause ulceration and possibly perforation, and thus lead to the formation of a local abscess, or to general peritonitis, or to some abnormal communication with the duodenum, colon, or portal vein. When once a gall-stone has descended from the gall-bladder, other stones, if they exist,

are apt to follow; and moreover, their passage is generally more readily and speedily effected than that of their pioneer.

Symptoms and progress.—The presence of gall-stones in the bladder or hepatic ducts does not necessarily cause symptoms, and in a large number of cases is from first to last unattended with symptoms. Gall-stones may, however, occasionally be recognised, in consequence of forming an irregular, hard, and sometimes crepitating lump in the situation of the gall-bladder. When their presence excites inflammation, we may look for tenderness, pain, and fulness in the same neighbourhood, with more or less obvious febrile disturbance. But unless any more distinctive phenomena arise, the exact nature of the affection can scarcely be diagnosed positively. Such phenomena are: the formation of an abscess superficial to the bladder in the abdominal parietes, and the ultimate escape of gall-stones with the other contents of the abscess; and the discharge of gall-stones through an ulcerated opening into the duodenum or colon, and their escape with the fæces, or their arrest in the small intestine, followed by enteritic symptoms. It must not be forgotten, however, that each of these phenomena may arise without having been preceded by any clear symptoms of inflammation of the gall-bladder.

The symptoms most characteristic of the presence of gall-stones are those which depend on their dislodgement and subsequent passage along the cystic and common ducts. They resemble in many important respects those due to the transit of a renal calculus along the ureter, and are mainly: more or less severe pain, coming on suddenly, and lasting with irregular intermissions and exacerbations a few hours or several days; faintness, nausea, and vomiting; and the consequences of impediment to the escape of bile into the intestines. The pain (frequently termed hepatic colic) varies in its intensity, situation, and quality. Sometimes it is comparatively slight, sometimes so severe that the patient writhes and cries out with agony; its character is aching, cutting, tearing, or burning, and it is generally attended with an unbearable sense of tightness, constriction, or cramp. It is usually referred to the pit of the stomach or to the umbilicus, whence it extends to the back between the shoulders, to the chest or to the shoulder-tip, or down into the lower part of the abdomen. But its situation is often somewhat indefinite, and may be such as to simulate the passage of a stone along the ureter. There is seldom any material tenderness, and pressure sometimes affords relief to the pain. Hepatic colic is said to be further characterised by often coming on suddenly two or three hours after a meal, at the time when the passage of food along the duodenum excites the flow of bile from the gall-bladder and biliary passages. It often comes to a sudden end in consequence either of the slipping back of the stone into the gall-bladder, of its arrest at some point in the course of the cystic or common duct, or of its escape into the bowel. The faintness, nausea, and vomiting are not in necessary

relation with the severity of the pain; the patient may be simply chilly, or he may have severe rigors; he may merely feel faint, or he may fall into a state of actual syncope or collapse, with cold and pallid surface, profuse perspirations, and imperceptible pulse; he may complain simply of nausea, or he may suffer from severe and protracted vomiting. The syncopic attack has proved fatal. A gall-stone may pass on from the bladder to the duodenum with all the above symptoms, and yet cause no material stoppage of bile. In a large number of cases, however, its presence in the common duct is followed by more or less complete retention, which reveals itself by the vomit (if it continue) ceasing to be bilious, by the stools acquiring a pale clay colour, by the urine in from twelve to twenty-four hours becoming tinged with bile, and by the development a little later of general jaundice. The supervention of jaundice, after such symptoms as have been detailed, is almost pathognomonic of the passage of a biliary calculus, or at all events of a foreign body, along the common duct. The diagnosis cannot, however, be regarded as positive, unless the calculus be discharged per anum. And hence, in all cases of suspected hepatic colic, it is important to examine the fæces carefully from day to day. This should be done by diluting them with water, and passing them through a sieve with sufficiently small meshes to retain any small solid bodies which may be present in it. If the pain and other symptoms continue for some little time, more or less inflammation is likely to arise at the seat of disease; and tenderness and fulness may then come on, together with more or less febrile disturbance. And even after the escape of a calculus, such pain and fever, and even jaundice, may continue for some little time. The passage of one biliary calculus is often, if not generally, succeeded at irregular intervals by the passage of others—the later attacks, however, being as a rule both milder and of shorter duration than the first. This repetition of similar attacks is a further indication of the nature of the patient's malady. It may be added that the passage of biliary gravel, which has been sometimes discovered in the fæces in large quantities, and inflammation of the neck of the gall-bladder, may present many of the symptoms which attend the passage of calculi.

The consequences of the arrest of gall-stones in the small intestine have been described under the head of intestinal obstruction; those of their long-continued or permanent retention in the common duct will be considered under that of obstruction of the hepatic ducts.

Treatment.—The general treatment of gall-stones is very unsatisfactory; we can neither dissolve them nor remove them; nor if they have once formed can we prevent them from becoming larger. And even as regards prophylaxis, all that can be said is that those whom we believe liable to them should eschew all such habits as seem likely to engender them. They should live wholesomely and abstemiously, and take a sufficiency of exercise daily. The habitual use of alkaline

waters has been recommended, but the evidence in favour of their virtues is altogether valueless. For the paroxysm of hepatic colic, our main reliance must be placed upon morphia or opium, given in sufficiently large doses, and sufficiently frequently, either by the mouth or hypodermically, to relieve the patient's sufferings. Belladonna has also been largely recommended, mainly with the object of relaxing spasm, and so aiding the onward passage of the stone; but it is certainly not so beneficial in its effects as opium. The inhalation of chloroform, short of producing insensibility, often affords signal relief. To assuage the vomiting, Dr. Prout long ago recommended the use of copious draughts of warm water, containing from one to two drachms of carbonate of soda to the pint. This practice is still largely followed, and believed to be efficacious. In addition to these remedial measures, the warm bath, hot fomentations to the epigastrium, and counter-irritants may generally be employed with advantage.

XI. OBSTRUCTION OF THE HEPATIC DUCTS.

Causation. Obstruction of the hepatic ducts is an incident of frequent occurrence of more or less importance, in a large number of the morbid conditions of the liver which have already been discussed; it is also the most frequent cause of long-continued and intense jaundice, if not actually the most frequent cause of jaundice; and on these grounds demands some special consideration. The causes of obstruction are, in some cases, inflammatory thickening of the mucous membrane of the ducts, or accumulation of inspissated mucus or other kinds of inflammatory exudation; in some the presence of stricture; in some the growth of polypoid tumours; in some the impaction of calculi or other foreign bodies. In other cases they are to be sought in inflammatory infiltration of the tissue of the lesser omentum or of Glisson's capsule, or in the development in these situations of syphilitic, carcinomatous, or other growths involving or compressing the ducts. Further, tumours springing from the stomach, pancreas, or neighbouring parts, and aneurysms may press upon the common duct and obstruct its channel.

Morbid anatomy.—Obstruction may take place in any of the ducts at any part of their course; and the effects on the ducts behind the impediment, and on the liver-substance with which they are in relation, will be the same in kind wherever the obstruction is situated; the bile becomes arrested in its flow and altered in character, the implicated ducts undergo dilatation and other changes, and the liver-cells whose products they receive become jaundiced, fatty, and sometimes disintegrated.

If complete obstruction take place in the common duct, the dilatation of ducts which ensues is almost universal; the common duct not unfrequently attains the size of the duodenum, and the ducts ramifying throughout the liver acquire proportionably large dimensions. The condition of the gall-bladder under such circumstances varies; sometimes it shrinks or shrivels up, sometimes it retains pretty nearly its normal bulk, sometimes it becomes, like the rest of the excretory apparatus, enormously distended. The consequences of obstruction, as respects the biliary fluid, are that it generally gets thin and watery, and at the same time of a dark green or brown colour; but it may also become turbid from admixture with mucus or pus; sabulous from the deposition of solid matter—pigment, or cholesterine; grumous from containing blood; or, when the bile ceases to form or to flow, transparent, colourless, and viscid. The last kind of fluid may be met with in the gall-bladder when, after closure of the cystic duct, it dilates (as occasionally happens) into a mucous cyst. The consequences, as regards the walls of the ducts, are also very various. In most cases they become thickened; but in some they become attenuated; in some inflammation with excess or modification of secretion takes place, in some ulcerative destruction. In the last case, perforation of the common duct may occur, with the development of an abscess in its vicinity, or rupture into the peritoneum; or more or less general destruction of the walls of the bile-ducts may ensue with the formation in their place of irregular biliary channels, bounded by the eroded hepatic tissue, and communicating, it may be, with branches of the portal vein. Such channels may be converted into branching abscesses. The effects of obstruction, on the liver generally, are, in the first instance, gradual and uniform increase of bulk, which may be maintained for several months; and then gradual atrophy, the organ however not so much shrinking in all its dimensions as becoming wrinkled, thin, and flabby in consistence. The hepatic texture becomes soft, loose, and œdematous (yielding on pressure a considerable quantity of thin greenish fluid) and jaundiced, or before long of a dark greenish hue. On microscopic examination, the hepatic cells are usually found more or less deeply bile-stained, and often containing granular pigment and oil-globules. In some cases the cells after a time undergo degeneration; and all that remains of the hepatic texture may then be the framework of connective tissue, vessels, and the like, together with a greater or less abundance of free oil-globules, granules of precipitated pigment, and cell-nuclei. The tissues moreover usually yield an abundance of leucine and tyrosine.

It has been assumed throughout the foregoing account that the obstruction is complete and permanent. It need scarcely be added that obstructions are often merely temporary, that whether temporary or permanent they are not unfrequently incomplete, and that under either of these circumstances there will be more or less important

modification in the progress and consequences of the secondary pathological lesions.

Symptoms and progress.—It is always important, for the sake both of prognosis and of treatment, but often quite impossible, to determine the exact cause of obstructive jaundice. Our diagnosis in each case must rest on a careful consideration of its history and progress and on a close investigation of the phenomena which come under our immediate observation. It is not, however, so much with this subject that we have now to deal as with the special symptomatic consequences of obstruction. These, which have already been pretty fully considered, may be divided mainly into those dependent on absence of bile from the alvine evacuations, those due directly to the changes going on in the liver, and those arising from the accumulation of bile and effete matters in the blood.

The consequences of the absence of bile from the bowels have been sufficiently discussed.

Alteration in the bulk of the liver is a sign of considerable value. Its primary enlargement is indicated on the one hand by the gradual rise of the hepatic dulness into the chest, on the other hand by the gradual emergence of its lower edge from under the ribs and its extension for two or three inches below its normal level. If the gall-bladder also undergo distension, it may generally be readily recognised as an elastic or fluctuating swelling coming out from beneath its accustomed notch. In rare cases the distended common duct has itself been felt as a fluctuating tumour. When the later atrophic changes set in the enlargement of the liver ceases, and the organ undergoes slow diminution in bulk; but this change reveals itself less by general shrinking than by diminution of thickness—the free edge often becoming peculiarly thin, so that, if the abdominal walls be flaccid and spare, it may often be readily grasped between the finger and thumb. Some degree of fulness, weight, tenderness, or pain is not unfrequent in the situation of the liver, during the progress of its enlargement; especially, of course, if inflammatory changes supervene.

The jaundice of complete obstruction is generally very intense. It first reveals itself by the presence of bile-pigment in the urine at the end of from twelve to thirty-six hours after bile has ceased to flow into the bowels. Yellowness of the conjunctivæ and skin usually supervenes in the course of the third day. If the obstruction continue, the intensity of the jaundice rapidly increases, and after a time tends to assume a greenish or brownish tint. The colour is liable to variations of intensity even when no discharge of bile into the bowels takes place, and by no means necessarily increases with the duration of the case; indeed, it not unfrequently happens that it undergoes manifest diminution during the later periods of the disease.

It is chiefly in jaundice from obstruction that we may look for the

occurrence of many of those additional phenomena which have already been adverted to, such as yellow vision, itching, cutaneous eruptions, and petechial and other forms of hemorrhage; and it is with this alone that xanthoma has any connection. As a rule, there is no elevation of temperature; and there is no necessary affection of the tongue or loss of appetite.

It is almost needless to say that, in those cases in which the obstruction is temporary only, in those in which the obstruction of the main duct is, and remains incomplete, and in those in which (as in hypertrophic cirrhosis) the impediment to the escape of bile involves some of the minuter tubes only, the symptoms will vary more or less widely from those which have just been detailed; especially the evacuations will probably still contain bile, the liver will undergo little or no enlargement, the jaundice will be slight, and the other symptoms which associate themselves with these conditions will be developed slightly or late, or not at all.

The duration of life in cases of jaundice with complete obstruction varies a good deal. In some cases the patient dies in the course of a few weeks; in some he survives for periods varying between six and twelve months; while occasionally life is prolonged to two, three, or more years. The causes of death also are various. Sometimes death is due to rupture of the hepatic or common duct, or of the gall-bladder, with consequent peritonitis; sometimes to the supervention of hepatic inflammation with suppuration and some one or other of their results; sometimes to intestinal or other hemorrhage; sometimes to so-called 'biliary toxæmia;' most frequently, however, it results from gradually increasing emaciation and debility. Further, patients enfeebled by this disease are very apt to be attacked with pneumonia, dysentery, dropsy, or other complications, and to be thus carried off. In some cases recovery takes place even after complete obstruction has lasted for a considerable length of time; the indications of this event being the reappearance of bile in the fæces, the gradual disappearance of pigment from the skin and urine, and in association therewith general improvement in the patient's health.

Treatment.—In the treatment of jaundice from obstruction our first object should of course be to remove the mechanical obstacle to the escape of bile from the liver. It need scarcely be said, however, that this can never be effected but by indirect measures, and in a large proportion of cases never effected at all. But in reference to this subject we must refer the reader to those articles which deal with the various conditions to which obstruction may be due. The question we have here specially to consider is—How shall the jaundice and the consequences it entails be best treated? Unfortunately, we can do little, and that little is mainly hygienic. The patient's bowels should be regulated if necessary by mild laxatives; the functions of the kidneys and of the skin (by which emunctories bile is now almost solely eliminated)

should be promoted by the use of diluents, diuretics, warm clothing, and warm baths with rubbing or shampooing; his appetite should be sustained and his gastric digestion improved, if need be, by vegetable tonics or stomachics, with which the carbonates of the alkalies may often be beneficially combined; his general health should be maintained, partly by the exhibition of vegetable tonics and iron, partly by the habitual use of nutritious unstimulating food from which fatty matters and alcohol are as far as possible excluded, partly by attention to hygienic conditions, more especially to warm clothing, the avoidance of chills or sudden vicissitudes of temperature, change of scene, moderate exercise, and early hours. Of particular remedies it may be observed that Frerichs recommends lemon-juice as a valuable diuretic in these cases, and that Dr. G. Harley advocates the use of inspissated ox-gall in gelatine capsules, to be given in doses of from five to ten grains two or three hours after each meal. It need scarcely be added that when complications arise—gastric catarrh, diarrhœa, hemorrhage, or head-symptoms—they will probably need each its appropriate treatment. In those cases in which the gall-bladder becomes excessively distended, the question as to the propriety of puncturing it may arise. The operation is obviously one not to be lightly entertained, or to be performed without the most ample precautions.

XII. JAUNDICE WITHOUT OBVIOUS OBSTRUCTION OF DUCTS.

Causation.—The varieties of jaundice here referred to are more particularly those which occur in the specific febrile disorders, such as yellow fever, ague, relapsing fever, and pyæmia. It is possible, too, that under the same head must be included the jaundice which occasionally attends pneumonia, rheumatism, snake-bites, phosphorus-poisoning, and those other morbid conditions of the liver in which the secreting cells are directly involved. The jaundice which is said to arise under the influence of strong mental disturbance, and that of new-born babes, may possibly also be placed in the same class. It must be remarked, however, that there is still considerable uncertainty in respect of the intimate pathology of the jaundice attending these various affections; it is very probable that obstruction of the smaller ducts, or, as Virchow holds, catarrhal obstruction of the intestinal portion of the common duct, may eventually be proved to be the cause of jaundice in some of them; there seems little doubt that in others it is actually due to changes going on in the colouring matter of the blood; and it is possible that in some of them it may be the consequence, as Frerichs holds, of an abnormal diffusion of bile, arising in some alteration in the supply of blood to the liver, and defective metamor-

phosis or consumption of bile in the blood; and in some, as Dr. Murchison believes, of excessive reabsorption of bile with or without excessive secretion.

Morbid anatomy.—In most of the cases here referred to the liver is found post mortem to be pale and anæmic, and soft or flabby, and the hepatic cells either quite normal, or, as especially in phosphorus-poisoning, unusually granular or studded with droplets of oil; in some the generally pallid tissue presents patches of still more marked pallor, which are often separated from the surrounding parts by wide but irregular zones of slight congestion. The appearances, as a rule, are certainly not very striking, and scarcely indicative of serious hepatic disease.

Symptoms.—The jaundice is almost without exception very slight; it creeps on gradually; it does not attain any intensity in the skin; and the pigment passed with the urine is in small quantity. Moreover, the motions almost always still contain bile. There is no doubt that in many of these cases the symptoms which the patient presents are extremely grave. Yet there is no good reason to believe that as a rule they are due in any important degree to the hepatic disorder; for while the grave symptoms are usually such as characterise the disease which the jaundice complicates, those cases in which jaundice appears are not generally more serious than those from which it is absent, and the jaundice does not as a rule bring with it any specific symptoms.

Treatment.—The forms of jaundice now under consideration seldom call for special treatment. Their presence, however, may furnish a hint as to the desirability of employing laxatives, and promoting the action of the skin and kidneys.

XIII. MALIGNANT JAUNDICE. (*Yellow Atrophy of the Liver.*)

Definition.—There is one form of disease in which jaundice is associated with a remarkable group of symptoms, which for convenience, if not on other grounds, may be separated from the cases which have just been considered; it is that which is sometimes termed malignant jaundice, and to which Rokitansky has given the name of 'yellow atrophy of the liver.'

Causation.—Cases of malignant jaundice have been observed chiefly, if not solely, in adults, and in women far more frequently than men. Moreover, in a large proportion of cases, the patient has been attacked during pregnancy. It is also a remarkable fact that the onset of the disease appears to have often been determined by some sudden and intense mental emotion. Among other assigned causes may be enumerated syphilis, typhus, and miasm.

Symptoms and progress.—Malignant jaundice frequently comes on without premonitory signs; but in a considerable number of cases it is preceded for a few days, or even for a few weeks, by slight gastro-intestinal catarrh, with which probably, sooner or later, some degree of jaundice is associated. Among the first symptoms which usually arise to indicate the gravity of the attack are vomiting, and especially the vomiting of coffee-ground fluid due to gastric hemorrhage, intense headache, irritability, and restlessness. To these soon succeeds delirium, which is sometimes low and muttering, sometimes noisy, and frequently violent and maniacal. The patient's manner is agitated, there is generally more or less tremulousness of limbs, and in a large proportion of cases convulsions soon manifest themselves. These may vary in character; they may be general or local; and may present the features of simple rigors, or assume an epileptiform or tetanic form. After a short time, the condition of delirium or convulsion passes into one of quietness and stupor, which gradually deepens into profound coma, usually attended with dilated inactive pupils and stertorous breathing. But, besides the remarkable combination and sequence of symptoms here enumerated, other phenomena present themselves which are of considerable significance and importance in reference to diagnosis. The pulse during the earlier period of the disease, or that of excitement, is characterised by remarkable and sudden variations in frequency, but is generally abnormally quick; with the supervention of coma, however, it gets more uniformly rapid, and at the same time more and more feeble, until probably it can be no longer felt at the wrist. The tongue soon becomes coated, and generally before long assumes the typhoid character; it gets dry and brown or black, and sordes accumulate upon the teeth. There is often some uneasiness and tenderness in the hepatic region; and in addition, it can often be determined by careful examination that there is a gradual diminution in the area of hepatic dulness. The bowels usually are confined; and the motions passed in the course of the disease present a gradual diminution, and at length, it may be, total absence, of biliary colour. The urine probably is secreted in normal quantity, and acid; but it becomes jaundiced in a greater or less degree, urea and phosphate of lime diminish, and sometimes wholly disappear, to be replaced by leucine, tyrosine, and extractive matters, which, when the urine cools, sometimes form a peculiar greenish-yellow sediment. The skin is usually cool and dry. The jaundice, which sometimes precedes, sometimes follows, and sometimes appears simultaneously with, the other initial symptoms of the disease, increases in depth with the duration of the malady, but rarely, if ever, attains any high degree of intensity. There are yet two other features of striking importance: the one is the total absence of febrile temperature; the other a general tendency to hemorrhage. The latter shows itself by hæmatemesis, by the appearance of petechiæ and bruise-like extravasations beneath the

skin, or by more or less profuse discharges of blood from the nose, bowels, or other mucous surfaces.

The most striking phenomena in the clinical aspect of malignant jaundice are, the combination of slight jaundice with grave cerebral disturbance, hemorrhage from and into various organs and tissues, profound change in the composition of the urine, absence of fever, and the almost invariably fatal issue of the disease. Death may occur within twelve or twenty-four hours, but generally supervenes between the second and fifth day, and is rarely delayed beyond a week.

Morbid anatomy.—In all typical cases of the disease the post mortem conditions are remarkable and characteristic. The most obvious change is manifested by the liver. This may be of natural size, but is usually shrunk to half or even one-third of its normal bulk—its surface being then wrinkled and flabby. On section it is found to be of a nearly uniform pale yellow colour, with little or no indication of the constituent lobules, or evidence of vascular injection. Frerichs says that in some cases the lobules are separated from one another by a dirty greyish-yellow substance. The bile-ducts and gall-bladder usually contain either colourless mucus, or a thin fluid only very slightly tinged with bile. On microscopic examination, the hepatic cells are found to have disappeared more or less completely—in some cases not one is discoverable; and in their place may be observed either simple granular matter, or this intermingled with oil-globules and precipitated bile-pigment. Leucine and tyrosine also may sometimes be recognised in the hepatic substance and hepatic veins. There is usually some enlargement of the spleen. The only other morbid phenomena of importance are: occasional fatty change of the glandular epithelium of the kidneys; extravasations of blood (usually petechial) beneath the surface of the peritoneum, pleura, and pericardium, in connection with the gastro-intestinal and other mucous membranes, and occasionally in the substance of the lungs, liver, spleen, and kidneys; and the presence in the blood (which does not as a rule display any change visible to the naked eye) of large quantities of leucine and urea.

What the nature of the malady under consideration may be, is by no means satisfactorily established. By some it is regarded as a primary disease of the liver. Frerichs, who (following Bright) takes this view, looks upon it as a parenchymatous inflammation of the organ, attended with little exudation, but with obstruction to the passage of blood through the vascular network at the periphery of the lobules, and consequent degeneration and death of the hepatic cells. According to this view, the jaundice and other characteristic symptoms of the disease are secondary to the hepatic lesion. Some, on the other hand, look upon the hepatic affection as the consequence of some general blood-disease, due either to the absorption of some noxious chemical substance, or to the presence of a poison of organic origin

having some affinity with those of the infectious fevers, or of pyæmia. In the latter point of view some of the graver symptoms would be referrible to the primary disease of which the hepatic disorder is a consequence; but others might still be attributable to the morbid condition of the liver. It would not be difficult to adduce plausible arguments either against or for either of these hypotheses. We may, however, point out that while, on the one hand, there is nothing in the clinical phenomena of these cases to indicate their inflammatory origin; there is, on the other hand, ample proof, from the occasional supervention of the symptoms of malignant jaundice in cases of occlusion of ducts, that extensive destruction of the secreting structure of the liver, with suppression of bile, is fully competent to induce all the phenomena of the disease under consideration. We must confess, indeed, that, while not quite committing ourselves to the inflammatory origin of the hepatic changes, we are disposed to regard the disease as primarily hepatic.

Treatment.—Nothing can well be less satisfactory than our knowledge in reference to the treatment of malignant jaundice. Active purgation has been recommended, especially in the early stage; it is difficult, however, to understand why. Again, those who look upon the disease as of inflammatory origin advocate the local abstraction of blood, and other antiphlogistic measures, during the inflammatory stage. But unfortunately this, if it exist at all, exists only during that preliminary period in which there is nothing to distinguish cases of malignant jaundice from cases of catarrhal affection of the biliary ducts. Considering that in this disease there is a large accumulation of effete matter in the blood, on which it seems probable that some of the grave symptoms are dependent, there are grounds for the employment of diuretic and diaphoretic measures. In the absence of more obvious indications, we must either do nothing, or treat the more prominent symptoms: that is, so far as we are able, check vomiting, arrest hemorrhage, overcome constipation, promote the action of the skin and kidneys, soothe during the stage of excitement, and during that of stupor and coma and failing strength employ counter-irritants and stimulants.

XIV. DISEASES OF THE PANCREAS.

A. *Introductory remarks.*—Very little of clinical value is known about the diseases of the pancreas. This is due: partly to the comparatively small size and deep situation of the gland; partly to the fact that its functions have much in common with those of the salivary and duodenal glands, and even with those of the liver itself; but chiefly perhaps because it is rarely affected excepting secondarily or in association with diseases of neighbouring organs.

In reference to the diagnosis of pancreatic disease, we must recollect: that this organ is situated in front of the aorta and behind the stomach, deep in the epigastric region, and on the level of the first lumbar vertebra; that any tumour which may be developed in it will be discoverable in this situation only (a situation however which may be equally affected by aneurysms of the aorta or cœliac axis, or by tumours involving the posterior wall of the stomach, or originating in the retro-peritoneal glands), and will probably be immovably fixed there; and that any pain and tenderness which may attend its lesions will probably be referred to the depth of the epigastric region and to the back, in the situation of the upper lumbar and lower dorsal vertebræ. We must also recollect that the function of the organ is to secrete a large quantity of fluid, which differs little from ordinary saliva in either its chemical composition or its office, and is an important agent in the emulsification of fat, in the conversion of starch into dextrine and sugar, and in the reduction of albuminous matters into a form favourable for assimilation. It may therefore be reasonably believed that the retention or suppression of the pancreatic fluid will be attended with more or less serious impairment of nutrition; and, if the food contain much starch or fat, with the unwonted appearance of starch or fat in the evacuations. The abundant discharge of fat by stool, indeed, has been not unfrequently noticed in cases in which the pancreas has been seriously diseased.

B. *Hyperæmia and inflammation.*—Of these conditions but little can be said. They are occasionally recognised post mortem, but for the most part in cases where no suspicion of pancreatic disease was entertained during life. *Abscesses* are sometimes discovered in the gland, and occasionally large abscesses; but they are usually small and of pyæmic origin. *Catarrhal inflammation* of the duct is probably not uncommon in connection with the same affection of the common hepatic duct, and may, like that, lead to temporary or even permanent obstruction. In chronic ulcer of the stomach the subjacent pancreas not unfrequently becomes implicated in the course of the extension of ulceration; and thus its eroded substance may after a time form the floor of the ulcer.

C. *Morbid growths.*—The pancreas is often the seat of such formations; but they are rarely, if ever, of primary origin within it. They are sometimes a consequence of the generalisation of malignant tumours, but are much more frequently due to extension of disease from the stomach, retro-peritoneal glands, or peritoneum. It is, however, in carcinoma of the pyloric extremity of the stomach that the pancreas most frequently becomes involved. Of the several varieties of malignant disease to which it is liable, scirrhus is the most common; but the encephaloid, colloid, and melanotic forms have each been met with.

D. *Calculi* are occasionally discovered in the pancreatic ducts, and

more especially in the principal duct. They have the same chemical and other characters as other salivary calculi—consisting mainly of phosphate of lime with some animal matter, and varying from minute granules up to the size of a filbert. When small they are sometimes present in vast numbers; when large they are usually solitary, and more or less completely obstruct the duct in which they lie.

E. *Obstruction of the pancreatic ducts.*—When these channels get blocked up, whether by calculi or stricture, or by their compression by, or involvement in, malignant or other growths, the ducts behind undergo gradual dilatation from the accumulation of secretion within them. The chief enlargement occurs in the principal duct, which becomes elongated and tortuous, irregular in form, and sometimes sufficiently dilated to admit the finger. The secondary ducts also become dilated, but in a less degree; and the whole organ consequently increases in bulk, and on section appears at first sight to be made up of a congeries of cysts—the secreting tissue between them being more or less atrophied. Solitary cysts, apparently due to the dilatation of obstructed ducts of small size, are occasionally discovered in glands which are in other respects healthy. Their only pathological importance arises from the fact that they may, from their size and situation, be readily mistaken for tumours or cysts of much more serious import. They may attain the size of an orange.

F. *Symptoms and treatment.*—It would be a waste of time to discuss the diagnosis of the various lesions which have just been passed in review; the special phenomena which must be looked for as indicative of pancreatic disease have already been sufficiently considered; and for the recognition of additional features special to each variety of lesion, the practitioner must be guided by his general knowledge of pathology and of the pathology of the pancreas. In the great majority of cases pancreatic disease will doubtless remain undetected during life.

It would be equally a waste of time to enter upon the discussion of the treatment of pancreatic affections.

CHAP. VI.—DISEASES OF THE GENITO-URINARY ORGANS.

SECTION I.—DISEASES OF THE KIDNEYS.

I. INTRODUCTORY REMARKS.

General Physiological and Pathological Considerations.

THE urinary organs comprise the kidneys, ureters, bladder, and urethra. The diseases of all these parts are of high interest to the physician; but those of the kidneys and ureters come more especially under his observation and treatment, and it is mainly to them, therefore, that attention will be directed in the following pages.

The sole function of the kidney is to separate from the blood, in association with water, a number of effete, waste, and surplus matters which are constantly being added to the blood from various sources. But the urine, as it escapes from the urethra, contains in greater or less proportion certain additional matters—mucus and the like—which are yielded to it by the various mucous surfaces over which it passes, and by the glandular organs which open upon them.

The urine, thus constituted, varies in composition within wide limits, even in health. In disease, where the nutritive and destructive processes are variously modified, and where the functional activity of important organs is in different degrees diminished, impaired, or exalted, the composition of this fluid undergoes still greater variations; and, indeed, there are some cases (as for example that of diabetes) in which, the kidneys remaining sound, the nature of the disease under which the patient labours is revealed almost solely by the peculiarities which the urine presents. But especially the composition of the urine is largely and importantly modified by diseases of the urinary organs, which tend on the one hand to impede the discharge from the blood of the proper urinary constituents, and on the other hand to add to the urine matters which are wholly foreign to its normal constitution. It is obvious, therefore, that the careful investigation of the urine may be expected to throw important light, not only on the varying processes connected with healthy nutrition, but also on the pathology of many of those morbid conditions in which the kidneys are not distinctly implicated, and above all on the nature of the diseases of the kidneys themselves and of the several organs in relation with them.

But again, when the urinary organs are the seat of disease, and oppose (as they then generally do) a more or less complete obstacle to

the elimination of urea and other such products from the blood, it is clear that this fluid must soon become surcharged with effete and presumably injurious matters of a specific kind, and that we must, therefore, expect specific morbid consequences sooner or later to ensue.

It is also clear that many diseases of these organs must be attended with both local and general indications and symptoms which are totally independent of the functional derangements which are associated with them—local phenomena, such as pain and tumour; general phenomena, such as inflammatory fever and some forms of cachexia.

The morbid phenomena, therefore, which are associated with, and result from, diseases of the kidneys may be properly and conveniently divided: first, into those which are special to these organs and depend directly on the impairment or perversion of their normal functions; and, secondly, into those which in a certain sense are common to these and other similarly affected constituent portions of the body.

In accordance with the foregoing observations, we propose to give a brief account: first, of the composition of the urine in health and disease; second, of the specific consequences of the retention of urea and other such matters in the blood; and, third, of the non-specific morbid phenomena which attend and characterise lesions of the urinary organs.

A. *Characters and Composition of the Urine.*

The urine is a transparent, limpid, straw- or amber-coloured fluid, of saline taste, and for the most part of acid reaction, which deposits, on standing, a filmy cloud of mucus, and occasionally an opaque, reddish, powdery sediment. Its acidity increases for a few days with exposure to the air, and at the same time urates, uric acid, and oxalates are deposited. Then it undergoes putrefaction, becomes alkaline and ammoniacal; earthy matters, including crystals of triple phosphate, fall; and bacteria and torulæ make their appearance in it.

The quantity passed in twenty-four hours fluctuates within wide limits: it may, however, be reckoned usually at between two and three pints in the adult, but may range from one to four pints. The specific gravity also presents a wide range: it commonly lies between 1015 and 1025, but temporarily may fall to 1005 or less, or rise to upwards of 1030. The acidity which, when the urine is emitted from the bladder, is an almost unfailing characteristic of it, is liable to a good deal of variation of intensity; and, indeed, as Dr. W. Roberts shows, that which is secreted an hour or two after meals is generally alkaline, although its alkalinity is commonly masked by its admixture in the bladder with acid urine already there or subsequently added to it. The acidity depends mainly on the presence of acid phosphates and urates, and in some degree also on traces of lactic, oxalic, and other acids.

The degree in which the specific gravity of urine exceeds that of distilled water depends on the solid matters—the special urinary con-

stituents—which are contained in it. The proportion which these hold to the watery constituent may be approximately estimated, according to Trapp's formula, by doubling the last two figures of the number which indicates the specific gravity. Thus, 1000 parts of urine with a specific gravity of 1015 contain 30 parts of solids; and 1000 parts of urine with a specific gravity of 1025 contain 50. Hence the amount of solid matter in healthy urine usually varies from three to five per cent. It is generally, however, far more important to know the actual amount of solid matter that is passed daily than the ratio which the solid matter holds to the very variable quantity of water with which it is mixed. This knowledge can be gained by collecting and mixing all the urine passed in the course of twenty-four hours, and then examining quantitatively a measured portion of its bulk, or, more readily though less accurately, by the method above given.

The solid matters of the urine are very numerous; and they vary largely, both in the relative proportions in which they are excreted and in their aggregate amount. The urea especially is remarkably modified by age, sex, diet, and other circumstances, so that the amount which may be taken as the average may be halved or doubled independently of any impairment of health. The following table is designed to show at a glance the relative proportions of the chief constituents contained in an average specimen of the urine of an adult, and the total quantities of each which might in such a case be discharged in twenty-four hours. The specific gravity is assumed to be 1025, and the temperature 32°:—

Urinary constituents.		Percentage composition.		Daily aggregate in grains.	
Water			95·000		19000·0
Urea	CH_4N_2O	2·500		500·0	
Uric acid	$C_5H_4N_4O_3$	·042		8·5	
Kreatinine	$C_4H_7N_3O$	·075		15·0	
Hippuric acid	$HC_9H_8NO_3$	·075		15·0	
Pigment, mucus, odorous matters, xanthine, &c.	Extractive	·600		120·0	
Total organic matters		——	3·292	——	658·5
Chlorine		·500		100·0	
Sulphuric acid		·150		30·0	
Phosphoric acid		·250		50·0	
Potash		·175		35·0	
Soda		·600		120·0	
Lime		·018		3·5	
Magnesia		·015		3·0	
Total fixed salts		——	1·708	——	341·5
General total			100·000		20000·0 or 45⅓ oz.

and starvation. Urea is a feeble base, and exceedingly soluble; and has, therefore, under ordinary circumstances, no visible influence over the condition of the urine. It forms no sediment, and cannot be detected in it except by chemical processes. Under the influence of the mucus of the bladder, and some other circumstances, it is readily converted, with the aid of water, into carbonate of ammonia. Urea crystallises in white silky needles, or transparent four-sided prisms, the ends of which are often formed by one or two inclined planes. Such crystals may often be obtained by evaporating a drop of urine—especially febrile urine—on a glass slide. If an excess of colourless nitric acid be added to urine concentrated by evaporation, the mixture will become almost solid with crystals of nitrate of urea. These occur in rhombic prisms or plates which are colourless, and have a silky lustre. For the quantitative determination of urea various methods have been employed. The following are probably the best:—

a.[1] With a solution of mercuric nitrate, urea forms a white gelatinous precipitate, containing one equivalent of urea to four equivalents of mercuric oxide. The determination of urea by Liebig's method is based on this reaction. A standard solution of mercuric nitrate is prepared of such a strength that 10 cubic centimetres are equivalent to one gramme of urea. This is done by dissolving 71·5 grms. of pure mercury in nitric acid, and diluting with distilled water to one litre. Before urine is precipitated by this solution its phosphates must be removed by the addition of a mixture of one part of cold saturated solution of baric nitrate to two parts of a similar solution of baric hydrate. Further, if albumen be present, it must be separated by boiling and the addition of a few drops of acetic acid.

The process is thus performed:—Two volumes of urine (say 40 c.c.) are mixed with one volume (20 c.c.) of the baryta solution. After shaking well, the mixture is poured on a dry filter, and 15 c.c. of the clear filtrate (equal to 10 c.c. of urine) removed to a small beaker. Mercurial solution is now slowly added from a burette so long as precipitation occurs: But to find the exact point when all the urea has been precipitated, it is necessary to employ some such indicator as sodic carbonate. Several drops of a solution of this salt are scattered over a white plate; and by means of a glass rod a little of the mixture in the beaker is brought in contact with the soda. So long as there is any free urea present no change of colour takes place at the point of contact; but as soon as the mercury is in excess, a yellow precipitate results. The moment, therefore, the yellow colour shows itself distinctly, enough mercuric solution has been added. To attain an accurate result, the experiment should be performed a second time. Supposing 19 c.c. of the mercuric nitrate were required by the 10 c.c. of urine,

[1] 1 gramme = 15·432348 grains = the weight of a cubic centimetre of distilled water at 39·2° F.

1 litre = 1000 cubic centimetres = 61·024 cubic inches = 35·2754 fluid ounces.

this would indicate a percentage of 1·9 urea in the urine. If, however, it should be found that more than 20 c.c. are needed, the urine for the second examination must be diluted by adding half as much water as the excess of mercuric nitrate solution employed above 20 c.c. Thus, if 30 c.c. were required in the first precipitation, the excess is 10 c.c.; therefore, 5 c.c. must be added to the 10 c.c. of urine before the second precipitation. If, on the other hand, much less than 20 c.c. have been used, then for every 4 c.c. less the 20, ·1 c.c. must be deducted before calculating the percentage of urea.

b. A very easy method for the estimation of urea depends on the measurement of the nitrogen evolved when the urea is decomposed by a hypochlorite or hypobromite. Urea yields in this way all its nitrogen, less 8 per cent. Different apparatus are used; but in all of them there are mechanical arrangements to bring about the gradual admixture of the urine with the test solution, and to collect the gas evolved. 5 c.c. of a 2 per cent. solution of urea yield about 37 c.c. of gas. The collecting tubes are generally graduated so as to express at once without calculation the percentage of urea present in the urine experimented upon. The volume of gas given by, say, 5 c.c. of a 2 per cent. solution of urea, which is about 37 c.c., is taken as indicating 2 per cent. of urea, and the collecting tube is graduated accordingly.

3. *Uric acid* ($C_5N_4H_4O_3$) *and urates.*—Uric acid is derived from the same source as urea, and is liable to slight fluctuations in quantity under much the same circumstances as urea. It is readily decomposed by oxydising agents into several less complex substances, of which urea (to which it contributes the whole of its nitrogen) is the most important. It may, in fact, be regarded as representing a stage in the conversion of albuminous matter into the urea. It is exceedingly insoluble in water, and hence, when free in the urine, forms a crystalline deposit. It is, however, generally combined with a base, especially ammonia or soda, and in this form is much more soluble, though still liable to form a sediment. The main interest, indeed, attaching to the presence of uric acid and urates in the urine resides in the fact of their tendency to be deposited, and to take part in the formation of gravel and calculi.

Free uric acid often falls during the acid fermentation taking place in urine which is kept; and, when met with in fresh urine, it is generally in consequence of the acid reaction of that fluid being excessive. It may be readily recognised by the character of its crystals. These may form reddish grains visible to the naked eye, but are generally microscopic objects. The forms which they assume are various, and depend largely on the quality of the urine in which they are found. They are generally lozenge-shaped or rhomboidal, with the angles more or less rounded, and vary in thickness, so as to form, on the one hand, mere films, on the other, short flattened cylinders or prisms. When

abundant they are often grouped together into stellate or variously shaped clusters. If any doubt as to the nature of the deposit exist, it may be set at rest by converting it into murexide. This may be done by placing a little of it in a porcelain dish, adding to it a drop or two of strong nitric acid, and heating the whole to dryness. If now, when the residue is cool, a rod dipped in caustic ammonia be applied to it, the beautiful purple colour, characteristic of murexide, is developed.

Urates, comprising chiefly those of ammonia and soda, are often deposited in an amorphous condition, forming a powdery sediment which clings to the vessel, and which, from its attraction for the colouring matter of the urine, varies in tint from a light fawn to pink. Like uric acid itself, they generally precipitate in acid urine, but, unlike uric acid, they mostly fall in concentrated urine, especially when it becomes cool. The formation of uratic sediments often occurs in the urine of healthy persons, especially in cold weather; it often, however, attends and indicates the presence of catarrh or other febrile states, or derangements of the liver or other chylo-poietic viscera. Amorphous urates are readily recognised by the fact that urine which is turbid from their presence becomes perfectly clear and transparent when boiled. Further, the addition of acids causes the formation of crystals of uric acid; and murexide may be developed by the method already indicated. Urate of soda is occasionally present in the shape of small globular concretions beset with conical spikes. These form in the urine while it is yet in the urinary cavities, and are liable to cause much irritation and to lead to the development of calculi. They have been especially observed in the case of children suffering from febrile symptoms.

4. *Xanthine* ($C_5H_4N_4O_2$) is a waxy, white, non-crystallizable substance, almost insoluble in cold water. When heated with nitric acid it dissolves without evolving gas; and the residue left on evaporation acquires when heated with caustic potash a beautiful violet-red colour.

5. *Cystine* ($C_3H_7NSO_2$, or $C_3H_5NSO_2$) contains 25·5 per cent. of sulphur. It is closely related chemically to taurine, and hence probably furnished by the liver. It is a neutral body, insoluble in water, and crystallizes in hexagonal plates. It is dissolved by the mineral acids with decomposition, and by the caustic alkalies without. The best way to obtain the characteristic crystals is to dissolve the cystine in a solution of ammonia and allow the solution to evaporate.

6. *Leucine* ($C_6H_{13}NO_2$) and *tyrosine* ($C_9H_{11}NO_3$).—These are formed under the same conditions and are generally associated together. Pure leucine occurs in white crystalline scales, has a fatty feel, and dissolves in water. In the impure state, as observed in urine, it often assumes the form of roundish concentrically marked yellowish bodies which have some resemblance to fat-globules. If a small portion

of leucine be saturated with nitric acid and the mixture carefully evaporated to dryness, it leaves an almost transparent residue which turns yellow or brown on the addition of solution of caustic soda, and yields an oily drop when reheated. Tyrosine forms a white, silky, glistening mass, consisting of fine needle-like crystals, which are grouped in radiating clusters and sometimes in dense globular masses. It gives a red colouration when boiled with Millon's reagent; and a violet hue when gently warmed with sulphuric acid and a drop of solution of perchloride of iron is added. The urine of patients suffering from yellow atrophy of the liver often deposits spontaneously a greenish-yellow sediment consisting of crystals of tyrosine, and on evaporation yields numerous crystals of the more soluble leucine.

7. *Colouring matters.*—The normal pigments of the urine are derived from the colouring matter of the blood, and, according to Schunck, are two in number. He names them respectively *urian* and *urianine*. They are of a dark yellow colour and syrupy consistence, have a high atomic constitution, and contain nitrogen. Their excess or deficiency has little special pathological importance. Another urinary pigment has been described by Dr. Thudichum under the name of *urochrome*. The pink colouring matter, however, termed *purpurine* or *uro-erythrine*, is a pathological product. Its chemical constitution and source have not been ascertained; but it is common in febrile and inflammatory affections and in cases of organic disease, especially of the liver, and has a remarkable affinity for uratic sediments to which it clings and imparts its special tint. *Indican*, the peculiar body by whose decomposition indigo-blue and indigo-red are obtained, has been ascertained by Schunck to be a normal constituent of urine. It is to this source that the occasional presence of indigo-blue in decomposing and morbid urine appears to be due.

8. *Odorous matters.*—The peculiar smell of normal urine is due to the presence of minute proportions of certain volatile organic acids, which need not be specified. This smell is well marked when the urine is acid; but when it is alkaline from fixed alkali it acquires a sweetish odour instead, and when alkaline from decomposition becomes ammoniacal. Diabetic urine has a peculiar sweetish smell.

9. *Grape or starch sugar. Glucose. Dextrose* ($C_6H_{12}O_6$, H_2O).—A trace of this substance is frequently, if not always, present even in healthy urine. Occasionally it is found in excess under the influence of various morbid conditions of the system. Its habitual presence in quantity is the distinctive feature of the malady known as diabetes mellitus. Diabetic urine is usually of high specific gravity, has a sweet taste, very rapidly develops torulæ, ferments on the addition of yeast with the disengagement of carbonic acid gas, and (as one of the names of its saccharine constituent implies) rotates the plane of polarisation to the right.

Many tests for the presence of sugar, some founded on the facts above enumerated, have been devised. Of these *Trommer's* or *the copper test*, which depends on the influence possessed by grape-sugar in decomposing the salts of copper and throwing down the insoluble red sub-oxide, is probably the best. It may be applied as follows:—

Mix the suspected urine with half its volume of solution of caustic potash or soda. If much precipitate be produced, it should be separated by filtration. Then add a drop or two of a dilute solution of sulphate of copper, and heat the mixture in a test-tube. Even before the boiling point is reached (if sugar be present) the characteristic precipitate will begin to appear; and as soon as this point has been attained the heat should be withdrawn, since other substances besides sugar effect by prolonged ebullition a reduction of cupric salts. The effect of this process on diabetic urine is that after a few seconds the mixture suddenly turns of an intense opaque-yellow colour, and in a short time an abundant yellow or red sediment falls to the bottom. The test is best applied by using a ready-made alkaline solution of tartrate of copper. Moreover, by means of a standard solution of this kind the quantity of sugar in urine can also be estimated.

a. Fehling's solution, which is employed for the above purpose, is thus prepared:—Dissolve 34·64 grammes of pure crystallised sulphate of copper in 200 c. c. of distilled water. Separately dissolve 173 grms. of pure crystals of Rochelle salt in 480 c. c. of solution of caustic soda (sp. g. 1·14). Mix the two solutions and dilute up to a litre. 10 c. c. of the mixture contain ·3464 grms. of cupric sulphate or ·108 grms. of cupric oxide, and represent ·05 grms. of pure anhydrous grape-sugar. This mixture has a great tendency to spoil by keeping. To obviate this it is advantageous to prepare the solutions as follows:—Dissolve 34·64 grms. of cupric sulphate in distilled water, dilute up to a litre, and keep in a separate bottle. Dissolve 173 grms. of Rochelle salt in 350 c. c. of distilled water, and heat to boiling: on cooling add 600 c. c. of solution of caustic soda (sp. g. 1·12) that has been previously boiled, and make up to a litre with distilled water. The second solution is to be kept in a separate bottle, and to be mixed with the former in equal proportions when required for use.

To estimate the amount of sugar present in diabetic urine dilute 10 c. c. of urine with distilled water up to 200 c. c.; and pour into a Mohr's burette. This dilution is to reduce the sugar below 1 per cent. Then place 10 c. c. of Fehling's solution, or 20 c. c. of the mixture last considered, in a small flask, add distilled water up to 50 c. c., and boil the whole over a Bunsen's flame—the flask resting on some wire gauze immediately below the Mohr's burette. Then allow the diluted urine to flow slowly into the boiling copper solution until the blue colour has nearly disappeared. After this point the urine must be added more cautiously, and the flask well-agitated after each addition. The precipitated sub-oxide settles rapidly on removing the flame, thus

allowing any tinge of blue in the supernatant fluid to be readily seen on holding the flask obliquely over a white ground. So long as any trace of colour remains more urine must be added, and the boiling must be continued. To make sure that all the copper has been precipitated, a little of the test-mixture should be filtered and tested with ferro-cyanide of potassium and acetic acid. The appearance of a brown colouration or precipitate indicates the presence of copper. When once the examination of the urine has been commenced it should be completed as soon as possible, to prevent any re-solution of the sub-oxide. Supposing 60 c. c. of the diluted urine have been required by the 10 c. c. of Fehling's solution, then every 60 c. c. of the diluted urine, or every 3 c. c. of the urine itself (since it has been diluted twenty times), contains ·05 grms. of sugar, or about 1·6 per cent.

b. Another method, known as *Knapp's*, may be employed. It possesses certain advantages over Fehling's process. The test solution is easier to make, it keeps a long time without alteration, the analysis requires a shorter time, and the termination of the reaction is more easily determined. The process is based on the power possessed by grape-sugar of reducing to the metallic state the mercury contained in a boiling alkaline solution of mercuric cyanide—100 parts of sugar reduce 400 parts of cyanide. The standard solution is prepared by dissolving 10 grms. of the cyanide in 600 c. c. of distilled water, adding 100 c. c. of solution of caustic soda (sp. g. 1·145) and diluting up to a litre with distilled water. 40 c. c. of the mercuric solution (containing ·4 grms. of the cyanide, and equivalent to ·1 grm. of sugar) are heated in a flask, and the diluted urine, as in Fehling's process, slowly added until the whole of the mercury is precipitated. The mercury falls rapidly, and the completion of the process can be ascertained by bringing a drop of the supernatant fluid in contact with a piece of wet filter paper which has been exposed to the fumes of hydrochloric acid, and subsequently to sulphuretted hydrogen. A trace of dissolved mercury gives at once a yellow or brownish colouration.

Both of the above analyses should be repeated a second time in order to insure accuracy.

10. *Amorphous phosphate of lime* ($Ca_3,2PO_4$).—This precipitates only in alkaline urine; it forms an amorphous sediment like that of the urates, but does not carry with it the urinary colouring matter. The application of heat increases the precipitate, and not unfrequently causes it. It is dissolved, however, on the addition of a drop or two of nitric acid. It often forms an iridescent pellicle on the surface.

11. *Crystallised phosphate* (CaH,PO_4).—Dr. Roberts regards this sediment, which is rare, as an accompaniment of grave disorders. It occurs in rods and needles, which are often arranged in tufts and stars.

12. *Ammoniaco-magnesian phosphate* (H_4NMg,PO_4) always falls in ammoniacal urine. Its crystals occasionally appear in slightly acid urine; but are much more frequently observed in that which is

alkaline, and then often associated with the amorphous phosphate. They are occasionally met with as an habitual constituent of freshly-voided urine. The ordinary crystalline form is that of a triangular prism with bevelled ends. But this is liable to numerous modifications.

13. *Oxalate of lime* (CaC_2O_4, $2H_2O$).—The presence of oxalic acid in urine is not surprising considering that it, with carbonic acid, is one of those ultimate substances into which organic matters become reduced. Its presence is doubtless in the majority of cases due to such reduction, but sometimes it depends on the ingestion of articles of diet, such as rhubarb, which contain it. It occurs in the urine in combination with lime, usually forming small oblique octahedral crystals, and occasionally dumbbell-shaped bodies. The crystals generally fall, entangled with the mucus, and when large may be seen as shining points with the naked eye. Their occasional presence is a matter of little importance; but when they are of habitual occurrence there is reason to fear the formation of oxalate of lime calculi, and there is often some obvious impairment of health. Oxalate of lime rarely occurs in alkaline or neutral urine. It is readily soluble in the mineral acids; and precipitable from solution by excess of ammonia.

14. *Carbonate of lime* ($CaCO_3$) is sometimes deposited as an amorphous powder in alkaline urine, and is occasionally found in the form of minute rounded calculi, with a well-marked concentric structure.

15. *Albumen.*—This substance is seldom met with in healthy urine, and its presence, in any quantity at least, is one of the most significant indications of renal disease. It is observed under various circumstances. Whenever suppuration occurs in connection with any of the urinary organs, and pus is discharged into the urine, albumen is present in small proportion. In many specific fevers and other febrile disorders, albuminuria is liable to occur. In congestion of the kidneys, due to heart disease, bronchitis, or obstruction of the renal veins or arteries, again, albuminuria is frequently observed. The most important causes of this condition, however, are inflammation of the kidney, and those various chronic lesions which are usually comprehended in the term 'chronic Bright's disease.' It has occasionally been discovered in healthy urine shortly after a meal of eggs.

The presence of albumen in the urine may always be recognised by its coagulation under the influence of heat or nitric acid. To apply the former test, a portion of the urine should be placed in a test-tube and then boiled by means of a spirit-lamp. If it contain albumen, opaque flakes form in it, which render it more or less turbid, and gradually fall to the bottom of the glass. If there be much albumen present, turbidity appears before the urine begins to boil; if there be only a trace, actual ebullition is essential to its production. In the employment of heat one or two precautions are necessary to be observed. In the first place, albumen is not precipitated if the urine be alkaline, and hence such urine should first be acidified by the

addition of a few drops of acetic acid. In the second place, in slightly alkaline or neutral urine, heat is apt to throw down a deposit of amorphous phosphates. These, however, dissolve on the addition of an acid. The nitric acid test may be employed as follows:—A test-tube should be filled to a depth of ½ or ¾ inch with strong nitric acid, and then a small quantity of urine should be slowly poured down the side of the inclined tube so that it may rest on the acid without mixing with it. If albumen be present, a white cloud soon appears in the layer of urine which is in contact with the acid. The fallacies which may arise in connection with this test are: first, that the urine of patients who are taking cubebs or copaiba is apt to become slightly turbid under the influence of nitric acid; second, that in concentrated urine and such as is rich in urea, some deposition of urates or of nitrate of urea may occur; and, third, that the addition of a very minute proportion of nitric acid does not always precipitate albumen, while the addition of an excessive quantity may prevent its precipitation altogether. A saturated solution of picric acid also precipitates albumen. The relative quantity of albumen present in any specimen of urine may be roughly but conveniently estimated by boiling the whole of the slightly acidified portion placed in a test-tube, and then allowing the coagulated flakes to subside.

16. *Blood* may be found in the urine in various proportions and in different conditions, and may be furnished by any part of the urinary tract, from the kidneys downwards. The greater the quantity of blood passed, and the nearer its source to the external urethral orifice, the less will it deviate from the normal condition of blood, and the more readily will it be recognised. Its presence may be due to injury, congestion, inflammation, carcinomatous and other like growths, concretions, or parasites, involving either the substance or pelvis of the kidney, or some other part of the excretory apparatus, such as the ureter, bladder, or urethra. Hæmaturia occasionally also follows the use of cantharides or other drugs, and is frequently met with not only in those febrile disorders (small-pox, scarlet fever, and the like) which are attended with albuminuria, but in purpura, scurvy, and other affections which assume a petechial character. When much blood is effused, it occasionally coagulates in the bladder; and may even coagulate more or less imperfectly in the chamber pot. When present in smaller quantities and diffused uniformly throughout the urine, it imparts to it a slight degree of opacity or turbidity, and a tint resembling that of a dilute solution of the compound infusion of roses, or a peculiar smoky or dirty reddish brown hue varying in depth and distinctness according to the quantity of blood present. Sometimes the urine resembles porter. On standing it usually deposits a grumous or coffee-ground-like sediment. The presence of blood is additionally proved by the detection of albumen in the urine by the usual tests, and by submitting a specimen to microscopic examination,

when almost always blood-corpuscles will be readily detected, sometimes disc-like, sometimes globular, sometimes crenate, occasionally retaining their colouring matter, but usually colourless, having imparted their pigment to the fluid in which they float. In a peculiar affection shortly to be described—paroxysmal hæmaturia—although the urine contains abundance of blood, distinct blood-corpuscles are rarely detected. And occasionally (as Dr. Mahomed has shown to be especially the case at a certain period in scarlet fever, prior to the occurrence of albuminuria and anasarca), the colouring matter of the blood alone transudes into the urine, where it may be detected either by the spectroscope or by the guaiacum test. The latter may be applied as follows:—Place a drop or two of the urine in a small test-tube, add one drop of tincture of guaiacum and a few drops of ozonized ether; agitate, and then allow the ether to collect at the top. If blood pigment be present, the ether acquires a blue colour, leaving the urine below colourless. No saliva must be mingled with the urine, and the patient must not be taking any salt of iodine.[1] Further, unless the tincture be freshly prepared the reaction is liable to fail.

17. *Bile.*—The colouring matter of the bile and the biliary acids are found in greater or less abundance in the urine in cases of jaundice. The former may, according to its amount, impart merely a yellowish tint, scarcely or not at all distinguishable from that of normal urine, or any variety of shade between this and a deep olive green. Bile-stained urine seen by reflected light often looks almost black. The presence of biliary pigment in the urine may be readily detected by the addition of strong nitric acid, which produces, where the fluids first come into contact, an evanescent succession of green, blue, violet, and red tints. The test may be applied, either by placing a few drops of urine and a few drops of nitric acid close to one another on a white porcelain surface, and then allowing them to come together; or by putting a little nitric acid at the bottom of a test-tube, and pouring a small quantity of urine carefully on the top of it without allowing them to mix. In the former case the play of colours takes place at the line of mixture, in the latter in the horizontal plane of contact. Dr. G. Harley considers that the presence of the biliary acids in the urine is characteristic of jaundice from *retention* of bile. For their detection the following process (a modification of Pettenkofer's) may be employed:—Add a few drops of syrup to the urine, and then shake briskly in a test-tube until a froth has formed. Next allow a drop of strong sulphuric acid to flow down the side of the tube. As soon as the acid reaches the froth a beautiful purple colour develops rapidly. The reaction is facilitated by gently warming the side of the test-tube.

18. *Casts.*—In almost all cases in which albuminuria or hæmaturia is due to morbid conditions of the secreting structure of the kidneys, and occasionally in specimens of urine which seem to be free from both

[1] Mahomed, Med.-Chir. Trans. vol. lvii.

blood and albumen, microscopic cylinders which have been moulded in the urinary tubules, and are therefore termed casts, may be detected with the aid of the microscope. Of these several varieties may be distinguished. The following enumeration comprises the more common of them. *a. Epithelial casts* consist of renal epithelium. Occasionally the epithelium differs little from the normal epithelium of the tubules. More commonly the cells are granular and degenerating or studded with oil-globules. In other cases the casts are formed mainly if not entirely of new-formed cells, which then assume an embryonic character, and have therefore more or less resemblance to pus-cells. *b. Hyaline casts.* These present two well-marked varieties of which one may be termed mucous, the other waxy. The former are exceedingly translucent and delicate, and consequently may readily escape detection. They are colourless, homogeneous, or ground-glass-like, with little or no refractive power, soft and flexible. They present soft but definite edges, are generally narrow and often of considerable length. They are proteinous, but not fibrinous; and are unaffected by acetic acid. The waxy casts also are transparent and homogeneous; but they are highly refractive, and therefore present well-marked shaded edges. Moreover they are brittle, are apt to present transverse fractures, and vary largely in diameter and length. Like the former they are not acted on by acetic acid; but they readily absorb biliary, blood, or other colouring matters. *c. Granular casts.* These vary in size, but are often of considerable bulk, and are studded more or less thickly and more or less irregularly with granular matter, which often renders them perfectly opaque. They are hyaline, generally waxy, casts which have either undergone granular degeneration in a greater or less degree, or are studded with or enveloped in the débris of degenerating cells. Indeed, compound granule-cells are often seen distinctly imbedded in them. *d. Fatty casts* are characterised by the presence of obvious fat-globules, which are sometimes of considerable size. Such globules may be observed in either epithelial, hyaline, or granular casts. The fatty matter is not pure olein, but seems generally to be a mixture of this with cholesterine and some albuminous matter. *e. Blood casts.* Generally in renal hæmaturia the casts consist, in a greater or less degree, of coagulated fibrine entangling the corpuscular elements of the blood. The basis of the casts is here purely fibrinous; it is fibrillated, and dissolves in acetic acid. The blood corpuscles may present pretty nearly their normal characters; they are generally, however, compressed and angular, and are often broken down and individually undistinguishable. The casts are necessarily more or less deeply pigmented. Blood corpuscles or blood pigment may be present in greater or less proportion in epithelial or hyaline casts.

It occasionally happens that the various casts above described, and more especially perhaps the waxy and granular casts, contain crystals of either uric acid, oxalate of lime, or triple phosphate, or

granules of urate of soda, or refractive globules looking like oil, but consisting of the crystalline bodies just named in combination with animal matter.

As regards the sources of urinary casts, it is important to bear in mind that the convoluted tubes of the kidneys which are functionally the most important can scarcely yield them, inasmuch as they are of comparatively large diameter, and are separated from the straight or collecting tubes by the narrow loops of Henle. Indeed there is little doubt that casts found in the urine come exclusively from the loops of Henle and the straight tubes, that the descending limbs of the loops furnish the smallest, the ascending limbs those of intermediate size, and the straight tubes, and more especially their terminal portions, the largest casts.

On these and other grounds the significance of casts is less than is generally supposed. They are often absent in cases of albuminuria or chronic Bright's disease; and mucous casts are occasionally observed in jaundice, and in cases in which there is neither albuminuria nor kidney disease, and even in health. Epithelial casts usually imply acute affections; large hyaline, fatty and granular casts, chronic and degenerative disease; while mucous casts have no special significance.

19. *Mucus and pus.*—In normal urine but little mucus is present; it falls as a scarcely perceptible cloud, and contains perhaps traces of vesical and urethral epithelium, and in the female squamous vaginal epithelium. When, however, there is any inflammatory condition of the mucous lining of the urinary channels or reservoirs, the mucous secretion becomes increased, cells are discharged in excess, and immature forms, in other words, cells identical with those of pus, are produced in greater or less abundance. The transition between mucus and pus is almost imperceptible. The discharge, if sufficiently abundant, renders the urine turbid and slightly albuminous; and a sediment, which may present a greenish-yellow hue, presently forms. If the urine retain its acid reaction, this sediment is readily miscible with the urine; if, however, it become, as it is very apt to become, alkaline, then the sediment becomes tenacious and ropy. The secretion of inflammatory mucus has a remarkable influence in promoting the decomposition of urea; the urine, therefore, in these cases has a great tendency to become ammoniacal, to deposit earthy phosphates, and to acquire irritant properties. The abnormal secretions here described are most commonly furnished by the inflamed mucous membrane of the pelvis of the kidney, or bladder. But it must not be forgotten that pure pus may be poured into the urinary passages, either from renal abscesses or in consequence of the rupture of some neighbouring abscess into them, and that cells identical with pus-cells may escape from the renal tubules. Pus can be readily recognised by its microscopic characters.

20. *Fat*, excepting in connection with renal casts, is of rare occur-

rence in the urine. The presence of fluid fat in the form of globules is said to have occasionally been observed. Crystals of cholesterine also have been met with. In a case of Dr. Murchison's, the cholesterine was traced to a pyonephritic cyst. The most interesting cases of fatty urine, however, are those in which this fluid presents a milky or chylous character, due to the presence of fatty matter in a molecular or amorphous condition. In these cases the urine contains albumen, fibrine, and leucocytes, in addition to fat; it hence tends to coagulate spontaneously; it coagulates with heat; a creamy layer rises to the top when it is allowed to stand; and it may be rendered clear, and the fat be separated, by agitating it with ether. Many of the globules which are commonly regarded as fat, and look like it, are really composed of some of the crystalline constituents of the urine in combination with animal matter, as may be shown by the effects of reagents and the appearance of a cross when examined with polarised light.

21. *Morbid growths.*—Tubercle, carcinoma, and other growths are apt to arise in various parts of the urinary organs; and it might hence be supposed that their characteristic elements should be occasionally discovered in the urine. It must be exceedingly rare, however, for such specific indications to be met with in connection with disease of the kidneys. In villous growths of the bladder, fragments may, no doubt, be detached and occasionally discovered in the urine. It must be borne in mind, however, that the cells of the vesical epithelium have a great resemblance to typical cancer cells, and may be easily mistaken for them.

22. *Spermatozoa* are sometimes present in the urine, and may be readily recognised in the sediment. Their presence is of little clinical importance, unless other symptoms combine to indicate the existence of abnormal spermatorrhœa.

23. *Animal and vegetable organisms.*—Hyatids are occasionally developed in the urinary organs, or hydatid cysts may open into them. The urine under such circumstances may present actual hydatids or echinococci, or fragments of one or the other. The peculiar laminated character of the hydatid membrane, and the hooklets of echinococci, are, under the microscope, quite unmistakable objects. In the endemic hæmaturia of Egypt, the Cape, Natal, and other parts of Africa, the symptoms are due to the presence, in the veins of the pelvis of the kidney, ureter, and bladder, of a small unisexual parasite, termed the bilharzia hæmatobia. The presence of this affection may be recognised by the discovery in the urine of the ova and free embryos of the parasite. The filaria sanguinis hominis is found in some cases of chyluria.

Sarcinæ have been observed in the urine when passed from the bladder. Lastly, bacteria and penicillium form rapidly in urine undergoing decomposition, and the yeast-plant in that of diabetic patients.

contraction of the smaller blood-vessels; hypertrophy of the heart; anasarca and other dropsical effusions; local congestions and hemorrhages; inflammation of different organs, mainly those of the thorax; and, lastly, various functional diseases of the digestive and other organs, but, above all, of the central nervous system.

1. *Thickening and contraction of the smaller blood-vessels.*—Dr. G. Johnson showed some years since that in cases of chronic renal disease the walls of the minute arteries, both in the kidneys themselves and generally throughout the system, became extremely thick, and at the same time much contracted. He attributed the thickening to hypertrophy of the muscular coat and the narrowing to the tonic contraction of this coat, and regarded the combined phenomena as an effort of nature to oppose the transmission of poisoned blood to the tissues. The thickening of the arterial tunics and the contraction of the arterial channels in chronic renal disease are now established facts. It has, however, lately been maintained, more particularly by Sir W. Gull and Dr. Sutton, that the thickening is the result not of muscular hypertrophy, but of a 'hyaline-fibroid' conversion; that it is in fact a change not unlike that which occurs in cirrhosis of the liver and sclerosis of other organs—a change which in a sense may be regarded as degenerative. In these latter views, so far as we have stated them, we are disposed to concur.

2. *Hypertrophy of the heart,* independent of valvular affection, has long been recognised as one of the most obvious attendants on chronic kidney disease. The hypertrophy is general, and associated with more or less dilatation; but the changes are, perhaps, more obvious in the left ventricle than elsewhere. Dr. Quain has shown that the thickening of the walls is due in some degree to increase of the connective tissue, in other words to a kind of sclerosis; there is no doubt, however, that it is mainly dependent on muscular overgrowth, and that the stimulus to this overgrowth consists in some obstacle which the heart's action has to overcome. But since the valves and larger arteries are all, for the most part, healthy, this obstacle is not presented by them. There are obvious reasons why the veins must be considered to be inoperative in the matter. We are compelled, therefore, to look to the small arteries and capillaries. And that the obstruction really does reside in these vessels is clearly shown by the high tension which by sphymographic observation has been proved to prevail throughout the arterial system in such cases. It was formerly believed that the obstruction was caused by some abnormal attraction between the capillary blood-vessels or the tissues outside them and the morbid blood. It is, however, doubtless due to the contraction of the channels of the capillary arteries. Dr. Sibson has shown that generally in these cases the contractions of the two sides of the heart are not quite synchronous, and that there is a tendency, therefore, to reduplication of the heart's sounds.

3. *Anasarca and other dropsical effusions.*—Kidney disease is one of the most frequent causes of general anasarca. This condition often reveals itself first in regions in which the connective tissue is lax, as the scrotum, eyelids, and conjunctivæ, and is often recognised in the face before it appears in the lower extremities. There is generally neither lividity nor dilatation of veins; but the swollen surface presents an anæmic, wax-like character. Its cause is somewhat obscure. It is evidently not passive, for there is neither venous obstruction nor venous hyperæmia; nor again is there any obvious impediment to the healthy action of the lymphatic vessels. It must then be due either to some peculiar tendency in the serum of the blood to transude through the capillary vessels, or to the sweating of this fluid through the walls of the smaller arteries in consequence of the heightened pressure which the blood within them exerts. In reference to this question it should be mentioned that Dr. Mahomed has recently shown that in scarlet fever there is a stage, preceding the occurrence of anasarca and even the appearance of blood or albumen in the urine, during which high arterial tension prevails as demonstrated by the resistance of the pulse to pressure and the form of the pulse-trace, and during which also the colouring matter of the blood may sometimes be recognised in the urine. The anasarca is not merely subcutaneous, but may involve the tissues of the larynx, the pulmonary texture, and other parts of the system; and is commonly associated with effusion into the several serous cavities. It is usually attended with a dry skin and considerable diminution of urine; to which circumstances and to co-existent anæmia the presence of dropsy is no doubt in part attributable.

4. *Congestions and hemorrhages* are among the consequences of kidney disease. The most important of them are: effusion into the substance of the brain, causing apoplectic symptoms; effusion into the choroid and retinal coats of the eye (albuminuric retinitis), attended with aching across the temples and at the occiput, and leading to atrophic changes and more or less impairment of vision, or even absolute blindness; and effusion into the lung-substance, producing the condition known as pulmonary apoplexy. The causes of these hemorrhages are, in part, the same as induce anasarca; but in chronic renal disease there is a marked tendency to atheromatous and fibroid degeneration of arteries, and hence effusions of blood are in some cases due to rupture of diseased and enfeebled vessels.

5. *Inflammatory affections* are of frequent occurrence. The most common and serious of these are inflammations of the pericardium and pleuræ, of the larynx, bronchial tubes, and lungs. But inflammation may also affect the abdominal viscera; and, indeed, no part is wholly exempt from liability to it. When anasarca is present it is of course common for an erythematous blush to make its appearance somewhere

or other on the surface, and even for erysipelas or superficial gangrene to occur.

6. The *functional consequences* of renal disease are very numerous. Dyspepsia, nausea, vomiting, and diarrhœa, the former three especially, are common phenomena, even when the stomach is healthy. Palpitation and dyspnœa, or hurried respiration, are not unfrequently observed in cases in which the heart and lungs present little if any sign of disease. Drowsiness, headache, irritability, hypochondriasis, and even more or less maniacal disturbance and wakefulness, are all of them liable to arise. But the most serious of the functional disturbances of the nervous system are coma and convulsions. These are generally preceded by some of the less grave mental phenomena above enumerated. The convulsions occur in paroxysms which almost exactly simulate those of true epilepsy, and, associated with coma, often succeed one another at short intervals until they terminate in death. Coma or apoplectic symptoms may occur independently of convulsions.

D. *The non-specific Morbid Phenomena which attend on and characterise Lesions of the Kidneys.*

Other symptoms which attend and indicate the presence of renal disease are totally independent of impairment or suppression of the proper functions of the kidney. These are, symptoms which are determined by the locality of the diseased organ, and such as are referrible to it as a focus of inflammation or other morbid processes. Among the former may be comprised pain and tenderness, tumour, and the effects of pressure; amongst the latter the general symptoms of inflammatory fever when the organ is inflamed, the cachexia which attends the development of malignant disease, and the anæmia which results from the continued escape of blood, or of that important element of the blood—albumen.

II. PYELITIS.

Causation.—Inflammation of the lining membrane of the kidney may be excited in various ways. It seldom results from exposure to cold, or arises in association with ordinary nephritis. It may, however, be induced by the use of certain medicinal irritants, such as cantharides and turpentine, which probably induce at the same time a similar condition in the lining membrane of the bladder, and in the secreting tissues of the kidney. But its most frequent cause is direct irritation of the mucous surface, due either to the constant fretting of a renal calculus or to the influence of unhealthy discharges or decomposing urine, as occurs in cases of long-continued obstruction of the

urinary passages. Independently of the last condition, vesical inflammation is apt to creep by continuity along the ureter to the pelvis, and thence to the infundibula and calyces.

Morbid anatomy.—The anatomical signs of pyelitis are congestion, thickening and softening of the mucous membrane, sometimes associated with interstitial hemorrhage; and the discharge from its surface of mucus containing shed epithelial cells and pus-like corpuscles, and, it may be, blood. If the affection be persistent or intense, other phenomena probably supervene; the thickened mucous membrane may become opaque, yellow, or grey, and lose its vivid redness; suppuration may arise; false membranes may be formed; or ulceration may take place. Further, the effect of the unhealthy products of the mucous surface upon the urine is to render it ammoniacal and to promote the precipitation of earthy phosphates, which are then apt to concrete in more or less abundance on the inflamed surface. Other changes which are liable to ensue in the course of pyelitis depend on impediment to the escape of urine from the inflamed cavity; they are dilatation of the pelvis, infundibula, and calyces, and atrophy of the secreting structure. Again, inflammation may extend by continuity from the pelvis to the renal substance, and abscesses may consequently form in it. Suppurative pyelitis, especially if it be confined to one kidney, and pus can escape freely from it by the natural passages, may continue for years with little or no additional mischief; and even if complete obstruction of the ureter arise, it is possible that the whole thing may become quiescent, the expanded, atrophied, and indurated renal substance losing all its functional power, and the pus in the dilated calyces and the rest of the renal cavity drying up into a creamy, putty-like, or mortary substance. In other cases, however (and this may happen whether the ureter be wholly or only in part obstructed), the renal abscess takes another course. It behaves, in fact, as any other abscess originating in the vicinity might behave. It first transgresses its original renal limits, and then forms sinuses which enlarge and burrow in various directions. Thus, they may perforate the diaphragm, and open into the pleura or lung; or they may discharge in the loin; or they may rupture into the peritoneum, or open directly into the adjoining colon; or, descending along the psoas muscle, they may point under Poupart's ligament, or gravitate towards the lesser trochanter; or lastly, passing into the pelvis, they may communicate there with the rectum, bladder, or vagina.

Symptoms and progress.—The specific symptoms of pyelitis comprise pain and tenderness in the loin, irritability of the bladder, and modification of the quality of the urine. The pain in the loin is apt to shoot into the abdomen, and especially downwards to the labium or testis of the corresponding side and along the inner aspect of the thigh. The tenderness reveals itself, and the pain is aggravated, during movement of the body; but especially if the affected side of

the abdomen be firmly grasped, or the thigh be flexed by its own muscular efforts on the abdomen, in which case the enlarging bulk of the psoas muscle presses on the inflamed organ. There is probably irritability of the bladder, with pain and scalding in micturition. The water is more or less turbid from the presence of mucus, or it contains pus or blood, or both. It is usually acid; but, after a time, is apt to become ammoniacal from the decomposition of urea, and then to deposit amorphous and crystalline phosphates. It does not necessarily contain renal casts. Their presence indicates of course simultaneous involvement of the secreting structure of the kidney. Sometimes the discharge of pus is profuse; and both in this and in other cases the products of the inflamed surface are not unfrequently passed intermittently—temporary obstructions probably taking place in the ureter, in consequence of which they are retained and accumulate in the renal cavity with aggravation of local symptoms, and the urine becomes for the time comparatively clear and healthy. The general symptoms are mainly those of inflammatory fever. This assumes for the most part a remittent character, and is often attended with rigors. Vomiting and diarrhœa are not unfrequent.

The symptoms, progress, and results of pyelitis differ in different cases. If one kidney only be affected the disease may continue indefinitely without any very serious impairment of the patient's health—indeed, the organ may become totally disorganised with little or no obvious detriment to health; but on the other hand the formation of an abscess is in any case attended with many risks, and its continuance may cause death either by slow exhaustion, aggravated probably by the presence of hectic fever, or lardaceous degeneration, or by the supervention of some intercurrent affection. When, however, both kidneys are involved, as may be the case in calculous pyelitis, and as nearly always takes place when pyelitis is secondary to bladder disease, the symptoms which the patient presents are necessarily greatly aggravated, and the probability of an early fatal issue is much increased. For in addition to the risks which attend disease confined to one kidney, we have now the additional risks which arise from the liability to more or less complete retention of urea in the blood, and those which flow from the comparatively wide extent of the inflamed district. The patient passes into a typhoid condition, attended with muttering delirium, and not unfrequently complicated with epileptiform convulsions and coma.

Accumulation of pus in the kidney may be suspected when the discharge of pus with the urine ceases suddenly and continues in abeyance; it may also be suspected when, following upon symptoms indicative of pyelitis, rigors take place and at the same time throbbing pain and tenderness manifest themselves in the region of one of the kidneys. The diagnosis of an abscess must be based partly on the persistence of the above conditions, partly on the presence of increasing

fulness in the neighbourhood referred to. If the abscess point externally all doubt will be speedily removed. Under other circumstances many difficulties will necessarily present themselves.

Treatment.—In the treatment of pyelitis it is of primary importance to ascertain its cause, and to remove or obviate it if possible. Thus, when it depends on retention of urine, from stricture, enlarged prostate, or paralysis of the bladder, our aim must be (if not to cure these lesions) at all events to empty the bladder periodically and if necessary to wash it out with antiseptic solutions; when it depends on the presence of renal or vesical calculi, we must endeavour to remove them, or, failing this, to maintain rest; if the inflammation be connected with gout, scrofula, or any other special cachexia, it will probably be well to modify our treatment accordingly.

When pyelitis is acute and the local symptoms are severe, it may be necessary to remove blood from the loin either by cupping or by leeches, and to use hot fomentations, poultices, ice-bags or equivalent applications. Counter-irritants, too, always excepting cantharides, may be employed. The administration of opium in doses sufficiently large and sufficiently often repeated to relieve pain and procure ease and rest, is of essential importance. Moderate purging, voluminous bland clysters, and hot baths are also valuable aids. When the disease assumes a more chronic character local measures become less important, and opiates also are comparatively little needed. It may, however, still be desirable to give the latter in small doses, or to administer some other form of sedative or anodyne, such as hyoscyamus, belladonna, or chloral hydrate. But tonics and nutritious diet now become our most valuable remedial agents; among the former, quinine and the other vegetable bitters and iron—particularly the perchloride—and cod-liver oil, must be especially enumerated. If the urine be alkaline nitro-muriatic acid or some other mineral acid may be beneficially combined with the other remedies. Buchu, pareira brava, and uva ursi, so much appreciated by surgeons in the treatment of chronic inflammation of the urinary bladder, are probably equally useful in the treatment of pyelitis. If the stomach be irritable, as it not unfrequently is, our treatment must be modified with the object of overcoming this irritability. When there is clear indication of the formation of an abscess in or around the kidney, an early and free opening should be made into it, for by that means not only may the extension of the abscess in other directions be prevented, but the cure of the disease will not improbably be effected.

III. CIRCUMSCRIBED AND SUPPURATIVE NEPHRITIS.

Causation.—The chief causes of the conditions about to be considered are:—obstruction of the renal arteries or arterioles by thrombi or (in the case of cardiac disease or pyæmia) by emboli; extension of inflammation from the pelvis of the kidney or other neighbouring parts; and accidental injury.

Morbid anatomy.—The results of arterial thrombi or emboli are the same in the kidney as elsewhere. If the obstructed vessel be of large or medium size, the district to which it leads becomes deeply congested, blood accumulates and stagnates in the arteries, veins, and capillaries, and escapes from them, by rupture or otherwise, not only into the intertubular tissue but into the Malpighian capsules and convoluted tubules. The affected district is at first of a deep red or reddish-black colour and well defined, resembling a patch of pulmonary apoplexy; but gradually it becomes decolourised and acquires a more or less opaque, buff-coloured, cheesy aspect, when, if it be examined microscopically, the small vessels will be found loaded with pigment-granules and oil, and the epithelium of the tubules fatty and disintegrating. Sometimes it softens, sometimes suppurates. But the disintegrated tissues may also undergo absorption, and a patch of cicatricial tissue result. In the embolism of cardiac disease, and especially in that occurring in pyæmia, the infarctions are for the most part small and numerous, and speedily suppurate. In such cases, on removing the capsule, beads of pus each surrounded by a congested halo may be seen projecting from the surface of the organ; and on making a vertical section small abscesses or groups of abscesses, similarly surrounded, may be observed extending in a radial direction from the periphery to the mucous surface. These may vary from mere points up to the size of a filbert or walnut. They originate in the intertubular spaces, but soon involve and destroy the tubules themselves and the other renal structures. When inflammation extends from the pelvis of the kidney there is often general congestion with enlargement of the organ; but the special feature of such extension is the formation, in both medulla and cortex, of minute close-set abscesses grouped in comparatively large and well-defined but not very numerous clusters. Abscesses of the substance of the kidney are attended with various results. Sometimes their contents gradually concrete into a material like thick cream or moist plaster of Paris, consisting of disintegrated and fatty cells, molecular matter (partly earthy, partly oily), and cholesterine. In the most extreme examples of this kind of change the glandular substance of the kidney is hollowed out into a series of cavities, each one corresponding to a medullary cone and its associated cortical lobule, which are bounded externally, and separated from one another

and from the pelvis, by thin fibrous laminæ or dissepiments. Sometimes the abscesses open and discharge their contents into the infundibula; sometimes they extend beyond the limits of the kidney; and then in either case become indistinguishable, pathologically and clinically, from suppurative pyelitis.

Symptoms.—It would be almost impossible to lay down any definite rules for our guidance in reference to the diagnosis of the above affections. In a large number of cases the renal symptoms are necessarily more or less completely masked by the graver morbid conditions with which they are associated. Thus when renal abscesses result from embolism, pyæmia, or inflammation commencing in the pelvis of the kidney, the febrile or typhoid symptoms referrible to the primary malady may perhaps become in some degree aggravated, the prospects of amelioration somewhat diminished, the fatal event hurried; but probably nothing points specially to implication of the substance of the kidney. Even if the urine be scanty or contain blood, albumen, casts, pus-cells, or leucocytes, there is nothing to show that such conditions may not be the result of some other variety of renal inflammation. If large abscesses form, the symptoms and consequences will be those of suppurative pyelitis.

The *treatment* of these cases (if they call for treatment) does not differ from that of pyelitis.

IV. ACUTE BRIGHT'S DISEASE.

(*Acute Albuminous, Desquamative, or Tubal, Nephritis.*)

Causation.—This affection may be produced by many different causes. It may result from simple extension from the inflamed pelvis in pyelitis; it may be due to the influence of cantharides and other poisonous substances; and it frequently accompanies erysipelas, pneumonia, and such like grave inflammations, as also variola, measles, cholera, and other specific fevers. Its most important causes, however, are exposure to cold or wet, and the scarlatinal poison. It occurs also in pregnancy.

Morbid anatomy.—In acute Bright's disease the morbid process implicates in a greater or less degree all the renal textures and for the most part is generally diffused and involves both kidneys equally. (*a.*) The vessels—and more especially those of the medulla, the Malpighian tufts, and the stellate veins on the surface—become more or less deeply congested; and occasionally, undergoing rupture, blood escapes from them into the interstitial tissue, or into the Malpighian bodies and tubules. (*b.*) Proliferation of the nuclei in the membrane

Dropsy always comes on early, and soon becomes general and abundant, and although liable to vary somewhat in degree, is, on the whole, very persistent. With the increase of dropsy the surface gets more and more pale and waxy-looking. The gradual supervention of anæmia is generally a striking feature of the disease. This is in part apparent only and due to the presence of anasarca; but it is referrible mainly to actual deterioration of the blood determined in some degree by the daily abundant loss of albumen. As in ordinary anæmia, persons with delicate skin not unfrequently display a fallacious appearance of bloom in the cheeks. Together with the above symptoms patients suffer more or less from debility, restlessness, dyspnœa, loss of appetite, vomiting, and other symptoms of gastro-intestinal disturbance. Among the sequelæ or complications of the disease the following may be enumerated:—dropsical effusion into the pleuræ, lungs, or glottis; inflammation of the lungs, pleuræ, pericardium, or peritoneum; erythematous, erysipelatous, or gangrenous inflammation of the dropsical skin, more especially that of the lower extremities and external genital organs; hypertrophy of the heart—this is no doubt much less marked and less common than in chronic interstitial nephritis, but it certainly not unfrequently takes place when the disease is much prolonged; albuminuric retinitis—this again is a much rarer phenomenon in parenchymatous nephritis than in connection with the contracted granular kidney, yet it is occasionally observed, and may be so extreme and attended with so much hemorrhage as to render the patient almost absolutely blind. Lastly, uræmic symptoms are not infrequent, especially headache and sickness, followed by convulsions or coma.

Patients may of course recover from this disease, and the milder the attack the more likely is recovery to ensue. Favourable symptoms are: increase in the amount and diminution in the specific gravity of the urine, with disappearance of albumen, restoration of the functions of the skin, and subsidence of dropsy. Casts may continue in the urine after albuminuria has ceased. If a cure take place it is generally within six months. Recovery is in many cases fallacious; the patient improves to a certain point only—it may happen indeed that both albuminuria and dropsy disappear—and then after remaining pretty well for a time he has a relapse of which he dies; or the disease is prolonged by alternate remissions and relapses for several years. Death usually occurs in from three to twelve months, generally within six, either from extreme asthenia or from one of the complications which have been enumerated. But if we reckon the duration of the disease from the scarlatinal or other acute affection to which it may have been remotely due, it must be measured by years, and may be as much as ten or fifteen.

B. *Chronic Interstitial Nephritis. (Contracted Granular Kidney. Gouty Kidney.)*

Causation.—Chronic interstitial nephritis is more common in men than in women, and is met with almost exclusively in advanced life. It rarely occurs under forty years of age. The causes which determine it are not well understood. There is reason to believe that the tendency to it is sometimes hereditary. It is certain, too, that it is often combined with wide-spread changes of a similar kind in other organs. It is frequently associated with gout, and there is some obscure but undoubted connection between it and chronic lead-poisoning. Alcohol has certainly not the same tendency to produce this state of kidney that it has to cause cirrhosis of the liver. Nevertheless, there is good reason to believe that a small number of cases may be referred to abuse of drink. Again, it must, we think, be admitted, that the various inflammatory affections of the kidney already described, especially that originating in scarlet fever, and parenchymatous nephritis, tend in the course of years to produce a contracted granular condition of the organ, scarcely if at all distinguishable from that due to primary interstitial nephritis.

Morbid anatomy.—The *contracted granular kidney* is in distinct anatomical relation with the scarlatinal kidney, inasmuch as in both of them the morbid process commences in and implicates essentially the renal vessels and interstitial texture—the affection of the tubules and of their contents being secondary. Nevertheless, it cannot be pretended that they usually stand to one another in the relation of cause and effect. The typical contracted granular kidney is much smaller than the healthy organ, and occasionally not more than an ounce or half an ounce in weight. Its capsule is adherent, and on removal apt to carry with it small portions of the cortical substance. The surface is nodulated like that of a cirrhosed liver (the nodules being perhaps as large as hemp seeds) and of a deep reddish hue. On section the cortex is found to be much reduced in thickness, the medulla atrophied though in a less degree, and the texture of the organ generally dark coloured and dense. Cysts of various sizes, and in more or less abundance, are often observed studding the secreting structure, but more especially the cortical portion. On microscopic examination, the Malpighian bodies are seen to be largely changed, they are much reduced in size, their capsules are thickened and laminated, and their capillary tufts are welded into almost homogeneous lumps; the convoluted tubes are more or less atrophied, sometimes denuded of epithelium or lined with embryonic cells, sometimes stuffed with fatty contents, sometimes filled with hyaline casts, sometimes reduced to fibrous filaments or bands, scarcely distinguishable from the surrounding tissues, sometimes converted into microscopic cysts

lined or not with distinct epithelium; the loops of Henle and the straight tubes show less important changes, nevertheless their epithelium may present more or less fatty degeneration, they may be blocked up with hyaline casts, and occasionally some of them are converted into strings of cysts; the vessels, more especially the arteries, are much thickened, and at the same time reduced in calibre, while the larger ones are probably also atheromatous; and finally, the connective tissue of the organ is more or less extensively hypertrophied. This fibroid growth occurs mainly along the interlobular vessels, extending vertically from the surface to the junction of the cortex and medulla, and thence spreads horizontally so as to involve in the first place, and most importantly, the immediately contiguous Malpighian bodies and convoluted tubes, leaving the straight tubes which occupy the centres of the lobules comparatively free. This distribution explains both the special atrophy of the Malpighian bodies and convoluted tubes, and the granular condition of the surface of the organ—the depressions corresponding to the intervals between lobules, the elevations to the comparatively healthy central portions which contain the straight tubes. In the early stage of the disease, as in the early stage of cirrhosis of the liver, the kidney is little if at all diminished in size, the granulations on the surface are absent or only slightly developed, and a new growth of embryonic tissue may be observed in all those regions which subsequently undergo contraction and atrophy. The changes above described occasionally affect limited parts of otherwise healthy kidneys; and often manifest themselves ultimately in the attenuated and compressed kidney-structure, seen in advanced hydro-nephrosis. Further, as has already been pointed out, the fatty and granular conditions are not unfrequently associated. In which case the kidney is larger and more irregular in form than the simple granular kidney: its superficial granules are larger and paler; and its cortex is mot with whitish and yellowish patches, due to areæ of fatty degeneration.

The cystic kidney. — There is probably no essential distinction between the cystic kidney and that which has just been described, notwithstanding that the former may attain the bulk of a bullock's kidney, and the latter is usually unnaturally small. It has been mentioned that in the granular kidney obvious cysts are of common occurrence. There is no limit, indeed, to their size and number; the cause which produces them at one or two points in one case may be in general operation in another case, and hence, in place of half a dozen we may have hundreds or thousands; and instead of being no larger than a pea or marble many of them may attain the size of a pigeon's egg or a still greater bulk. In some of the more remarkable cases of this kind the kidney during life constitutes an easily recognisable tumour, and post mortem may have the aspect of a multilocular ovarian growth—consisting of little else than a congeries of cysts, probably measuring seven or eight inches in length, and weighing between two and three pounds. The contents of the cysts vary in character even in the same

case. They are sometimes limpid, sometimes thick and treacly, sometimes solid and jelly-like. They may be colourless or straw-coloured, or may present any tint between this and a dark brown or red. They may be clear, turbid, or opaque. They usually contain more or less albumen and the ordinary salts of the serum of the blood, but rarely if ever any special urinary constituents. The more viscid accumulations probably contain colloid matter. Among microscopic constituents are observed in different cases granular or fatty matter, disintegrating cells, decomposing blood, and cholesterine. Further, the cysts are often lined with pavement epithelium. There is reason to believe that they originate both in Malpighian bodies and in portions of renal tubules which, owing to inflammatory or degenerative changes, have been cut off from their connection with the rest of the secreting structure of the kidney. The abundant microscopic cysts observed in many cases of granular kidney certainly originate in convoluted tubules which, losing their epithelium and undergoing degeneration, become obliterated at points, distended in the intervals, and thus acquire a moniliform character. Another view, originally proposed by Mr. Simon, is that they arise in extravasated and overgrowing or dropsical renal epithelial cells.

It is a curious fact that in some of the most typical cases of cystic kidney similar cysts have been abundantly present in the liver.

Symptoms and progress.—The symptoms of granular kidney usually come on insidiously, and do not attract notice until the disease has made considerable progress. Indeed, it often happens that it is the occurrence of some complication that first calls attention to the presence of renal mischief. The patient, who had formerly enjoyed good health, gets thin, weak and anæmic without obvious cause; he suffers from dyspepsia, has loss of appetite, nausea, and perhaps actual sickness; he complains of shortness of breath and palpitation; he is liable to bronchitic attacks; his eyesight becomes impaired; and he probably notices that he makes more water than he was accustomed to make, and that he has to get up several times in the night to pass it. At length he seeks medical advice, and the urine is found to be of persistently low specific gravity, and probably to contain more or less albumen. In other cases attention is first seriously directed to the patient's condition in consequence of puffiness of the conjunctivæ or eyelids, or swelling of the legs or scrotum. In other cases, again, the first clear intimation of disease is the supervention of severe sickness or diarrhœa, or paroxysms of extreme dyspnœa, or œdema of the larynx, or the development of tremors not unlike those of paralysis agitans, or attacks of sudden blindness without visible optic changes and tending to remit, or uræmic convulsions, or an apoplectic seizure. The early symptoms, indeed, which are also in many respects those of the established disease, are multiform; and they are frequently masked by the presence of associated visceral lesions, more especially of the heart,

lungs, liver, intestinal canal, and brain. Nevertheless there are certain phenomena which are specially characteristic of the disease and indicate its presence, and to these we will now direct attention.

The *urine* is almost always abundant, pale, limpid and acid. Three or four pints, or even eight or ten, may be passed habitually during the twenty-four hours. Its specific gravity is low, varying from about 1003 or 1004 to 1010 or 1012, and it contains but little urea or other normal urinary constituents. Nevertheless the total amount of urea discharged daily is often fully up to the normal standard. Albumen, though generally present, is in small proportion. It is sometimes, however, in excess; sometimes wholly absent. Microscopic casts, too, are scanty, and may be readily overlooked; they are for the most part hyaline and granular. The condition of the urine may, however, vary: sometimes because the renal disease is not one of pure interstitial nephritis, sometimes as a consequence of temporary congestion or inflammation; and late in the disease it is apt to become scanty, of comparatively high specific gravity, and at the same time deficient in urea. *Dropsy*, so common in other forms of Bright's disease, is often absent in this; and even when present, is for the most part slight and variable and of late occurrence. It is sometimes limited to the conjunctivæ. But occasionally it becomes extreme; and especially in those cases where also the urine becomes scanty, and mainly, therefore, towards the close of life. It is in association with the contracted granular kidney that *thickening of the walls of the small arteries* and *hypertrophy of the heart* mainly occur. The degree of these changes is generally proportioned to the length of time during which the renal disease has been in progress, and to the degree to which the kidneys have shrunk. The hypertrophic condition of the heart is generally revealed by its heaving pulsation and increased area of dulness; and the general arterial affection by heightened tension of the larger arteries and incompressibility and prolonged tidal wave of the pulse. At a late period, however, the heart becomes enfeebled.

In association with the continuance of the conditions here discussed, the patient becomes more and more enfeebled and incapable of exercise, and probably emaciates; he complains of dryness of mouth or thirst, loss of appetite, flatulence and nausea, and especially at a late period of constant and distressing sickness; his bowels probably are variable; he has attacks of difficulty of breathing, coming on mainly at night-time, and presenting a good deal of resemblance to those of ordinary asthma; he suffers also from headache, giddiness, or sense of oppression or weight, is often disposed to somnolence, and becomes apathetic. His skin is harsh and unperspiring, and his complexion probably sallow; but he rarely becomes distinctly anæmic, or suffers from lumbar pain.

Many complications are apt to arise in the course of the disease and especially towards its fatal close. Inflammatory affections are com-

mon, more particularly inflammation of the pericardium and pleuræ, œdema of the glottis, bronchitis, and pneumonia. Functional nervous disorders also are extremely common, and among the most characteristic of them. They comprise (besides headache, somnolence, tremors, sickness, and, we may add, delirium) epileptiform attacks or coma, which are frequently preceded by these or other nervous phenomena, and attacks of amaurosis without obvious affection of the eyes, which are apt to come and go, but after a time to end in absolute blindness. Affections referrible to the circulatory organs, again, are of frequent occurrence; epistaxis and bleeding from the stomach and bowels and other mucous membranes are often observed; retinal hemorrhage or albuminuric retinitis is far more common in this than in any other form of kidney disease, and is not unfrequently one of the earliest indications of its presence; in addition to the characteristic thickening of the smaller arteries, atheromatous and calcareous changes of the arterial system are commonly present, and partly from this cause, partly from excessive blood-pressure within the vessels, hemorrhagic effusion into the substance of the brain is very liable to occur; again, thrombi are apt to form in the pulmonary arteries, leading to pulmonary apoplexy, and also in the systemic vessels and cavities of the heart. Lastly, it may be observed that granular degeneration of the kidneys is occasionally associated with similar disease in the liver and other organs, and that dysenteric ulceration not unfrequently supervenes.

Chronic interstitial nephritis is essentially a disease of long duration. It may certainly continue for ten years or more. The causes of death have been sufficiently indicated in the last paragraph; but the most frequent cause is uræmic poisoning, sometimes with convulsions, more frequently with coma.

C. *Treatment of chronic Bright's Disease.*

The treatment of chronic parenchymatous nephritis is essentially the same as th of the acute disorder. Abstraction of blood, however, is less likely to be required. Perspiration should be promoted by the measures previously discussed or by the Turkish bath. The bowels should be kept freely open by saline purgatives. Diuresis should be solicited by bland drinks, alkalies, and digitalis. Moreover the stimulant diuretics, broom, juniper, squill, and nitric ether, which are unsuitable in the acute disease, may be given with advantage here. The rapid development of anæmia points significantly to the use of iron, and there is no doubt that ferruginous preparations, and especially the perchloride of iron, are more valuable in this than in any other form of nephritis.

In dealing with cases of granular disease the incurability of the lesion must not be forgotten. If no special symptoms are present it may be desirable to promote the action of the skin by the wearing of

flannel and the use of baths, to keep the bowels open by occasional mild purgatives, to give tone to the system by the employment of iron in combination with vegetable bitters, and to support strength by wholesome nourishing diet, not superabundant in quantity, and comprising a small proportion only of animal food. Late in the disease, when the urine becomes scanty, and dropsy or indications of uræmia present themselves, the promotion of urine and drastic purgation are called for.

In all forms of chronic Bright's disease special symptoms require to be treated as they arise. For dropsy the most effectual remedial measures have already been enumerated. But when the accumulation of fluid is extreme it may need to be removed by surgical means. For this purpose 'acupuncture' or the puncture of the skin with a needle in several places just above the ankle, or in the scrotum or some other dependent part, may be performed, or incisions may be made in the same localities. Or better still, a fine trocar and cannula (according to Dr. Southey's suggestion) may be inserted, and retained *in situ* for some days without inconvenience. It must not be forgotten, however, that erythema and sloughing are apt to follow this slight operation, apparently from the irritating effects of the escaping serum on the integument over which it flows. To avert this danger it is well to anoint the surface previously with sweet oil. Uræmic poisoning may often be obviated or cured by the use of drastic purgatives. When convulsions are present the inhalation of chloroform often affords relief. It is needless to lay down rules with regard to the treatment of uræmic asthma, uræmic dyspepsia, and the many other complications of chronic Bright's disease.

In all cases when either convalescence is in progress or the symptoms are of a chronic character hygienic and tonic treatment is of the highest importance, and especially residence in a genial climate; moderate out-of-door exercise stopping short of fatigue, wholesome unstimulating diet and early hours are likely to be beneficial.

In conclusion it may be pointed out that in all varieties of Bright's disease the use of certain drugs is fraught with danger. Of these, opium, mercury, and cantharides are the most important examples.

VI. CONGESTION OF THE KIDNEY.

Causation.—Congestion is present in a greater or less degree in all inflammatory affections of this organ, in many febrile diseases, and as a consequence of the action of certain irritant poisons. The form of congestion, however, which we are now about to consider is that passive congestion which arises in the course of obstructive cardiac and pulmonary diseases.

Morbid anatomy.—This condition is characterised in its early stage by congestion, enlargement, and softening of the kidneys. The veins are especially overloaded, and more particularly the stellate veins of the outer surface and those of the medulla. If the congestion continues, induration takes place, due to slow increase of the interstitial fibrous tissue of the organ, and ultimately more or less atrophy of the Malpighian bodies and of the other secreting elements, including fatty degeneration of the cortical epithelium.

Symptoms.—In this affection there is not generally much to attract attention to the condition of the kidneys beyond scantiness of urine, and the presence in it of albumen, and occasionally of blood, and of casts which are hyaline or granular, or formed in part or wholly of disintegrating blood-corpuscles. The albumen is generally scanty, but sometimes it is very abundant. The specific gravity is usually high. Inflammation readily supervenes. As a rule the general symptoms due to renal congestion are so inextricably intermingled with those of the disease to which the congestion itself is due, and which in fact they closely resemble, that they do not admit of separate recognition. Occasionally, however, uræmic poisoning and other common consequences of Bright's disease are distinctly developed.

The *treatment* is mainly that of cardiac or pulmonary disease, as the case may be; and the employment of remedies calculated to relieve renal congestion, more especially purgatives, diaphoretics, and unirritating diuretics.

VII. TUBERCULAR DISEASE OF THE KIDNEY.

Morbid anatomy.—For the most part tubercles are developed in the kidney as a comparatively late event of general tuberculosis, give rise to few or no symptoms, and are of little clinical importance. Sometimes, however, tuberculosis is primary in the kidneys, or at all events may be found post mortem to be as far advanced in these as in other organs; and under such circumstances the renal affection is a material, possibly the chief, item of the patient's illness. When tubercles are abundant and far advanced in the kidneys, they are probably always present also in the mucous membrane of the urinary organs—pelves, ureters and bladder—and even in the vesiculæ seminales and testes, or in the uterus and Fallopian tubes.

Tubercles appear in the first instance as grey granulations scattered mainly in the cortex, but occurring also in the medulla. It is in this form that they are generally discovered. After a while they increase in number and in size, coalesce into larger masses, undergo caseous degeneration, soften, and perhaps suppurate.

Under such circumstances the kidney may become considerably enlarged, riddled with cavities of various sizes containing either cheesy matter, tubercular detritus, or pus, and studded in the intervals with unsoftened tubercles. The destructive process may proceed so far, indeed, that the whole of the secreting structure becomes converted into a series of large tubercular cavities, of which one corresponds to each cone and its associated portion of cortex. These cavities may either communicate by ulceration with the pelvis of the kidney, or remain separated from it, in which case the contents change after a time into a creamy or mortary material like that already adverted to as due to the drying up of ordinary renal abscesses.

Tubercles affect the mucous lining of the pelvis and ureter in precisely the same way as they affect other such surfaces; miliary granulations appear in scattered groups in the substance of the membrane, become caseous, and then disintegrate, producing shallow circular pits, the surfaces of which generally present more or less tubercular detritus. The junction of neighbouring pits leads to a greater or less extent of superficial destruction, and the formation of an irregular sinuous-edged ulcer. There is generally also more or less thickening of the subjacent and surrounding tissues. The pelvis generally becomes dilated. The ureter, on the other hand, usually gets narrowed or even obliterated.

Symptoms and progress.—In considering the symptoms of renal tuberculosis it is almost impossible to separate them practically from those due to the associated affection of the urinary passages; and it is not difficult to surmise what the main symptoms of these united conditions must be. They are, indeed, essentially those of chronic pyelitis; and comprise pain and tenderness in the loins, tumour possibly, irritability of bladder with perhaps pain or scalding in passing water, and the discharge of urine containing a greater or less abundance of mucus, but more generally pus, and occasionally it may be a little blood together with débris of tissue. The urine is said generally to be scanty, and not to contain renal casts; but the discovery of casts must not be taken to disprove the presence of renal tubercle, nor is the scanty secretion of urine by any means constant. The reaction of the urine is for the most part slightly acid; but, as in cases of non-specific pyelitis and cystitis, is apt to become alkaline from decomposition. The course of renal tuberculosis is essentially unfavourable; for, independently of the slow but sure destruction of the renal tissue, which must ultimately lead to a fatal result, the local disease sooner or later becomes associated with the development of tubercles in other organs. The symptoms and progress of any case will necessarily vary according as the phenomena due to the urinary apparatus or those referrible to implication of other organs preponderate. It is important to bear in mind that the symptoms of renal tubercle and of tubercle of the urinary passages are not in any sense specific; and that their dia-

gnosis must mainly rest on the detection of similar disease in the lungs or elsewhere.

Treatment.—The treatment of renal tubercle comprises that of tuberculosis and that of chronic pyelitis.

VIII. SYPHILITIC DISEASE OF THE KIDNEY.

Lardaceous infiltration of the kidney is a common attendant on advanced syphilitic cachexia; but specific syphilitic affections of this organ are exceedingly rare. Very few cases of distinct gummatous tumours are recorded; but occasionally, on examining the bodies of persons who have suffered from syphilis, and in whom gummata or their remains are visible in other organs, the surface of the kidney presents well-marked linear and stellate depressions corresponding to localised induration and atrophy of tissue. These are most likely of syphilitic origin; but have probably never given any indication during life of their presence. As regards diagnosis, all that can be said is that when patients with advanced syphilis present symptoms indicative of renal disease, they are probably due to lardaceous infiltration, but may possibly result from the formation of gummata.

IX. MORBID GROWTHS OF THE KIDNEY.

Morbid anatomy.—Several varieties of tumour are met with in the kidney. Fibromata sometimes attain a large size, so large, in fact, as to be easily recognisable during life. But they do not, so far as we know, produce any inconvenience or symptoms beyond such as depend on their situation and bulk. The only tumours that have any practical interest are those possessing malignant properties.

1. *Lymphadenoma* generally occurs in the kidney as a secondary or late event in the gradual generalisation of the disease. The renal growth occurs in patches which at the surface of the organ are circular, pale, and scarcely elevated, and are prolonged into its substance in a wedge-like form. Other patches are wholly imbedded in the substance of the organ. On microscopic examination the cells which constitute the growth are found to occupy the intertubular spaces only—the tubules and Malpighian bodies, which may remain healthy, being surrounded and separated from one another by them.

2. *Sarcoma* has occasionally been observed in young children. It is probable, however, that many infantile renal tumours, which have

been described as cancerous, were really examples of sarcoma. The disease seems to attack one kidney only, to cause enormous enlargement of the organ, and to be undistinguishable during life from cancer.

3. *Carcinoma* may be primary or secondary. When secondary it rarely attains large dimensions; when primary it is generally limited to one kidney, and this soon forms an enormous tumour. Renal carcinoma is, almost without exception, of the encephaloid variety, and usually highly vascular. It commences in the form of one or more isolated tumours, which gradually invade the adjacent renal structure until the greater part or the whole of the organ is involved. While this process is going on the kidney becomes enlarged, but still probably on section presents the outlines of its original divisions. With the continuance of the growth, however, all traces of renal structure get obliterated, and the kidney is converted into a simple carcinomatous mass, still probably presenting the form of the healthy organ, but attaining the size it may be of a cocoa-nut or large melon, and weighing several or many pounds. In the progress of its growth it becomes adherent to surrounding tissues and organs which may then be involved by continuity; and it develops nodular, papillary, or even villous outgrowths into the cavity of the pelvis and infundibula. The carcinomatous kidney is of course liable to all those changes which generally characterise carcinoma; it presents consequently, in addition to growing tissue, patches or networks of caseous and fatty degeneration, hemorrhagic effusions, and tracts of liquefaction. The ureter is not unfrequently involved, and even when not distinctly cancerous, is apt to become thickened and more or less completely occluded.

Symptoms and progress.—The recognition of secondary growths in the kidney, whether they be lymphoid, sarcomatous, or cancerous, is a matter of little importance; and that of primary carcinoma is, until the disease is far advanced, often extremely difficult. The chief circumstances to be taken into consideration in forming a diagnosis are: first, the very gradual development of symptoms; second, the frequent discharge of blood in quantity with the urine; third, the gradual formation of a tumour in the situation of the kidney; fourth, the appearance of secondary cancerous growths; and, fifth, the occurrence of progressive emaciation, debility, and cachexia. The symptoms, in fact, are mainly those common to cancerous growths, together with such as depend on the situation of the tumour, and modification of the urinary secretion. Of these three symptomatic groups the latter two only call for further remark. The development of cancerous tumours is sometimes painless; sometimes, on the other hand, the patient suffers from frequent paroxysms of the most intense agony; and generally sooner or later there is manifest local tenderness. The tumour is characterised by originating deep in the lumbar region; and (as it grows and fills more or less of the abdominal cavity) by its position, by its fixation, by its general rounded form, and very importantly by the

fact that it is almost invariably crossed by the ascending or descending colon, the presence of which may often be seen, and always recognised by percussion. The veins in the abdominal walls on the affected side are often much dilated; and not unfrequently from the pressure of secondarily affected glands, œdema of the corresponding lower extremity or of both extremities comes on. A cancerous kidney generally feels hard, but is sometimes yielding, and may be so soft as to give a deceptive sense of fluctuation. It often enlarges so greatly as to fill its own side of the abdomen, and occasionally not only fills this, but encroaches to a great extent on the opposite side. It has been pointed out that the urine often contains blood. Hemorrhages occur at irregular intervals, and are sometimes so profuse and frequent as to blanch the patient. It must not be forgotten, however, that in many cases no hemorrhage whatever takes place; and that in many the urine from first to last is perfectly healthy. The latter circumstance is in great measure due to the fact that the ureter of the affected side often becomes impervious even at an early stage of the disease. Cancer-cells rarely if ever find their way from the kidney into the discharged urine, and, even if present there, would probably be undistinguishable from the epithelial cells of the bladder. The affection with which renal cancer is most apt to be confounded is renal calculus associated with pyelitis and distension of the cavity of the kidney.

The liability to error is increased when gravel or small calculi are, as is not uncommon, present in the pelvis of the cancerous organ. In the early stages of cancer, indeed, it is often impossible to discriminate between it and calculous pyelitis. Later on its recognition is more easy, but then the diseased organ is apt to be mistaken for an ovarian, splenic, or hydatid tumour.

Treatment.—In the treatment of renal cancer there is nothing to be done beyond endeavouring to relieve the patient's symptoms. Opiates are here invaluable.

X. PARASITIC AFFECTIONS OF THE KIDNEY.

Animal parasites seldom affect the urinary organs, at any rate in temperate climates. The *Strongylus gigas* and *Pentastoma denticulatum* have been so rarely observed in the kidney that no practical interest attaches to them. Hydatids are much more frequently met with there, and the *Bilharzia hæmatobia* is common in the vessels of the urinary organs in certain tropical countries. Of the *Filaria sanguinis hominis* in relation to the urinary organs, we shall speak under the head of chyluria.

A. *Hydatid cysts* of the kidney are far less common than hydatid cysts of the liver. Still many authentic cases are on record. The anato-

mical characters, progress, and consequences of renal hydatids present nothing distinctive beyond the facts that the enlarging cysts have the usual situation and connections of renal tumours, and that they not unfrequently rupture into the pelvis of the kidney and discharge their contents with the urine. It must not be forgotten, however, that hydatid tumours may originate in the sub-peritoneal tissue in the neighbourhood of the kidney; and that both these and hydatids occupying other situations may open into the pelvis of the kidney or into the bladder. If suppuration takes place in the cyst of a renal hydatid, the case becomes essentially one of abscess of the kidney.

The *treatment* of renal hydatids is the same as that of hydatids of the liver.

B. The *Bilharzia hæmatobia* seems to be the cause of an endemic form of hæmaturia, common in Egypt, at the Cape of Good Hope, and elsewhere. The parasite is of a worm-like form, and three or four lines in length. The female is longer than the male, and filiform; the male is comparatively thick, and in the act of copulation encloses the female in a gynæcophoric canal. It is supposed to be swallowed with the food, and thus to gain entrance into the system by the stomach, but it specially inhabits the mesenteric veins and those of the large intestine, bladder, ureter, and pelvis of the kidney. Its presence in the small veins of the urinary organs gives rise to lenticular patches of inflammation in the mucous membrane, which yield mucus and sometimes blood, ulcerate, and discharge shreds of tissue charged with ova. The patient consequently presents more or less irritability of bladder, and passes urine containing these several ingredients. He often falls also into a state of anæmia and debility. When the ureter or renal pelvis is affected, obstruction to the flow of urine may arise, pyelitis and hydro-nephrosis may ensue, and the patient's symptoms may hence assume a more serious character. The ova may form the nuclei of urinary concretions. The presence of these creatures in the mucous membrane of the large intestine is apt to produce dysenteric symptoms, which, however, are rarely severe. The recognition of the disease depends on the discovery of the ova in the urine.

Treatment.—It is doubtful if vermifuge medicines are of any efficacy in this affection; injections, however, into the urinary bladder may act beneficially on so much of the disease as involves that viscus. The forms of injection which are beneficial in the treatment of thread-worms naturally suggest themselves—namely, bitter infusions, or solution of perchloride of iron. Dr. J. Harley prefers solution of iodide of potassium. For general treatment, tonic remedies are indicated.

XI. LARDACEOUS DEGENERATION OF THE KIDNEY.

Causation.—The causes of lardaceous degeneration of the kidney are the same as those of lardaceous degeneration of the liver and other organs; and indeed the liver, kidneys, and spleen are generally concurrently affected.

Morbid anatomy.—The lardaceous kidney increases in size with the amount of degeneration present, and may attain a weight of twelve ounces or more. When the disease is little advanced it is apt to escape recognition by the naked eye; when, however, it reaches a high degree, the organ is somewhat waxy, pale, and homogeneous in texture, and presents a slight degree of translucency. The morbid change usually commences in the vessels of the Malpighian tufts, but very soon spreads from these to the afferent and efferent vessels, the intertubular plexus, the interlobular arteries, and the vasa recta. The hyaline walls of the urinary tubes and Malpighian bodies also suffer, but for the most part somewhat later than the vessels. The degeneration here is always most advanced in the large collecting tubes, and diminishes in degree as one proceeds from these to the Malpighian capsules, which in fact generally remain unaffected. The epithelial cells are rarely if ever involved, but are often granular, and even distinctly fatty. Lardaceous change is apt to be superadded in the course of ordinary fatty and granular degeneration of the kidney, in which case the several morbid conditions are variously intermingled. Waxy casts may generally be detected in both the cortical and the medullary tubules.

Symptoms and progress.—The presence of lardaceous change in the kidney does not necessarily give rise to any special symptoms until the disease is far advanced. The symptoms then, if not of themselves distinctive, become distinctive when the history of the patient, the condition of his other viscera, and his general state are all taken into consideration. They are mainly as follow: the urine is increased in quantity, pale, of low specific gravity, and poor in urea; it contains more or less albumen, and casts which have not necessarily any special character, but are often waxy, yet rarely if ever lardaceous; micturition is generally frequent; there is often some degree of anasarca, but it is not usually abundant; and the patient is anæmic. In these respects the symptoms are not unlike those due to the granular kidney, but the heart does not become hypertrophied; there is absence of arterial tension; there is little tendency to uræmic poisoning; and although patients often suffer from serous inflammations, inflammation of the lungs, diarrhœa, vomiting, and hemorrhages, these complications are not distinctly referrible to the kidney disease, but are due in part or wholly to the presence of associated visceral lesions.

The *treatment* of lardaceous kidney is involved in the treatment of the affections to which it is secondary. Dropsy and other consequences, when they are sufficiently serious to demand separate attention, must be treated according to the principles already enunciated under the head of chronic Bright's disease.

XII. GRAVEL AND RENAL CALCULI.

Causation and morbid anatomy.—The presence in the urine, or the deposition from it, of uric acid and urates, of oxalate of lime, or of phosphates, is occasionally observed in various morbid conditions of the system, and even in states of apparently good health. Such occurrences rarely if ever call for medical interference.

Occasionally, however, the appearance in the urine of one or other of these, or of other rarer crystalline matters, persists for some time, or becomes habitual. If under such circumstances symptoms of ill-health manifest themselves, medical treatment is obviously demanded; and, indeed, even in the absence of symptoms, the danger of the formation of urinary calculi is so great that, if the peculiarity of the urine be recognised, it should, if possible, be counteracted.

The amorphous urates are sometimes found deposited in the renal tubules, but this is probably a post-mortem phenomenon only. Urate of soda, in stellate masses of acicular crystals, is now and then discovered imbedded in the substance of the kidney; uric acid also, in solitary or clustered crystals, is occasionally detected within the tubules, and again, in the form of small calculi, is sometimes found loose in the cavity of the kidney or adherent to the mammillary processes. The same may be said in regard to the infinitely rarer xanthine and cystine concretions.

Octahedra and dumb-bells of oxalate of lime, singly or in groups, may be met with in the urinary tubules, and occasionally also form small calculi, which lie loose or adherent within the cavity of the kidney.

The phosphates are rarely deposited, except in ammoniacal urine, and as a consequence of the decomposition of that fluid; they are, therefore, seldom, if ever, detected in the kidney except as secondary deposits around nuclei of other matters. Carbonate of lime, however, though much less frequently forming a urinary deposit, is occasionally met with in the form of small laminated globular concretions, either imbedded in the substance of the kidney, or free in its pelvis.

The minuter concretions above described are sometimes discharged with the urine in considerable abundance, constituting what is called 'gravel.' Small calculi, from the size of a pin's head to that of a

horse-bean, are also not unfrequently transmitted, with more or less delay, along the ureter to the bladder, and thence into the chamber-pot. Sometimes a solitary calculus is thus discharged, and there is never any recurrence; sometimes large numbers of calculi are discharged at intervals. In other cases these bodies remain in the renal cavity, gradually grow there, and finally, perhaps, form a complete cast of the pelvis, infundibula and calyces; or a considerable number of small calculi may become aggregated into that form.

The presence of calculi in the kidney generally leads to more or less pyelitis, and probably at length to abscess, hydro-nephrosis, or some other serious consequence.

Symptoms and progress.—The symptoms of 'gravel' are: pain of an aching or burning character in one or other lumbar region or side of the abdomen, probably shooting down to the testis or labium, and along the inner aspect of the thigh; frequent desire to micturate; soreness or cutting pain during micturition, especially at the end of the urethra in passing the last few drops; and nausea and sickness. At the same time the urine is generally clear, though it may deposit a greater or less abundance of a sand-like sediment, or show microscopic aggregation of crystals, with epithelial scales. The pain may, of course, affect both sides; and the patient's complaint may be limited to the lumbar or abdominal pain or uneasiness.

A renal calculus may never reveal its presence by symptoms, and may even lead to the disorganisation of the kidney without the least suspicion of disease having ever been excited. The special indications of the presence of a calculus are: first, the occasional occurrence of aching or burning pain in the situation of the kidney, resembling, but probably more severe than that attending the passage of gravel; second, the occasional discharge of bloody urine; and, third, the facts that the nephralgia and hæmaturia are often induced by jolting, jumping, and other forms of exercise, and that the pain may occasionally be relieved by change of posture. This becomes much more intense when the calculus enters the ureter, and continues intense so long as the stone is passing along that canal. The pain of renal or ureteric calculus may be traced along the ureter, shooting thence into the loin, radiating throughout the abdomen, and especially extending to the thigh, and labium or testis, which last often becomes retracted; it is attended with nausea and vomiting, and not unfrequently with rigors and faintness. It is often increased by the patient's voluntary attempts to flex the thigh on the abdomen. Further, tenderness may exist in the loin and along the course of the ureter. The pain due to the transmission of a calculus begins suddenly, and ends suddenly in a few hours, or after intermissions in the course of a few days, in consequence of the stone becoming either arrested in its course or discharged into the bladder. It need scarcely be said that the microscopic investigation of the urine often throws important light on the

diagnosis of cases which come under treatment; and further that, when one kidney has already been destroyed or rendered useless, the impaction of a stone in the opposite ureter may cause fatal suppression of urine. The symptoms of pyelitis, renal abscess, and hydro-nephrosis, which are frequent accompaniments or consequences of renal calculus, are elsewhere discussed.

Treatment.—The treatment of gravel and of renal calculus is for the most part identical with that of pyelitis—a subject which has already been fully considered. The pain, however, in so-called 'nephritic colic' is generally so much greater than in other forms of pyelitis that opium, rest, and local measures are all more urgently needed. Opium, especially, is our sheet-anchor. As valuable adjuvants we may enumerate purgatives, copious enemata, ice-bags hot applications or cupping to the loins, and especially the hot bath. Belladonna is sometimes useful when opium fails; and, when a calculus is descending, may be of special service in relaxing the spasmodic action of the ureter which takes place around it, and impedes its progress. The removal of a renal calculus by operative measures can scarcely be attempted unless the kidney be at the same time in a state of suppuration and have formed a manifest tumour.

In the intervals between the acute attacks, which, from their severity, call for special treatment, the question of the removal of the conditions on which the gravel or calculi depend presents itself for consideration. Our action here must be determined mainly by the nature of the sabulous matter which is habitually discharged.

If uric acid crystals or gravel are passed, it is certain that the urine is abnormally acid, and the exhibition of alkalies is demanded. The carbonate, acetate, and citrate of potash are probably the best for the purpose; and they should be given in such quantities as to render the urine constantly alkaline. Dr. W. Roberts has shown that the alkaline carbonates slowly dissolve uric acid calculi, and that the urine may be rendered and kept sufficiently charged with carbonate to produce this effect by administering to the adult forty or fifty grains of the acetate or citrate in 3 or 4 oz. of water every three hours. And hence he recommends that, if there be reason to believe that uric acid calculi are present in the kidney, the patient should be submitted to this alkaline treatment. Phosphate of soda also dissolves uric acid, and Dr. Golding Bird recommends its use in scruple or half-drachm doses. It is important at the same time to have regard to the patient's mode and habits of life and to any morbid conditions which may be present. Thus, valuable indications for treatment may be furnished by the fact that he is a *bon vivant* or of sedentary habits, or that he suffers from indigestion or gout.

Cystine and xanthine deposits and calculi may be treated in the same manner as those of uric acid.

Oxalate of lime, like uric acid, is generally precipitated in acid

urine, and indeed they are not unfrequently associated. Its presence in small quantity is often dependent on the use of certain articles of diet; when it is more abundant and persists, the patient frequently suffers from indigestion, or presents symptoms of mental depression. The direct treatment of oxaluria is not generally very efficacious. The patient's general health should be improved by tonic medicines and general tonic treatment, and by abstinence, so far as possible, from vegetables containing oxalate of lime, and from sugar and other substances which are readily convertible into oxalic acid. Nitro-muriatic acid is often recommended; while, on the other hand, alkalies seem sometimes to be efficacious.

Persistent alkalinity of urine from the presence of the fixed alkalies is rare and in itself not very important. It generally seems to be associated with some degree of ill-health and cachexia, and may be taken to indicate the need of tonic treatment and of generous diet. Mineral acids, especially the nitro-muriatic, and perchloride of iron, are valuable remedies.

Alkalinity from the presence of carbonate of ammonia is a much more serious matter. This always results from decomposition of the urine in the urinary channels, is indicative of cystitis or pyelitis, and necessarily leads to the deposition of crystalline phosphates. For the relief of this condition we must have recourse to the usual treatment of cystitis.

XIII. HYDRO-NEPHROSIS AND ATROPHY OF THE KIDNEY.

Causation and morbid anatomy.—Whenever any permanent impediment to the flow of urine occurs—whether it be in the urethra, bladder, or ureter; whether it be due to a calculus or any other obstacle within; or to some affection of the walls themselves, such as thickening and contraction, valvular folds or paralysis; or to pressure from without, caused by ovarian, uterine, or other tumours—the cavities above the seat of obstruction dilate and their parietes thicken, and at the same time the kidney structure becomes expanded and attenuated. The condition known as hydro-nephrosis results. If complete obstruction take place, excretion of urine continues for a time; but its accumulation causes more and more distension of the renal cavity, and more and more pressure on the renal structure, until at length the function of the organ ceases absolutely to be performed. In this case, equally with that in which pus accumulates, those portions of the renal cavity whose lining membrane is least resistant expand most; and consequently, while the pelvis and infundibula alter comparatively little, the calyces dilate until they form a series of sub-globular cavities sur-

rounded and separated from one another by atrophied kidney structure, and communicating by separate and comparatively small orifices with their respective infundibula. When the obstruction is partial, as well as during that period of total obstruction in which the renal elements are still excreting urine, this fluid changes in quality; it becomes less and less rich in solid constituents, pale, watery, and of low specific gravity, but remains for the most part devoid of albumen. Subsequently to the cessation of the proper urinary discharge, the fluid in the cavity may still increase in quantity and still undergo changes. Thus in advanced hydro-nephrosis it is generally watery but still containing traces of the urinary solids; it is often albuminous; sometimes charged with decomposing blood; sometimes more or less glairy and colloidal; and occasionally purulent. After a kidney has become completely hydro-nephrotic and ceases to secrete urine, various consequences may ensue. In some cases it remains for a long while almost stationary. In some the contents become slowly absorbed and the atrophied tissues shrink and indurate until at length a small, hard, lobulated cystic body, weighing perhaps from a drachm or two to half an ounce, remains. In other cases the dropsical kidney gradually enlarges until it forms a tumour several times the bulk of the healthy organ, and occasionally sufficiently large to fill at least one-half of the abdomen. Hydro-nephrosis from total or partial, and often valvular, obstruction of the ureter is not unfrequently congenital, and at the same time double, and hence hydro-nephrotic tumours are not altogether uncommon in new-born babes and young children.

Symptoms and progress.—As a rule the changes above described creep on (if no inflammation ensue) without producing symptoms, and without, therefore, calling for treatment. It is comparatively rare for the hydro-nephrotic kidney to become so large as to excite observation, still more rare for it to become so large as to exert by its pressure on surrounding organs any deleterious influence. But in these cases alone is diagnosis needed or indeed possible. The elements on which an accurate opinion must be based are the history of the case, the situation and relations of the tumour, its characters as to form resistance and fluctuation, and the constitutional symptoms which are associated with it. In addition to these there is a symptom of rare occurrence, but very characteristic when it does occur, and peculiar to cases of incomplete obstruction—namely, the occasional rapid but temporary subsidence of the tumour, attended with a sudden increase in the quantity of urine passed, and some change in its quality. In some cases the dilated organ suppurates, and a renal abscess with the usual symptoms of that condition supervene.

A hydro-nephrotic tumour is liable to be confounded with carcinoma and hydatids of the kidney or neighbouring parts, and with ovarian cysts. It is rarely fatal, except in those cases in which it is double, or where it is associated with other maladies, or where, from its bulk

and interference with other organs or from suppuration, slow exhaustion ensues.

Treatment.—The treatment is entirely surgical. If manipulation fail to drive the contents into the bladder, paracentesis may become necessary. To prevent danger from escape of fluid into the peritoneum this operation should be performed behind the line of colon which crosses the tumour.

XIV. MISPLACED AND MOVABLE OR FLOATING KIDNEYS.

Causation and morbid anatomy.—Misplacements of the kidneys are chiefly important in relation to the diagnosis of abdominal tumours. Sometimes, as a congenital peculiarity, one or both kidneys, instead of occupying their usual site, lie upon the brim of the pelvis. Sometimes one or both of them, though otherwise normally placed, are attached to the lumbar region by a peritoneal duplicature or mesonephron analogous to the mesentery, or lie freely movable in the lax retro-peritoneal connective tissue which surrounds them. Mobility of the kidney is said to be much more common in women than in men, and on the right than on the left side. Its cause is obscure. It may perhaps in some cases be a congenital defect; but it seems also occasionally to follow upon parturition, and possibly then to be connected with that general laxity of the abdominal parietes which parturition causes.

There is still considerable doubt on the part of many with respect to the occurrence of floating kidney. It is certain there is little post-mortem evidence in its favour; and that uterine fibroid, and other tumours have been mistaken during life for floating kidneys by competent observers. On the other hand, it must be borne in mind that such kidneys are probably never a cause of death. The question has, however, been set at rest affirmatively by a recent inquiry made for the Pathological Society.[1]

Symptoms.—The floating kidney projects more than natural (assuming an oblique position with the upper end pointing forwards and inwards), and is freely movable within narrow limits under the abdominal parietes. It may usually be perceived somewhere in the hypochondriac or umbilical region, between the navel and cartilages of the ribs; and if on the right side is apt to make its appearance just below the liver and to be mistaken for an hepatic tumour. If it be grasped, as it sometimes can be, a sickening sensation is produced, similar to that which results from squeezing the opposite loin; and sometimes a distinct falling in of the corresponding lumbar region with increase of

[1] Path. Trans. vol. xxvii. p. 467.

resonance may be clearly recognised. From its prominent and pendulous condition it is unduly exposed to pressure or injury, and consequently is apt to become more or less painful, tender, and swollen.

Treatment.—When a movable kidney is painful, rest, local applications, and the internal use of sedatives may be requisite. To protect it from injury, and at the same time to replace it to some extent, an abdominal belt may be worn with a concave pad beneath it adjusted to the form and position of the kidney.

XV. CHYLURIA. (*Chylous Urine.*)

Causation and symptoms.—This affection was first recognised and described by Dr. Prout, but since his time has been pretty frequently met with and investigated by other observers. It is characterised for the most part by the occasional or constant discharge of urine, which is milky when passed, coagulates on standing into a tremulous mass resembling blanc-mange, and then, becoming again liquid, furnishes a creamy scum and a pinkish or brownish sediment. The urine has, in fact, exactly those characters which would result from the admixture in varying proportions of normal urine and normal chyle. It presents the ordinary urinary constituents, but in diminished proportion to the whole bulk of fluid. And it also contains fibrine, the presence of which explains its spontaneous coagulability; albumen, as may be shown by the usual tests; fat in a molecular form, like the fat of chyle, the presence of which accounts for the milky character of the fluid when passed, and for the creamy scum; leucocytes; and occasionally red corpuscles, to which the coloured sediment is partly attributable. No casts, however, are ever detected in it; nor indeed is there any other evidence that the chylous material comes from the kidney. Further, it often happens in these cases that the urine which is passed is not milky, although probably presenting in all other respects the peculiarities which have been enumerated. It is, in fact, lymphous, and not chylous; there is no fat, and the coagulum is transparent like ordinary calves'-foot jelly. The presence of fat is, in some instances, observed mainly in the morning's yield; more commonly it characterises the urine passed shortly after meals.

Chyluria appears to be more common in tropical than in temperate climates, more frequent in adults than in children, and in females than in the opposite sex.

It manifests itself, for the most part, suddenly, is liable to intermissions, and occasionally, after lasting some time, disappears for years or even for life. It is attended with no special symptoms, excepting such as may result from the continuous drain of nutrient

fluid, and those directly connected with the condition of the urine and urinary organs; and its presence is compatible with apparent good health and even with long life. The characters which the urine presents have already been described; it may be added that chylous urine not unfrequently coagulates in the bladder, causing more or less discomfort and the discharge of coagulated material.

Pathology.—Dr. Prout attributed the disorder to a combination of two circumstances;—one a defect of assimilation which permitted chyle to mingle with the blood without being converted into blood, the other some renal default, in consequence of which unchanged chyle was allowed to sweat from the kidneys. But the blood has been examined in cases of chyluria without the detection of any abnormal chemical constituent in it; and not only, as has been already stated, is there no evidence during life to show that the kidneys themselves are diseased, but post-mortem examination equally fails to detect any structural change in them.

Dr. W. Roberts, basing his views partly on a case recorded by himself, and partly on one published by Dr. Vandyke Carter, suggested some years since an explanation of the phenomena of chyluria which, so far as it goes, seems to be correct for at least many cases. In these two examples there was chyluria, but there was also on the lower part of the abdomen and in the scrotum, enlargement of lymphatic vessels, with vesicular dilatations, which yielded abundance of lymph or chyle —exactly the same kind of fluid as that which was passed with the urine. In Dr. Carter's patient, the discharge of chyle from the urinary organs and that from the skin alternated. Dr. Roberts contended that in these cases the chyle in the urine and that yielded by the skin were derived from a common source—namely, rupture of vesicular dilatations of lymphatic vessels situated on the one hand in the mucous surface of the bladder, or that of some other part of the urinary tract, on the other hand, at the cutaneous surface; and he thence argued that chyluria generally depends on a similar lymphatic affection of the mucous membrane of the urinary tract. The disease, in fact, from this point of view, is identical with what has been described earlier in this volume under the name of elephantiasis lymphangiectodes. Many other cases of this association have since been recorded.

We have already discussed the interesting discovery by Dr. Lewis of the filaria sanguinis hominis in the urine, diseased tissues, and blood of patients suffering from chyluria and spurious elephantiasis in India; and have shown that there is good reason to believe that the parasite is (at any rate in many cases, possibly in all) the essential cause of these two affections; the frequent association of which is thus plausibly explained.

Treatment.—It is needless to enumerate all the remedies which have been employed in the treatment of chyluria; nothing appears to have ever been really efficacious, and it is clear, if the explanation above

given be correct, that nothing, except perhaps rest and astringents locally applied, is likely to be efficacious. Tonics may be needed in the anæmia which is apt to come on in the course of chyluria.

XVI. HÆMATURIA.

Causation and symptoms.—The presence of blood in the urine may be due to many different circumstances, but these have already been discussed in sufficient detail, and need not be further considered now.

It is not always possible to ascertain the source or the cause of hæmaturia. It may, however, be observed that, if it take place from the substance of the kidney, it will almost always be attended with the presence of blood-casts, and the urine will generally be more or less smoky; that, if it take place from any of the urinary passages, no casts will be present; and that if it be derived from the bladder or urethra, pure unmixed blood will probably be occasionally passed, either at the commencement or at the end of micturition, or at other times. Further, the more abundant the blood is, and the more it exhibits the ordinary characters of blood and tends to coagulate, the more likely is it to have been yielded by the urinary passages. The hemorrhage which attends simple congestion or inflammation of the kidneys or urinary channels is generally scanty. The most profuse hemorrhages are usually due to villous or malignant growths of the bladder or kidney, or to the effects of renal or vesical calculi. Profuse hemorrhage is said also to occur vicariously of menstruation. We have previously described the appearance which the urine presents when mixed with blood; and we must refer the reader to other parts of this chapter for an account of the lesions of the urinary organs liable to be attended with hemorrhage, and for the means by which their respective hemorrhages may be distinguished.

Treatment.—When the discharge of blood with the urine is scanty and of temporary duration, the loss in itself is a matter of little importance, and no special anti-hemorrhagic treatment is needed. But persistent small hemorrhages, as well as occasional profuse hemorrhages, require if possible to be arrested. The patient should be placed in the recumbent position, and kept perfectly quiet and cool. He should have ice to suck, or be supplied with cold drinks in small quantities. In addition, it is advisable to give by the mouth some form of astringent medicine, such as turpentine, gallic acid, or some other vegetable astringent, lead, perchloride of iron, or a mineral acid. But probably more valuable than any of these is ergot or digitalis. If there be reason to believe that the bleeding is taking place from the kidneys, ice

or cold compresses may be applied to the loins; if from the bladder, similar applications may be made to the perinæum or hypogastrium, and either cold water, or solutions of perchloride of iron or tannic acid may be injected into the bladder.

XVII. PAROXYSMAL HÆMATURIA. (*Paroxysmal hæmatinuria*)

Definition.—This is a remarkable affection, which was first distinctly described a few years since by Dr. G. Harley, and of which many cases have since been recorded. It is characterised by the sudden occurrence, at more or less irregular intervals, of severe rigors, followed by the discharge from the kidneys of urine loaded with blood—the patient's health between successive attacks being apparently good, or at all events not seriously impaired.

Causation.—Paroxysmal hæmaturia has hitherto been observed almost exclusively in males and in such as are of adult age. A few of the sufferers have previously had ague; but with this exception the patients have, apart from their renal affection, enjoyed good health, and have been apparently quite free from malarious taint. In all cases the onset of the disease is sudden, and almost without exception distinctly traceable to exposure to cold or draughts.

Symptoms and progress.—The patient, immediately after exposure or even in the course of it, begins to complain of chilliness and uneasiness across the loins—the latter condition speedily passing into more or less severe aching, the former into an extreme sense of general cold, attended with pallor or duskiness of surface, shrinking of skin, and severe rigors; together with which symptoms there may be weakness, stiffness or aching in the limbs, yawning, nausea and vomiting, and retraction of the testicles. During this time the temperature is lowered, and often by as much as two or three degrees. After the patient has been in this condition for half an hour, or it may be an hour or two, he is astonished to find on passing water that this fluid is exceedingly dark-coloured and turbid, not unfrequently resembling porter. The general symptoms now speedily abate, and the patient, after a little reactionary rise of temperature, but no sweating stage, appears at the end of a few hours to be perfectly well. The urine gradually loses its specific characters, and a little later perhaps than the patient's apparent restoration to health resumes its normal condition. The porter-like urine, which is generally faintly acid and of variable density, deposits an abundant grumous sediment, and contains a large quantity of albumen, together with granular and hyaline casts and probably crystals of oxalate of lime, but in place of blood-corpuscles (which are detected rarely and in small numbers) presents

abundant brownish granular matter, which is supposed to be due to the disintegration of these bodies. The onset of subsequent attacks is equally sudden with that of the first; and the succession of events is repeated exactly in them. Moreover, the later attacks, like the first, are generally distinctly traceable to the influence of cold: the slightest draught or the slightest chill being in many cases competent to evoke them. In some instances the paroxysms recur with almost ague-like periodicity; more generally, however, they come on at irregular intervals. Sometimes patients suffer from them once or twice a day, sometimes once or twice a week, sometimes at longer intervals, and they often lose their liability to them during warm weather. With such variations the disease may last for years, generally too without inducing any serious consequences as respects either the condition of the kidneys or the general health. The patient, however, often becomes anæmic, languid, and weak.

Pathology.—The pathology of paroxysmal hæmaturia is somewhat obscure. It has been supposed to have some relation with ague, with oxaluria, and with rheumatism. It has been regarded, on the one hand, as an affection of the kidney, on the other as a disease of the blood. But whatever view be ultimately adopted, there are certain facts which stand out clearly: namely, first, the dependence of the paroxysm on a cutaneous chill; second, the intense congestion of the kidney which attends the paroxysm; third, the relief of both congestion and paroxysm by a copious discharge of blood; and fourth, the independence of all these conditions of any structural disease of the kidney. The phenomena of the disease, indeed, are probably due to an influence transmitted from the skin to the vaso-motor nerves of the kidney, in virtue of which temporary congestion takes place.

Treatment.—Many remedies have been employed, but none with any striking success: quinine and arsenic on the ground of the periodicity which the disease presents; iron because of the patient's anæmic state; perchloride of iron, gallic acid, and lead for their styptic properties; and digitalis and ergot of rye on account of their influence in contracting the arterioles. The most important treatment, however, is the prophylactic:—during the paroxysm the patient should be placed in bed and kept warm; and at other times he should be cased in flannel and otherwise warmly clad, his feet and loins especially should be protected, and he should carefully avoid all exposure to draughts, all loitering in the cold, and riding in cold weather in an open vehicle.

XVIII. DIABETES. (*Diabetes Mellitus. Glycosuria.*)

Definition.—The most striking phenomenon of this disease is the excretion of urine containing a greater or less amount of glucose or grape-sugar. It is not, however, everyone whose urine contains glucose who can be said to suffer from diabetes. For it has been shown that this substance may be present in the urine temporarily or in small quantities in many affections involving hepatic congestion, such as injuries or organic lesions of the liver, and obstructive cardiac and pulmonary complaints, in certain affections of the central nervous organs, and also under the influence of particular articles of diet; while none of the other special phenomena of diabetes are either present or tend to become developed.

Causation.—The cause of diabetes is not known. It is certainly hereditary in some cases; it occurs at all ages, from infancy to old age, and in both sexes, though about twice as frequently in the male as in the female. It has been attributed to exposure, to habits of life, to injuries of various kinds, and to mental disturbance. In most cases, however, no cause whatever can be assigned or suggested.

Symptoms and progress.—Diabetes, for the most part, comes on insidiously. The patient perhaps observes, almost by accident, that day by day his urine is getting more and more abundant, his thirst is increasing, his appetite is getting voracious, and yet that he is losing flesh and strength. Occasionally it happens that he is also, and possibly first, struck by some peculiarities in his urine dependent on the presence of sugar in it. He finds that when drops of it fall upon his trousers or boots, a whitish powdery film is left after evaporation, or that flies, bees, or other insects are attracted to the contents of his chamber-pot, or to surfaces on or against which he has emptied his bladder. The prominent features of the disease are comprised in this brief sketch: they are, the excretion of an excessive quantity of urine loaded with glucose, intense thirst, voracious appetite, together with progressive emaciation and debility, followed after a longer or shorter time by death. These symptoms, however, present a good deal of variety, and many others of more or less importance are generally associated with them. We will discuss them *seriatim*.

The quantity of urine discharged is generally much larger than natural; so that the patient not only micturates frequently during the day, but is compelled to rise from his bed several times in the night in order to relieve himself. Its quantity depends, of course, mainly upon the quantity of fluid which he drinks, and therefore varies largely. It is sometimes little more than normal, but generally averages between six and twelve pints daily, and occasionally rises to twenty, thirty, or more. The urine is usually of a pale yellow colour, acid, clear and free from sediment, and has a peculiar odour which has been

likened to that of new milk, apples, or hay. Its specific gravity, notwithstanding the large quantity passed, is always abnormally high. It is rarely below 1035, often rises to 1045 or 1050, occasionally reaches 1060, and is said to have exceeded 1070. The cause of this density is the presence of an abnormally large proportion of solid constituents. As a rule, considerably more urea is discharged daily by diabetic than by healthy persons; but the amount of urea is usually very small in proportion to the quantity of fluid in which it is dissolved. The increase of specific gravity, therefore, is not due to that ingredient. It depends, indeed, almost entirely upon sugar. This varies of course considerably in quantity; but it generally forms from eight to twelve per cent. of the urine, and ranges from fifteen to twenty-five ounces daily. Its amount may, however, be much less than this, and also much greater. It is greatest after meals, and is always largely increased after the ingestion of sugar or starchy food. Under opposite circumstances it diminishes; and it may disappear absolutely if the diet be restricted to nitrogenous substances. Sometimes, under the influence of inflammatory affections, and again towards the close of the disease, the urine diminishes both in quantity and in specific gravity, and its sugar lessens or fails; sometimes it becomes albuminous, and hyaline casts may be found in it. Dependent in some degree on the irritant effects of the urine, the urethral orifice in the male, or the vulva in the female, becomes red and irritable, and even excoriated or eczematous. The sexual appetite is sometimes augmented in the beginning; but both that and virile power diminish before long, and then disappear.

One of the most distressing symptoms of which diabetic patients complain is extreme thirst; and it is one of the first symptoms to make its appearance. The appetite, too, is generally excessive, sometimes ravenous. This, however, is subject to considerable variation. Sometimes it is no greater than natural, sometimes it is much impaired; and there may even be nausea and absolute loathing of food. The latter conditions often come on towards the termination of the case. The mouth, fauces, and tongue are usually dry, clammy, and morbidly red. The gums are apt to retreat from the teeth, and these latter to become loose and fall out. The patient often complains of uneasiness or sinking at the epigastrium. The bowels usually are constipated, the motions scanty and dry; but occasionally, and not unfrequently ushering in the fatal event, dysenteric diarrhœa supervenes.

The skin of diabetic patients is almost always dry and harsh, though occasionally slight perspirations occur, and some patients perspire freely. There is often a tendency to itching; and various eruptions, especially eczema, psoriasis, and boils, are said to be of common occurrence. The hair sometimes falls out. The skin, or rather perhaps the patient generally, yields an unpleasant odour, like that characteristic of his urine.

The symptoms referrible to the heart and lungs are merely such as

usually attend wasting disease, namely, increasing feebleness and rapidity of pulse, and more or less shortness of breath, especially on exertion. The blood of diabetic patients contains glucose, of which as much as ·3 to ·5 per cent. has been detected by analysis.

Nervous phenomena of various kinds usually manifest themselves in the course of the disease. The patient becomes apathetic, morose or taciturn, or irritable, or towards the close drowsy or comatose. Insanity sometimes supervenes; and occasionally various forms of hyperæsthesia, loss of motor power, and the like. Impairment of vision is also a common incident of the disease; in some cases the patient loses simply the power of adjustment for near vision—he becomes prematurely presbyopic; in some he suffers from amblyopia; while in some soft cataract forms in one or both eyes.

But, besides the above phenomena, others come on which are not so much referrible to any one organ as to general impairment of nutrition and advancing debility. There is great susceptibility to external cold. A sort of hectic condition arises, occasionally attended with febrile elevation of temperature; generally, however, the temperature remains normal or falls a little below the normal. Emaciation is almost constant; the fat disappears, the muscles shrink, the frame becomes attenuated, the skin appears tightly drawn over the forehead and other parts of the face, and is thrown into fine wrinkles when expressional and other movements of the facial muscles are executed. Occasionally, on the other hand, and more particularly in elderly persons, the tissues remain overloaded with fat to the end. Towards the close of the disease anasarca, generally limited to the lower extremities, is of common occurrence. And not unfrequently gangrene takes place in the fingers, toes, or more extensive portions of the extremities, in the genitals, nose, ears, or other parts.

Another complication which is at least as common as any of the above, and on the whole of far more importance, is pulmonary phthisis. This attacks a large proportion of diabetic patients; and indeed of patients who die of diabetes probably one-half suffer from it. The affection is rarely if ever in the form of miliary tuberculosis, but almost invariably in that of caseous consolidation, with tendency to disintegration and the formation of cavities.

In some cases the progress of diabetes is exceedingly acute and rapid. Death has resulted from it after an illness of two or three weeks only. On the other hand, death may be delayed for ten years or more. For the most part, however, the patient succumbs in from one to three years. Recovery is exceedingly rare. The cause of death usually is asthenia, hastened in some cases by gangrene, dysentery, or phthisis; but not unfrequently the patient dies comatose. Diabetic patients bear fatigue, mental or bodily, very badly, and at an advanced period of their disease are apt, after such fatigue, to fall into a state of almost sudden collapse, from which they do not rally.

The above remarks apply to the usual form of the disease. It must be added, however, that in elderly persons and especially in such as are gouty, the urine not unfrequently contains sugar, it may be in large quantities, and yet few or none of the other symptoms of diabetes are present. The glycosuria under such circumstances may persist for years, either uniformly or with remissions, the patient perhaps passing at times more water than natural, and suffering more or less from dyspepsia, yet presenting no emaciation and no serious impairment of strength, and ultimately recovering, or dying, not of diabetes or its ordinary complications, but of some independent disease.

Morbid anatomy and pathology.—Morbid anatomy reveals little as to the nature and processes of diabetes. Excluding dysenteric affection of the bowels, gangrene of various parts, pulmonary tuberculosis, and cataract (which are not present in all cases, and present no distinctive characters), but little remains for description. The kidneys generally are enlarged and more or less congested, and the epithelia lining of the tubules is occasionally in a distinctly fatty condition. The liver and other chylo-poietic viscera, to which on theoretical grounds attention should be mainly directed, present no constant lesions. The former has occasionally been found cirrhotic, the latter inflamed; but far more commonly all appear healthy. The nervous system, again, has been examined with care, on account of the influence which certain parts of it have in causing glycosuria. Tubercular and other tumours have occasionally been discovered in the neighbourhood of the fourth ventricle; and Dr. Dickinson has recently drawn attention to the existence of small cavities, sometimes visible to the naked eye, originating in softening and degeneration of the tissues around some of the smaller arteries, and containing, when fully formed, simply serous contents. He has found them in most parts of the central nervous organs, but more particularly in the olivary bodies, the median plane of the medulla oblongata, and the grey matter in the floor of the fourth ventricle.

If the pathology of diabetes has not been completely elucidated, it has at any rate had much light thrown upon it during the last few years by the labours of Bernard and other investigators. It has been proved that the liver, besides manufacturing bile, is an organ for the conversion of albuminous and starchy matters (mainly if not entirely those obtained directly from food) into dextrine or glycogen—a starchy substance which exists in large quantities in the liver, and is readily convertible by ferments (and among others by a peculiar albuminous ferment existing in the blood but not yet separated from it) into glucose, or grape-sugar. It is probable that the healthy liver also converts sugar itself into glycogen, and that hence, amongst other duties, the liver opposes a barrier to the admixture of saccharine ingesta with the blood. What becomes of this glycogen, which is formed and accumulates in the liver, we need not stop to consider. It is certain,

however, that in health neither it nor glucose is discovered in the blood. Further, experiments made by Bernard, Schiff, and others have demonstrated the important influence which the nervous system exerts over the glycogenic function of the liver. It has been proved that by irritating various parts of the central nervous organs artificial diabetes may be induced—irritation of the floor of the fourth ventricle, particularly of a spot in it midway between the origins of the auditory nerves and par vagum, being especially efficacious in this respect. There is reason to believe that this spot is either the origin of, or in relation with, the tracts of sympathetic nerves which regulate the diameter of the hepatic vessels; and that through the agency of these nerves the vessels of the liver become actively dilated, upon which phenomenon congestion and glycosuria supervene. Schiff, by dividing the anterior columns of the cervical cord through which the sympathetic tracts above referred to pass on their way to the liver, also produces glycosuria; which, again, is probably dependent on dilatation of the hepatic vessels and hyperæmia, but dependent upon dilatation of paralytic origin, and which, like the diabetes it causes, is of comparatively long duration. Experiment would therefore seem to show: that diabetes depends on dilatation of the hepatic vessels, with accumulation of blood in them and rapid flow of blood through them, and consequently on increased or rather modified functional activity of the liver; and that this dilatation may be either active—the result of irritation of nerves—producing for the most part a temporary condition of diabetes, or passive—the result of paralytic dilatation—inducing as a rule a chronic form of glycosuria. The dependence of diabetes on hyperæmia of the liver has been demonstrated by other experiments in which hyperæmia has been brought about without the intervention of the nervous system; and is confirmed by the not unfrequent occurrence of some degree of the affection in pathological congestion of the liver arising from cardiac or pulmonary disease, from injuries to the liver, and from inflammation of the organ. According to these views, which, it may be remarked, only partially explain the dependence of diabetes on hepatic derangement, the occasional and temporary impregnation of the urine with sugar would seem to have an irritative, the typical forms of diabetes a paralytic, origin.

Treatment.—The treatment of diabetes is a subject of great interest, and has been regarded and conducted from all points of view with varying degrees of success. As with most other diseases, some cases of it are so serious from the beginning and so rapidly fatal that all efforts to arrest their progress are futile; while some cases are so slightly pronounced that the patients either remain in fair health in spite of their sugary urine, or appear to derive benefit from almost any treatment. Between these extremes lie the great majority of cases, which, if not admitting of cure, undoubtedly often admit of important alleviation by appropriate treatment. It may at once be stated that

the use of blisters and other local applications to the head or to the liver has been advocated and practised by various physicians, on the ground that one or other of these organs was at fault; and beneficial results have been recorded. Further, we may at once point out the general importance of promoting the functions of the skin by warm baths and warm clothing; of maintaining the regular action of the bowels; of alleviating, arresting, or curing dysentery and the other complications which so frequently attend the progress of diabetes; of preventing all unnecessary fatigue; and of putting the patient under those external conditions which are commonly regarded as conducive to health.

The most important point, perhaps, in the treatment of diabetes is the regulation of the patient's diet. It has long been proved that the abstention from sugar and from those other articles of food which are most readily convertible into sugar is always attended with a marked diminution in the quantity of sugar voided, in the specific gravity of the urine, and in the amount of that fluid secreted; and that in a very large proportion of diabetic patients there is at the same time gain of flesh with manifest improvement of health. For these reasons it is customary to debar the patient from certain alimentary matters, especially sugar in every form, and all vegetables or vegetable products whose nutritious qualities depend on sugar, starch, or related matters —among which may be enumerated bread, potatoes, rice, sago, tapioca, peas, beans, turnips, parsnips, carrots, and most fruits. There is good reason also to believe that alcohol in all its forms is pernicious. Among permissible foods are: first, green vegetables; second, all sorts of animal food, including milk, eggs, cheese, and butter; and, third, tea and coffee without sugar. It is found, however, in practice almost impossible to overcome the craving for bread or some equivalent for bread which soon arises under the use of a restricted diet. Various substitutes have been suggested and may be used temporarily; the most important being gluten bread, bran cake, and almond biscuits or rusks, to which may be added (as being more palatable, though more objectionable) toast uniformly and deeply browned. Lately Dr. Donkin has advocated the administration of skim milk, to the exclusion of all other food. He gives from six to eight pints daily to an adult. And it is certain that many patients in a short time get fairly reconciled to it, that they often gain strength and flesh under its use, and that at the same time the urine diminishes in quantity, in density, and in the amount of sugar it contains.

It has often been held important to restrain the patient from gratifying his intense craving for drink. It is cruel, however, to put such restraint upon him, and of very doubtful benefit. Acidulated drinks are said to be specially useful in assuaging his thirst, and, above all, dilute solutions of phosphoric acid.

Of all drugs, opium seems to be the most efficacious. It has long

been esteemed in the treatment of diabetes; and especially Dr. Pavy has latterly extolled its virtues. Diabetic patients are said to be little susceptible of the influence of opium, and may therefore take it with safety in comparatively large quantities. It is best, however, to commence with small doses, say half a grain, of the powder, three times a day, and gradually to augment them, according to their effect, until each dose is increased to five or six grains. A fair number of cases have been recorded in which great amelioration, if not absolute cure, has followed this treatment. Still more recently Dr. Pavy has employed, and apparently with considerable success, codeia, in doses commencing at about half a grain, three times a day, and gradually increasing to two or three grains.

Again, alkalies—bicarbonates, acetates, citrates—have been regarded as valuable remedies; as also has the hot vapour bath. Iron and other tonics are sometimes beneficial.

As respects the treatment of the masked diabetes of elderly people, it is impossible to lay down definite rules. It is generally needless to carry out the plans recommended above, at any rate to carry them out strictly or continuously.

XIX. DIABETES INSIPIDUS. (*Diuresis.*)

Definition.—Under these titles are grouped a number of cases, which are linked together and characterised by the association of extreme thirst with the excretion of a large quantity of pale limpid urine, free from sugar, and of low specific gravity.

Causation.—Diabetes insipidus is rare, but appears to occur at any age and in either sex. The causes to which it has been attributed are various. Among them may be mentioned tuberculosis, diseases of the brain, drink, accident and exposure. According to Trousseau and some others, it has a close relation to diabetes mellitus, not only in symptoms but in the facts that there is an hereditary connection between them, and that the former is occasionally a sequela of the latter. Bernard, moreover, has shown that diabetes insipidus, as well as glycosuria, may be produced by irritation of the floor of the fourth ventricle.

Symptoms and progress.—This affection sometimes comes on insidiously, sometimes quite suddenly. Its chief symptoms are the following:—First, the secretion of large quantities of urine; the quantity passed is often considerably larger than in saccharine diabetes; it may be as much as 20, 30, or 40 pints daily, or even twice as much; the urine, moreover, is pale, watery, of low specific gravity (often not above 1002, 1003, or 1004), and containing no sugar or other abnormal ingredient. Second, extreme thirst; this is proportionate to the diuresis, the

quantity of fluid drunk being equal or nearly so to the quantity eliminated.

Other symptoms vary. In some cases the patient appears to be well in all other respects, and, except for the continued presence of his infirmity, enjoys life, probably attaining old age. In some cases he presents all the usual indications of diabetes mellitus; he has a voracious appetite, a parched mouth, and dry skin; he becomes anæmic, sallow, emaciated, and weak; and after a longer or shorter time dies as ordinary diabetics die. In other cases, again, diabetes insipidus is from its commencement associated with the presence of tuberculosis or other lesions, and is thus a mere incident or complication of a more serious malady.

Morbid anatomy.—In a few cases which have been collected by Dr. W. Roberts, the morbid anatomy of diabetes insipidus is illustrated. There is little, however, in the recorded post-mortem examinations to throw light on the nature of the affection. In several of the cases the kidneys were atrophied, and in one hydronephrotic. There is some reason to suspect that in these the primary affection was renal. In others the kidneys were healthy, as also were they in a case which died under our own care. In this case, as in one of Dr. Roberts's, the patient suffered from tuberculosis, which probably caused death. Here undoubtedly the diuresis was symptomatic only.

Treatment.—There is little to say about the treatment of diabetes insipidus. Various remedies, including tonics and regulation of diet, have been tried. Trousseau and Rayer strongly recommend valerian in large doses. The former commences with two or three drachms of the extract daily, and generally pushes the treatment until the daily portion reaches an ounce. Baths are sometimes beneficial. The constant galvanic current, passed between the loins and epigastrium, has recently been tried by Dr. M. Seidel.

XX. SUPPRESSION OF URINE. (*Ischuria Renalis.*)

A. *Functional suppression of urine.*

More or less complete suppression of the urinary secretion, lasting for a longer or shorter period, is not unfrequent in the course of many different diseases or morbid conditions, among which may be especially enumerated malignant cholera, certain of the infectious fevers, acute enteritis, inflammatory affections of the kidneys, collapse, and hysteria. In many such cases the suppression is symptomatic only, and probably scarcely affects the patient injuriously; in others the retention of urea and other effete nitrogenous matters in the blood which

attends the suppression induces or aggravates typhoid phenomena and thus hastens death. It is remarkable, however, how sometimes, and more especially in cases of hysteria, the urine continues for many weeks at a time in almost complete abeyance—the patient going, perhaps, for two or three days at a time without secreting any, and then perhaps passing only an ounce or two in the course of the twenty-four hours—and yet the patient remains wholly free from evidence of uræmic poisoning. These several cases of suppression are considered elsewhere in connection with the diseases in which they occur, and need not further detain us now. For the treatment of these cases, simple diuretics, more especially copious bland fluids, the use of hot hip or other baths, and the application of counter-irritants to the lumbar region, comprise everything likely to be of real service.

B.—*Suppression of urine from obstruction.*

Causation and morbid anatomy.—Another class of cases of so-called 'suppression' is that in which the failure to discharge urine depends on the existence of some mechanical obstacle to the escape of urine, situated either in the pelvis of the kidney or, as is far more common, in some part of the ureter. The permanent obstruction of one ureter, its fellow remaining pervious, is, as we have already shown, a not uncommon accident; and on the one hand results in the production of hydro-nephrosis with ultimate wasting of the corresponding kidney, and on the other hand leads to increased functional activity of the opposite organ, which henceforth does the work of both. Obstruction of the ureter is most commonly due to the impaction of a calculus; and hence it is not altogether remarkable that a person who has had one ureter blocked up and one kidney destroyed should be liable to the occurrence at some future time of the same accident on the opposite side. And indeed it is generally in cases of this sort that mechanical suppression occurs.

Symptoms and progress.—The suppression of urine under these circumstances comes on suddenly. Sometimes it is, and remains, absolute; perhaps more frequently a small quantity of urine of low specific gravity, and containing little urea, is still passed at irregular and probably long intervals. It is very remarkable that in most cases of this kind, no matter how complete the suppression may be, the patient scarcely seems to suffer during the first seven or eight days. He may perhaps have a little nausea, there may be some degree of insomnia, and there may also be some failure of muscular strength; and this is all. At the end of this time, however, manifest symptoms of the effects of the retained poisonous matters on the system arise. They consist in the first instance in muscular tremors associated with distinct increase of muscular debility; and in the next place in slow, panting respiration, and contraction of the pupils. These

phenomena appear never to be absent, and they become more and more marked with the progress of the case. But soon other symptoms are superadded. The patient complains of anorexia and thirst with dryness of the mouth and fauces; he becomes drowsy, but sleeps only in snatches; and he may present a little occasional delirium. Death, which is rarely preceded by coma, and still more rarely by convulsions, takes place mainly by asthenia at the end of two or three days from the first occurrence of toxæmic symptoms. Throughout the patient's illness there is no fever; on the contrary, towards the close the temperature tends to fall; the pulse differs little in frequency from the normal, and the skin is often moist. The symptoms indeed are widely different from those which are ordinarily attributed to uræmia.

The diagnosis of cases of this kind is often facilitated by the combination of a history of some long antecedent attack of renal colic on one side, with present symptoms of an acute attack of the same kind on the opposite side. The calculus soon, however, becomes impacted, and then probably all local pain and uneasiness disappear. Further, there is no necessary pain or uneasiness in the loin.

Treatment.—For this affection we can do little or nothing. We may adopt such treatment as is recommended for renal colic in the hope that the stone, if there be one, may be aided in reaching the bladder; and we may endeavour, as Dr. W. Roberts recommends, by kneading the abdomen, to empty the distended ureter and coincidently it may be to dislodge the calculus.

Section II.—DISEASES OF THE PELVIC ORGANS.

The diseases of the genito-urinary organs, situated within the pelvis, are of extreme interest and importance; but they are claimed for so many departments of practice that it is difficult to determine to what extent they ought to be included in a work on medicine. We propose to discuss very briefly, and mainly in reference to diagnosis, those among them which are important on account of their liability to be confounded with, or to complicate, the diseases, already considered, of the other abdominal viscera.

I. DISEASES OF THE URINARY BLADDER.

1. *Inflammation* arises under many different circumstances, which need not be enumerated. It is characterised anatomically by congestion and thickening of the mucous membrane, with the secretion of

mucus, which may be simply abundant or may acquire the characters of pus. Sometimes submucous extravasations of blood occur, sometimes blood escapes from the surface. Occasionally ulceration takes place, or membranous pellicles form, or the mucous membrane itself or large patches of it exfoliate and are discharged. Occasionally, also, abscesses are developed in the substance of the vesical walls, or inflammation commencing at the mucous surface extends in depth until it involves the serous membrane.

The *symptoms* of inflammation of the bladder are mainly: pain and tenderness in the neighbourhood of the organ, therefore in the perinæum and immediately above the pubes, extending probably to the penis, to the sacrum or loins, and to the contiguous parts of the thighs; irritability of bladder, with constant desire to pass water; and the discharge of urine which, according to circumstances, presents only a slight cloud of mucus, or more or less abundant thick ropy mucus, or mucus mingled with blood, or pus. Sometimes the urine contains shreds of tissue, and frequently becomes alkaline and offensive. Cystitis may be acute or chronic, and varies greatly in its intensity and danger in different cases. When acute the general febrile symptoms may be very severe. Cystitis often leads to pyelitis; and further, the latter affection not only resembles cystitis in some of its symptoms, but inflammation, commencing in the pelvis of the kidney, is apt to travel along the ureter and thus to involve the bladder.

For the *treatment* of cystitis we must refer to surgical works and to what has been previously said in reference to pyelitis.

2. *Tubercle* affects the bladder but rarely, and is then almost invariably associated with tubercle of the kidneys and ureters, or (which is yet more common) with tubercle of the prostate and vesiculæ seminales. It is of the miliary variety, and tends, as in the intestines and on other mucous surfaces, to produce shallow circular ulcers, which, by coalescence, are apt to cause superficial destruction of some extent.

The *symptoms* are in themselves undistinguishable from those of chronic cystitis.

3. *Morbid growths.*—The most important of these are villous tumours and the several forms of malignant disease. The latter usually commence in the prostate or some neighbouring part, and are rarely of primary origin in the bladder. Tumours are generally attended with more or less pain, referrible to the bladder, and more or less interference with micturition. Moreover they are apt to be complicated, after a while, with symptoms of cystitis. Villous and malignant tumours are frequent sources of profuse hemorrhage. The latter are further characterised by sooner or later involving contiguous organs, and by inducing progressive cachexia.

4. *Dilatation.*—This condition depends on the accumulation of urine or other matters within the cavity of the bladder. It may occur in

paraplegia and other paralytic conditions from paralysis of the vesical walls, and also in hysteria. It is common in the later stages of many of the specific fevers, and during the typhoid condition, from failure of the reflex influence on which the evacuation of the bladder depends; and it is especially common as a consequence of obstructive disease, such as stricture of the urethra, enlarged prostate, or tumours of any kind involving or compressing the neck of the bladder. When the dilatation is chronic, and secondary to some impediment, the muscular walls become hypertrophied, and sacculi are developed. Under any circumstances the mucous surface is apt after a time to get inflamed; and the dilatation and inflammation are both of them liable, sooner or later, to involve the ureters and the cavities of the kidneys.

Symptoms.—In cases in which retention of urine is dependent on paralysis, or connected with the presence of febrile disturbance or the typhoid condition, the bladder may become enormously distended without causing any apparent suffering to the patient. Even in cases of chronic stricture and such-like conditions in which, although the dilatation of the bladder may be extreme, it has been slowly attained, the organ becomes remarkably tolerant of its burden, and the patient suffers comparatively little. In other cases his sufferings are often extreme. He complains of more or less general uneasiness, pain and tenderness over the hypogastric region, in the penis and in adjoining parts; but the pain is subject to frequent exacerbations, dependent on the violent but futile spasmodic efforts of the bladder to void its contents. In many cases, if the obstruction be not complete, more or less urine either constantly dribbles away or is passed in small quantities during the spasmodic efforts. The distended bladder forms a tense, ovoid tumour, which rises out of the pelvis from behind the pubes, and may extend upwards to the umbilicus or beyond. It occupies the middle part of the abdomen, and, unless it be largely sacculated, is symmetrical in form and position. The enlarged bladder can rarely fail of recognition if due attention be paid to the position and form of the tumour, to the perfect dulness on percussion which it presents, and to the characteristic pain which so often attends it and is evoked by manipulation.

Treatment.—When the bladder becomes distended in the course of fever and paralytic affections, equally as when it becomes distended in consequence of surgical diseases, the urine should be drawn off; and, if necessary, should be drawn off periodically. Further, if the urine be ammoniacal, or there be discharge of ropy mucus or pus, it may be well not only to empty the bladder, but to wash it out either with pure water or with dilute antiseptic solutions.

II. DISEASES OF THE UTERUS, FALLOPIAN TUBES, AND OVARIES.

A. *Metritis and Oophoritis.*

Causation and morbid anatomy.—Inflammation of the parts above named may result from many causes, but is most apt to occur during the menstrual period and after parturition. Inflammation may affect the mucous surface only of the uterus, or this together with the muscular coat; and in the latter case is apt to spread to the surrounding connective tissue and to the peritoneum; the Fallopian tubes are frequently involved. The inflamed mucous membrane gets congested, thickened and pulpy, and occasionally (especially in the puerperal variety) is thrown off as a slough. The surface may at first be dry, but soon secretes a thin fluid, and subsequently pus, with which blood may be mingled. When the muscular parietes are involved, they become soft, tumid, infiltrated with inflammatory products, and occasionally studded with spots of extravasation. Sometimes abscesses form. Inflammation of the ovary, which is said to be most common on the right side, is characterised by swelling, œdema, and congestion of the organ, and occasionally goes on to suppuration. Its peritoneal surface is often involved, and adhesions are then apt to form between it and neighbouring parts.

Symptoms.—Metritis is generally a trivial ailment, attended with slight febrile symptoms; but it is sometimes, and more especially in puerperal cases, a disease of extreme gravity, rapidly ending fatally with symptoms which bear a close resemblance to those of pyæmia or severe erysipelas, with the former of which, indeed, it is apt to be complicated. The local indications of metritis are weight, pain, and tenderness in the situation of the womb. Pain and tenderness are felt in the hypogastric region, and occasionally manifest tumour may be recognised in that situation. Uneasiness, pain, and soreness are referred to the sacrum or lower lumbar region, to the vulva and perinæum, and to the groins and inner aspects of the thighs. Further, tenesmus and painful or difficult micturition are often complained of. When the ovary is inflamed, the pain and tenderness are referred to the region which the ovary normally occupies, namely, the point of intersection of the horizontal line drawn between the anterior superior spines of the ilia and the vertical line which divides the median from the lateral regions of the abdomen. The organ lies much higher than is generally supposed, and is deeply situated. When swollen it may often be distinctly felt in this situation.

B. *Morbid Growths.*

1. *Tubercle* occasionally affects the uterus and Fallopian tubes; it commences at the mucous surface and leads to the gradual destruction of the subjacent tissues, and to the abundant accumulation of cheesy matter. Much more rarely tubercle is discovered in the ovaries. Tubercle of these organs is almost invariably associated with advanced tuberculosis of other parts, more especially of the peritoneum.

The *symptoms*, if any special to these organs be complained of, are those of subacute inflammation.

2. *Myomata* are common in the uterus. They probably never occur before puberty; and affect virgins, it is said, more frequently than married women. They originate in the substance of the uterine walls; if near the inner surface tending to form polypi, if near the outer aspect to form pedunculated outgrowths into the cavity of the peritoneum, if in the more central parts to remain imbedded. They vary in size from mere points up to masses of many pounds weight; and may be single or multiple. They are usually slow of growth, not unfrequently become stationary, and are liable after a time to undergo degenerative changes and to shrink. Similar tumours are occasionally developed in the ovary.

Symptoms.—In addition to the special symptoms due to their weight, to their pressure on neighbouring organs, such as the rectum and bladder, to their interference with parturition, and to their influence over the uterine functions, myomata frequently form masses which rise into the cavity of the abdomen, and may be recognised through the parietes as hard, rounded, or nodulated tumours. Such tumours necessarily vary in size, form, and position, and are generally more or less unsymmetrical. Independently of vaginal examination, their situation in the neighbourhood of the uterus, their shape and density, their slow growth, the circumstances under which they have arisen, and the absence of involvement of lymphatic glands and remote organs, and generally of progressive cachexia, will usually enable an accurate diagnosis to be made. It must not be forgotten, however, that pedunculated tumours of this kind are apt to become attached to other parts, and, especially after pregnancy, to be left in comparatively remote situations and thus to simulate renal or hepatic tumours, or tumours of other organs.

3. *Malignant disease* is liable to affect all the organs under consideration. The uterus is a frequent seat of its primary development; but this organ may also be affected secondarily. As a primary disease it usually commences between the ages of thirty-five and fifty; and generally takes its origin in the cervix or os. Carcinoma is probably its most common form, but epithelioma and sarcoma are neither of them rare. The ovaries, also, are not very unfrequently the seat of

cancerous or sarcomatous growths. These may be primary in them or secondary to growths elsewhere. They are generally associated with similar disease in either the uterus, the peritoneum, or other abdominal organs. Ovarian malignant disease results for the most part in the development of lobulated masses, which in their general outlines are not unlike ovarian cystic tumours; and, indeed, it is common for malignant disease of the ovaries to be associated with the more or less abundant development of cysts.

The *symptoms* of malignant disease of the uterus need not detain us. And with respect to those of malignant disease of the ovaries, it may be observed that the tumours would probably in themselves be undistinguishable from ordinary ovarian tumours, and that their recognition as being dependent on malignant disease must rest upon the progress of the case, the development of tumours elsewhere, the early appearance of cachexia, and the rapid downward tendency of the case.

C. *Cystic Tumours.*

Causation and morbid anatomy.—Cysts may arise either in the uterus, Fallopian tubes, or ovaries, or in connection with the peritoneal aspect of these organs.

1. *Dilatation of the uterus* may be caused by accumulation of menstrual fluid, owing either to obstruction at the os uteri or to imperforate hymen or vagina. This is an affection of early life. At a later period, owing to obliteration of the os, or to its obstruction by tumours or other causes, the uterus may become distended by the glairy secretion of its mucous surface. Under the latter circumstances the uterus rarely attains a greater size than the fist; under the former it may slowly acquire the bulk of the gravid organ, or even surpass it. The uterus may also be distended with blood.

2. The *Fallopian tube* occasionally undergoes dilatation. This condition is secondary to its closure or obstruction, generally at or near its junction with the uterus. The affected tube becomes elongated, tortuous, and sacculated and increasingly dilated towards its fimbriated extremity. Here it occasionally measures three or four inches in diameter.

3. *Ovarian cysts.*—But by far the most common and important cystic tumours are those which arise in the substance of the ovary. These may be simple or compound; may vary in size from that of a marble up to a bulk far beyond that of the pregnant uterus; and may form either a uniformly rounded or ovoid mass, or an irregular lobulated tumour. They may be limited to one ovary, or, as not unfrequently happens, may affect both organs in unequal degrees.

Cystic tumours of the ovary are rare before puberty. But from twenty or twenty-five upwards they are not unfrequent. They are most commonly met with between the ages of thirty and forty. The disease commences with the development of one or more small cysts

in the substance of the ovary. These gradually increase in size; and as they grow other cysts arise in relation with them, either in the ovary itself, or, if all manifest ovarian structure have disappeared, in the substance of the cyst-walls—a process which tends to go on indefinitely as well in the walls of the secondary and all other later generations of cysts, as in those of the primary cysts. The result is the development of a more or less complicated cystic mass, the characters of which differ according to a variety of circumstances.

In some cases the secondary cysts tend largely to grow from the outer surface of the primary cysts, and hence the tumour soon acquires a marked lobulated character; in some cases they appear mainly in the thickness of the party-walls between adjoining cysts, and the tumour becomes multilocular, and presents a good deal of resemblance in its structure to an accumulation of air-bubbles in a viscid fluid; in other cases the new growths are developed chiefly in connection with the inner surface of the cysts, and project or grow into their interior. The last mode of development presents several varieties; in some instances papillæ, villi or pedunculated cysts spring in groups from the lining membrane; in some instances, and on the whole more frequently, these intra-cystic growths resemble those from which they spring, themselves give origin to others, and thus tend gradually to fill and even to distend the cavity which they occupy.

The proportionate development of the constituent cysts presents great differences. In some instances one cyst becomes so large relatively to the others that the tumour becomes practically unilocular. In others the cysts are so numerous and small that the tumour appears to be nearly solid. And between these extremes all varieties may be met with. The enlargement of the tumours is due not solely to the formation of new cysts, but in great measure to the dilatation of the cavities already in existence. This is effected partly by the stretching of their parietes by their accumulating contents, and partly by the yielding of their parietes at points and the consequent coalescence of neighbouring cysts—a process which may be readily observed in all its stages in most ovarian tumours.

The contents of ovarian cysts differ largely. In many cases they are colourless; but they are often yellow, brown, or green. They may be transparent as water, or opaline, or perfectly opaque. They may be limpid, but are more usually glairy or viscid, and not unfrequently are thick, and glue- or jelly-like. When thick they often present a whitish or brownish sediment. Chemically they contain modified albumen, and either mucus or colloid matter, and sometimes altered blood. Corpuscles of various kinds, more or less degenerated, are generally present; sometimes pus. Cholesterine is often observed.

The walls of ovarian tumours vary greatly in thickness. Sometimes they are as thin as tissue-paper and perfectly pellucid. Much more frequently they are thick and tough, though varying in thickness in

different parts. The outer parietes, like the party-walls between cysts, have in the progress of their growth a tendency to become attenuated and to give way at points. In thin-walled tumours free communications are occasionally thus established between the cysts and the abdominal cavity, which consequently becomes distended with their profuse secretion. And even in thick-walled cysts such communications, attended with more or less escape of contents, are not unfrequent. The walls of ovarian tumours consist mainly of connective tissue, in which are not unfrequently found microscopic cysts, together with patches or masses of cell-growth having some resemblance to sarcomatous or adenoid tissue.

Among the various consequences of ovarian cystic tumours may be enumerated: the occurrence of inflammation or suppuration; rupture of the cysts with the discharge of their contents into the peritoneal cavity; the communication of suppurating cysts with the rectum, bladder, or other neighbouring hollow viscera; the occurrence of peritonitis or ascites; and pressure on the bladder, rectum, ureters, or iliac veins, with the usual consequences of such pressure.

Symptoms and progress.—The uterus dilated by fluid-contents takes the ordinary form, and occupies the usual situation, of the gravid organ; and from these and other considerations the nature of the lesion can always be pretty readily ascertained. For the diagnosis of these cases, however, and of those of dropsy of the Fallopian tubes, we must refer to works upon the special diseases of women.

Ovarian cystic tumours, in the early stage of their growth, either are unattended with symptoms, or produce discomfort by sinking into the pelvis and interfering by pressure with the functions of one or other of the neighbouring pelvic organs. At this period they are probably detectable through the abdominal parietes, or by vaginal examination. With the progress of their enlargement they rise into the abdominal cavity, and ultimately in some cases produce enormous distension, displacing the abdominal viscera, and even by direct or indirect pressure on the diaphragm interfering with the respiratory functions. An ovarian tumour of moderate size can generally be recognised as ovarian: by its obvious connection with one or other of the iliac regions, whence probably it extends into the adjoining parts of the abdomen; by its irregularity of form and the various degrees of resistance of its several lobules, with the probably distinct fluctuation of some; by its dulness on percussion; by its displacement of the intestines, and its mobility; and by the absence of pain, tenderness, cachexia, and secondary growths. When it has attained a large size, all evidence of its commencement at one side of the abdomen has probably disappeared. But there is usually even then distinct evidence of its development from the lower part of the abdomen in the fact that the intestines are displaced upwards and laterally; so that, in addition to tumour, there is complete dulness on percussion from the pubes upwards and outwards. The intestinal resonance

can generally be distinguished in the flanks, and that due to the stomach, transverse colon, and perhaps some of the small intestines above. The tumour in this case also is probably irregular as to both form and resistance; but it frequently happens, that one or two cysts preponderate largely over the others, and that distinct fluctuation may be felt in them. In some instances the bulk of the tumour consists of a single cyst; and the tumour may then not only fluctuate distinctly but present a fairly uniform rounded shape. Pain, tenderness, and fever are not necessary accompaniments of ovarian tumours; but these phenomena and others may arise during the progress of the disease. They depend on the supervention of one or more of those complications which have been previously enumerated:—namely, inflammation in the cysts or in the peritoneum which surrounds them, the development of ascites, or pressure on the pelvic organs. When the tumour becomes very large, the patient's gait resembles that of a pregnant woman; the legs get congested and anasarcous; loss of appetite and perhaps vomiting supervene; and the breath becomes short. Ultimately progressive emaciation and asthenia come on.

Ovarian tumours are generally easy of diagnosis. They are apt sometimes to be confounded with uterine, hydatid, or other growths springing from the pelvis, and indeed cannot always be distinguished from them. When of large size and mainly mono-cystic they may be mistaken for ascitic accumulations. But the anterior position of the ovarian tumour; the existence of resonance above and in the flanks, and of dulness over the whole of the tumour; the tendency for the abdomen to be thick rather than wide, and to present some degree of irregularity of form; and the total absence of any change in the level of the line separating the dull from the resonant regions when the patient shifts her position, are usually sufficient to enable an accurate diagnosis to be made. It must not be forgotten, however, that ascites is apt to come on in the course of ovarian dropsy, and that hence the two conditions are not unfrequently associated.

Treatment.—The treatment of ovarian cystic tumours alone calls for remark here. And even in reference to this subject we have little to say. Drugs have no influence direct or indirect over them. Of course if inflammation arise, the treatment usually adopted for peritonitis may be had recourse to; if the patient be weak and emaciated, tonics and nutritious diet may be prescribed; if she be suffering materially from the bulk of the tumour and its pressure on the stomach, diaphragm, or other organs, it may be tapped. But the only efficacious treatment is by the knife. The success of ovariotomy, or removal of the ovarian tumour by operation, has been so great of recent years, especially in the skilful hands of Mr. Spencer Wells, that all other forms of treatment have fallen into desuetude, excepting for those cases which from various circumstances are unsuitable for the radical cure.

III. DISEASES OF THE PELVIC PERITONEUM AND CONNECTIVE TISSUE.

We cannot conclude this section without directing attention to the remarkable tendency there is in the case of the pelvis, as there is also in that of the upper part of the chest, for diseases originating in one organ to implicate other organs in the vicinity, and for affections therefore originally distinct to cause almost identical ultimate results.

Inflammations commencing in the ovary, uterus, or vagina, in the rectum, cæcum, or bladder, in the serous membrane covering these organs, or in the connective tissue which invests them, or in connection with the bones or joints of the pelvis, are all apt to involve pelvic peritonitis with adhesions, infiltration and induration of the connective tissue of the pelvis, and the formation of abscesses which may burrow in various directions and open into the bladder, vagina, or rectum, or superficially in the perinæum, above the pubes, in the groin, or in the buttock. Further, as has before been pointed out, abscesses may gravitate from any of the parts situated in the abdomen or chest along the retro-peritoneal tissue, and thus induce the same consequences in the pelvis as though they had originated there.

Similar remarks may be made in reference to the consequences of tubercular disease of the uterus and Fallopian tubes, of the prostate and vesiculæ seminales, and of the bladder, the clinical phenomena of which are mainly those of sub-acute inflammation of the same organs.

Malignant disease of whatever kind soon spreads by continuity from the part in which it originates, and implicates all organs in its vicinity. If it commence in the uterus or vagina, it speedily infiltrates the surrounding connective tissue, and then presently involves, on the one hand the bladder, on the other the rectum, leading to free communications between these several viscera. Similarly malignant disease, commencing in the rectum or anus, in the bladder or parts at the neck of the bladder, or in the connective tissue investing these parts, or growing from the inner aspect of the pelvic bones, tends to the ultimate production of exactly similar results—to the formation in fact of a common excavation into which the several pelvic organs tend to discharge their contents.

In the above cases, but mainly in that of malignant disease, other consequences are liable to ensue, more especially, perhaps, implication of the peritoneum, the laying open of vessels with the occurrence of more or less abundant hemorrhage, and the involvement of nerves, particularly those of the sacral plexus, with the production of local pain and tenderness and of pain taking the course of the sciatic nerve and mistakable for sciatica, and apt to be followed by wasting of

the muscles, and cutaneous eruptions. Further, obstruction of the ureters with consequent hydro-nephrosis, retention of urine, impediment to the discharge of fæces, tenesmus, irritability of the bladder, and œdema of the lower extremities or of the organs of generation, are all liable to occur in different cases at different periods in their progress.

CHAP. VII.—DISEASES OF THE ORGANS OF LOCOMOTION.

I. RHEUMATISM. (*Rheumatic Fever.*)

Definition.—The term rheumatism is often applied to all inflammatory or painful affections of the fibrous and muscular tissues which are not clearly referrible to injury, gout, pyæmia, or any other well-recognised specific disease. With more precision it is used of inflammatory affections of the joints and other fibrous tissues which, depending apparently on some general or constitutional morbid state, have a tendency to migrate, or spread, as it were, by a kind of metastasis.

Causation.—The essential causes of rheumatism appear to be the same as those of pneumonia and many other varieties of idiopathic inflammation—namely, exposure to cold and wet, sudden chills, and long-continued exposure to any cooling influence which exercise or clothing fails to counteract. Getting wet through, and even having the feet damp and cold for any length of time, are common examples of the methods by which these agencies act. It must be allowed, however, that there are many predisposing causes which exert an important influence over the production of rheumatism. If we may accept the results of statistical enquiries, it seems clearly proved that the children of rheumatic parents are on the whole more liable to suffer than those who come of a non-rheumatic stock. Age certainly has some influence, for young infants seldom if ever are attacked with rheumatism, and the old, when they suffer, suffer mainly from its chronic forms or from its sequelæ. It is probably most common as an acute disease between the ages of ten and thirty. Sex has little influence, for although males on the whole are oftener affected than females, the difference is probably entirely referrible to the influence of their respective avocations. The most important of the predisposing causes are: first, the fact of having previously had an attack of rheumatism; and, second, the condition of the patient's health at the time of exposure. It is well known that those who are suffering from scarlet fever, those who have gonorrhœa, and women immediately after child-birth, are peculiarly liable to be attacked with rheumatism, which then often becomes modified in character. Further, inasmuch as rheumatism depends essentially on vicissitudes of temperature and other allied conditions, it is of specially common occurrence in cold, damp, and more particularly variable

climates, and during those seasons of the year in which these conditions prevail.

Morbid anatomy.—With one or two remarkable exceptions, to be presently adverted to, the morbid anatomy of rheumatism calls for little comment. The affected joints present more or less hyperæmia of the synovial fringes and of the parietal layer of the synovial membrane, with excessive effusion of synovial fluid into their cavities and exudation of serum into the soft tissues around. The former fluid may either still present the ordinary characters of synovia, or be turbid, milky, or flocculent. On microscopic examination, the epithelial cells of the synovial surface will be found swollen and plump, more or less fatty, and in some cases converted into granule cells; and similar organisms, together with cells of pus or mucus, will be recognised in the synovial fluid. Changes also go on (according to Cornil and Ranvier) in the articular cartilages. These depend mainly on nutritive irritation of the cartilage cells, which swell up, assume a globular form, and according to the usual routine (commencing with division of the nuclei) become filled with secondary cells, which speedily acquire special secondary capsules. This condition does not involve the whole extent of cartilage; but occurs in scattered spots which, when they implicate the surface, reveal themselves to the naked eye by their prominence and comparative softness. Striation of the hyaline matter of the cartilage frequently attends this process; and as this is mainly vertical in its direction, the cartilage may sooner or later acquire a velvety character. Rheumatic inflammation rarely results either in suppuration or in permanent disorganisation of the parts affected; sometimes, however, a joint gets filled with pus; sometimes ulceration of the cartilages takes place; and sometimes the tissues external to the cavity become infiltrated with inflammatory lymph, matted together, and indurated. The effects of rheumatic inflammation, discoverable post mortem in most other fibrous tissues liable to be affected, are yet more trivial than those just described, and need no special consideration.

The lesions of exceptional importance to which reference has been made are rheumatic affections of the heart and pericardium, and similar affections more rarely involving the lungs and pleuræ. These are more particularly pericarditis, endocarditis, pleurisy, and pneumonia, which are all fully discussed elsewhere under their respective names. In acute rheumatism the blood always contains a large excess of fibrine, and it is common after death to find large fibrinous coagula in the cavities of the heart and large vessels.

Symptoms and progress.—The symptoms of rheumatism are liable to great variety; and especially they are liable to vary according as the rheumatism is confined to certain organs or tissues, or becomes a more general disorder. The differences, indeed, between these two forms of the disease are so great that many regard them as entirely distinct affections.

The first form is generally traceable to the direct exposure to cold or wet of the part which becomes affected; it is often chronic in its course and intractable, yet by no means necessarily attended with indications of constitutional disorder. Sometimes it affects the fibrous structures of the soles of the feet; sometimes the muscles of the lumbar region (*lumbago*); sometimes the great sciatic nerve (*sciatica*); sometimes the intercostal muscles; sometimes the muscles of the neck or shoulder, and especially, perhaps, the sterno-mastoid (*stiff neck, torticollis*); in all of which cases the local symptoms generally suffice for the identification both of the part affected and of the nature of the disorder.

The more general and acute form of rheumatism, or '*rheumatic fever*,' as it is often called, sometimes comes on without warning, with elevation of temperature, alternate heats and chills, possibly rigors, and other usual indications of high fever, upon which, in a few hours, or perhaps a day or two, the characteristic local phenomena supervene. Sometimes, on the other hand, the acute symptoms of the general disorder suddenly supervene upon slight rheumatic pains which have been for some time flying about the limbs, or have been limited to some muscle, or fibrous expansion, or joint; or in the course of one of those varieties of localised rheumatism which have already been enumerated. But however the acute attack begins, its symptoms (apart from differences due to variations in severity and the occurrence of complications) present a great and striking simplicity.

The parts which usually and chiefly suffer are the larger joints, especially the wrists, elbows, ankles, and knees; but no joint enjoys immunity; and thus not only the hip and shoulder joints, and those of the spinal column, but those also of the clavicle, and those even of the carpus and tarsus, fingers and toes are all liable to be affected in the course of an attack. Further, the muscles of the limbs and other parts of the body by no means unfrequently suffer. The affected joints become very painful and exquisitely tender, so that the patient dares not move them and cannot bear that they be moved for him or touched, or even that his bed be shaken. They usually also get more or less obviously swollen, partly from effusion into the synovial cavity, partly from inflammatory infiltration of the surrounding tissues. This swelling is always most manifest in connection with those joints which are least thickly covered, especially, therefore, those of the hands and feet, and the wrists, elbows, ankles, and knees. It is mainly in these same joints, and along the course of the sheaths of the tendons in relation with them, that superficial inflammatory congestion, which is often absent, is seen. Rheumatic inflammation is generally characterised, not only by its tendency to attack joints successively, but by its relatively short duration in any one joint. Thus, for the most part, each joint which is implicated becomes painful, swollen, perhaps obviously congested, and recovers its normal condition, all in the course of a few days

or even a few hours; and as a rule neither pitting nor desquamation ensues. And thus, again, we often find that only one or two joints are acutely affected at a time; or that, if the patient complains of general implication of the joints, some present the earliest indications of inflammation, and others have attained their highest point, while most are in various stages of convalescence. It must not be forgetten that there is nothing protective in one attack of inflammation of a joint against subsequent attacks in the same joint; and that hence the implication of any one of these organs may be repeated indefinitely in the course of the same attack of rheumatism.

The other symptoms of the disease are in some respects almost as characteristic as the joint affection itself. The temperature is sometimes elevated only a degree or two, rarely rises above 105°, and generally falls short of this maximum by one or two degrees. It is liable to diurnal variations, which, although there is commonly a morning remission and an evening exacerbation, are on the whole irregular and untypical. Rigors are sometimes present. The skin almost invariably yields excessively copious sweats, which (although not actually more acid than healthy perspiration) are attended with a peculiar and almost pathognomonic sour smell. These, by their profuseness and long continuance, generally induce a more or less abundant eruption of sudamina, which are often seated on congested bases, and then apt to be mistaken for eczema. The pulse is quickened, but not generally in adults to beyond 100 or 110; it is regular, and as a rule more or less full and bounding. The respirations are somewhat accelerated; the tongue is for the most part thickly coated with a moist creamy fur, and occasionally becomes dry, brown, and fissured. There is failure of appetite with excessive thirst. The bowels are usually constipated. The urine is scanty, of high specific gravity, high-coloured and acid; contains abundant urates, which, with crystals of uric acid, commonly deposit on cooling; and presents an excess of urea and extractives, with a deficiency of chlorides. The patient is restless, sleepless, often pallid, wears an aspect of weariness, anxiety, or pain, but rarely presents delirium or other forms of mental disturbance.

There is no definite limit to the duration of acute rheumatism. Sometimes the patient recovers completely in the course of a day or two, or of a week; more commonly the disease persists for several weeks; and not unfrequently it becomes chronic, or is continued by successive relapses for a much longer period. It is generally observed that those cases in which the small joints are specially affected are of longer duration than those in which the larger joints mainly suffer. And, further, it not unfrequently happens that the febrile symptoms subside, while certain of the joints pass into a chronic condition of disease. Sometimes, owing to relaxation of the ligaments, certain joints remain more or less weak for an almost indefinite time; sometimes they continue stiff, swollen and tender; sometimes dislocation takes place, or

the opposed surfaces cohere, or the soft tissues around get matted together with chronic inflammatory infiltration, and the joints consequently become fixed or otherwise permanently impaired. Suppuration seldom occurs.

The complications of rheumatism are numerous and important. It may, however, be a question whether some of the so-called complications should not rather be regarded as integral parts of the disease equally with the inflammation of the joints. Rheumatism is characterised essentially by inflammation of the fibrous structures; for the most part (as has been pointed out) it is those of the joints which suffer, but those connected with muscles, nerves, and fasciæ are also liable to be involved. But of all, excepting those of the joints, the fibrous structures of the heart are most frequently implicated; and, indeed, the heart in this respect might be regarded as one of the joints, for it is at least quite as liable to suffer as any one of them. In a large proportion of cases of acute rheumatism, especially in young persons, the heart becomes involved in the course of the disease; occasionally the heart affection precedes that of the joints; and it may even be the only local rheumatic manifestation. The exact numerical relation between heart disease and rheumatism is very difficult to determine, partly because slight attacks of pericardial inflammation and the scanty formation of warty masses on the auricular aspect of the mitral valve may very readily escape detection during life, partly because, when once an attack of rheumatism has occurred with distinct cardiac complication, it is often impossible to be certain whether or not in subsequent attacks further cardiac mischief has accrued. The endocardium suffers more commonly than the pericardium. The symptoms and consequences of the various cardiac lesions are all fully discussed elsewhere; we may, however, point out that the supervention of cardiac complication is often attended with such slightly pronounced symptoms that it may either pass unnoticed or only be discovered on casual physical examination, while, on the other hand, its symptoms are sometimes so grave and dangerous that they entirely overshadow those of the general rheumatic attack. It need scarcely be said that, in every case of rheumatism, no matter how slight it is, the condition of the heart should be carefully watched. Pleurisy, pneumonia, and bronchitis, again, are not uncommon complications of acute rheumatism; of these, pleurisy is probably the most characteristic. Peritonitis, too, occasionally supervenes; as also jaundice and inflammation of the iris and sclerotic. Skin eruptions are specially apt to occur in the course of rheumatism. Reference has already been made to the frequent presence of sudamina; other eruptions are mainly varieties of erythema or roseola, and especially of those varieties included by Hebra under the generic term of erythema multiforme. They are (to give them their specific names), e. papulatum, e. circi-

natum, e. marginatum, and, besides these, e. nodosum, and, according to Trousseau, erysipelas.

It has already been pointed out that uncomplicated rheumatism, however severe it may be, is rarely attended with cerebral disturbance; nevertheless it occasionally happens that symptoms referrible to the central nervous organs break out with more or less of the suddenness that characterises the onset of cardiac mischief; or that of each attack of joint inflammation. The occasional severity and fatal character of these nervous complications has not unnaturally led to the belief that the membranes of the brain or cord have become implicated in the same way as other fibrous structures, and it is not improbable that in some cases this actually takes place. It must be acknowledged, however, that post-mortem examination rarely gives evidence of such implication. In some cases (especially if there be recent heart disease or pulmonary complication) the patient may have that kind of delirium which so often attends pneumonia and various specific febrile diseases —a delirium mostly occurring between sleeping and waking, and from which he can be easily roused. In some cases the patient has (more or less gradually developed) some form of mental alienation; while quiet in manner he gets suspicious and sly, taciturn and morose, has hallucinations, hears voices, sees visions, believes that he shall be poisoned or murdered or that the police are on his track, and may at any moment become violently maniacal. In some cases he becomes hemiplegic, paraplegic, or choreic, or even suffers from tetanic spasms, with lock-jaw and risus sardonicus. From all such conditions he not uncommonly recovers completely, but sometimes they are the precursors of coma terminating in death. Fatal coma is ushered in variously, with lowness of spirits, with insanity, with delirium or typhomania, with giddiness, headache, singing in the ears, or affection of the sight, with paralysis, or with convulsions. Sometimes it comes on suddenly with an apoplectic seizure: and death may ensue in from twelve to twenty-four hours, or even in four or five hours from the first appearance of the nervous symptoms. It has been observed that in some of these cases there is during the attack either an excessive flow of limpid urine, looseness of bowels, or both of these conditions.

Lastly, in relation to the complications of rheumatism, it must be pointed out that, although as a rule the temperature in this disease does not exceed 105°, it occasionally rises with great rapidity to 107° or 108°, or even 110°, 111° or 112°, and that such excessive rises are almost invariably of fatal augury. They always occur in association with some of the complications which have already been discussed, such as acute cardiac or pulmonary disease, or, above all, cerebral symptoms. Their connection with the first two complications is not difficult to understand; their connection with the last is certainly obscure, and none the less so that in some of the cases fatal with brain

symptoms the temperature presents no unwonted rise. It seems probable, however, that both the nervous phenomena and the hyperpyrexia are alike dependent on molecular disintegration referrible to the presence of some poisonous matter developed in the course of the disease and circulating with the blood; in connection with which suggestion it may be pointed out that the skin not only as a rule ceases to perspire profusely, but often becomes dry and harsh. Dr. H. Weber draws attention to the close resemblance subsisting between these cases and cases of sunstroke. The relations of rheumatism, through cardiac disease, with chorea and embolism are fully considered elsewhere.

It will be readily gathered from the foregoing account that, independently of all so-called complications, rheumatism is liable to present within certain limits many varieties of character. It may be acute, or it may assume a chronic form, and may, in fact, continue with relapses for months or years; it may attack chiefly the larger joints, or it may specially, as it were, select the smaller ones; instead of temporarily involving joint after joint in irregular succession it may spend its force on one or two and damage them irremediably; it may cause inflammation, mainly of the synovial membranes with effusion into the synovial cavities in one case, mainly of the soft tissues around the joints with infiltration of these tissues in another, or mainly of the fibrous sheaths of muscles or nerves in a third; when developed in connection with gonorrhœa it is peculiarly apt to become intractable and to lead to permanent injury of the affected joints; when it arises after childbirth or in the course of some of the specific fevers, the inflammation it evokes may assume a suppurative character. Yet, however long the duration of rheumatism, or however severe or various it may be in its local manifestations, it is seldom dangerous to life, unless it be through the instrumentality of one or other of the more serious complications which have been discussed; but these are fatal in a high degree, sometimes immediately, more frequently at a later period of life, in consequence of the more or less slowly developed effects of the organic lesions of vital organs which they induce.

Pathology.—It remains to say a word or two in reference to the pathology of rheumatism. Is it a local disease, or is it a constitutional disease? Does it depend on the presence of some specific poison circulating in the blood, on the action of the blood-vessels, on the action of the vaso-motor nerves, or on what? These are questions of considerable difficulty, and impossible to discuss fully in the space at our disposal. It seems to us, however, that there is little or nothing in rheumatism, in respect of its proximate cause, to distinguish it from pneumonia, bronchitis, nephritis, erysipelas, or any other example of local inflammation caused by exposure to cold, or cognate conditions. If these be constitutional diseases, so is rheumatism; if they be local diseases, rheumatism also is in the same sense a local disease—a disease,

that is to say, involving a special tissue, but one which happens to be largely distributed throughout the system. That the blood becomes abnormal in rheumatism is certain, and especially it is certain that it contains an excessive quantity of fibrine and of the products of disintegration of tissue; but these are the mere consequences of the rheumatic process, and have no more to do with the production of rheumatism than the similar condition of the blood in pneumonia has to do with the production of pneumonia. The proximate cause of rheumatism has been largely held to be a poisonous substance circulating in the blood, and the copious perspirations have been regarded as an effort of nature for the elimination of this poison; it has even been maintained that the poison is either lactic or some other acid. No excess, however, of lactic or any other acid has as yet been detected in the blood or perspiration of rheumatic patients, and if there be a rheumatic poison, which is possible, its discovery is in the future.

Treatment.—Innumerable remedies have been vaunted for the cure of rheumatism, yet it remains one of the most unmanageable complaints which physicians can be called upon to treat. Some advocate the use of iodide or bromide of potassium in frequent medium doses; some that of nitrate of potash in daily quantities varying between 1 and 3 ozs. largely diluted and taken as a drink; some that of alkalies, and more especially of the bicarbonate of potash in doses of from 20 to 30 grains given every hour or two; some recommend colchicum, some veratria, some guaiacum, some quinine, and some opium, in quantities sufficiently large and sufficiently frequently repeated to induce their respective specific actions. Dr. Garrod prefers a combination of quinine and bicarbonate of potash in about 5 and 30 grain doses respectively. Others trust mainly to local treatment: simple hot fomentations, hot fomentations with which alkalies and laudanum have been mixed, counter-irritation by means of spirit or turpentine, mustard plaisters or blisters. Blisters especially have been recently brought into prominent notice by Dr. Herbert Davies. Others, again, trust to 'packing,' or to vapour, hot-air, or hot-water baths.

By far the most valuable and efficacious treatment of acute rheumatism is that by salicylic acid or salicylate of soda. This, if given in sufficiently large and frequently repeated doses, has a marvellous power of reducing fever, and at the same time of relieving and cutting short the local inflammations and the pain attending them. Of the above preparations the salicylate is the most convenient and the best; but either may be given to an adult in 20 or 30 grain doses every two or three hours. The continuance of the drug must be determined by its effects. So soon as the temperature has fallen to the normal and pain has subsided it must either be discontinued or given in smaller or less frequent doses. It is necessary to watch the patient carefully during its administration, for various undesirable, if not dangerous,

symptoms are liable to supervene and to compel the discontinuance or diminution of the medicine. Among these the more important are albuminuria, sickness, noises in the ears, deafness, and especially delirium.

As a general rule a rheumatic patient should be kept in a comfortably arranged bed, well covered with bed-clothes, and protected if necessary by mechanical means from their undue pressure; perspirations should be encouraged, and the inflamed joints covered with cotton wool; pain should be relieved and rest obtained by the administration of opiates; thirst should be appeased and secretions encouraged by the administration of abundant diluents, such as lemonade, soda-water, milk, beef-tea, and broths; and nutrition should be maintained by the use of such food (mostly fluid and farinaceous) as the patient can be persuaded in reason to take. He should be placed in a warm room, well ventilated, yet free from draughts, from which indeed he should be protected by curtains. In addition some one of the lines of medicinal treatment above indicated may be pursued, or counter-irritation may be practised. As regards the use of blisters, we may state that they do, according to our own experience, afford almost immediate and marvellous relief to the pain of the inflamed joints in the neighbourhood of which they are placed, and that they may be applied to joint after joint in the progress of rheumatism without any ill effect whatever, but that they do not cut short the progress of the inflammation which they relieve, and have no influence whatever over the general progress of the rheumatic attack. It may be added that colchicum is said to be specially efficacious when the rheumatism is attended with marked dropsy of the synovial cavities; and that iodide of potassium or guaiacum is considered to be chiefly beneficial in chronic cases. During convalescence from rheumatism, great care should be taken to avoid cold and draughts, and the patient should be warmly clothed in flannel. He should, moreover, be put on a course of quinine and iron or some other tonic, well fed, and if necessary removed for a time at least to some more genial neighbourhood.

When rheumatism becomes chronic, or rheumatic pains are a source of trouble from time to time, or the patient suffers from rheumatism of certain fasciæ, muscles, or nerves, various measures are open for us to adopt for his relief. Hot-air baths, vapour-baths, hot-water baths, Turkish baths frequently repeated, are often exceedingly valuable. Counter-irritation, especially by means of blisters or stimulating liniments, hot fomentations, the application of belladonna or aconitia, or even the removal of blood by leeches, may be of more or less benefit. Opiates, especially given by subcutaneous injection, are often of marvellous efficacy. For general treatment, we may have recourse to the drugs which are supposed to be serviceable in the acute form of the disease; but those which are most likely to be of use now are probably iodide of potassium, guaiacum, quinine, iron, and other varieties of tonics.

For the treatment of the various complications of rheumatism we must refer the reader to the articles in which these affections are specifically considered. We may, however, observe that when cerebral or spinal symptoms manifest themselves, it is generally advisable to act freely on the bowels, to employ revulsive treatment, and to place our trust (as regards internal remedies) in opium and diffusible stimulants. If hyperpyrexia come on — if the temperature rise above 105° or 106°—then it may be advisable to reduce it by the application of external cold. This may be done either by sponging the patient's body with tepid or cold water, or by surrounding him with sheets kept moist and cool by pouring water over them from time to time, or best of all by placing him in a bath, the temperature of which may at the beginning stand at about 98°, but which is allowed to cool gradually to 60° or 70°. The patient may be subjected to such treatment for half an hour or even an hour at a time; but the propriety of continuing or determining it must be judged of by his condition. It should not be continued after he begins to shiver or look cold, or after his temperature has been reduced to the normal. But if the temperature rises it may need to be repeated frequently and at short intervals. There is no doubt that patients are often temporarily benefited by this treatment in a remarkable degree. It is less certain that their ultimate recovery is materially promoted by it.

II. RHEUMATOID ARTHRITIS. (*Chronic Rheumatic Arthritis.*)

Definition.—This affection, which consists essentially in a chronic irritative outgrowth of the cartilages and synovial fringes of the joints, associated with progressive destruction of those parts of the cartilages which are most subjected to pressure, has been described under various names, among which may be mentioned 'chronic rheumatism,' 'chronic rheumatic arthritis,' 'nodular rheumatism,' and 'arthritis deformans.'

Causation.—It occurs far more frequently in women than men; and comes on in them mostly, it is said, about the period when menstruation ceases. It may, however, commence at any time of life, and has been recognised even in young infants. Its cause is obscure. But it is certain: that many of those who suffer from it have had acute rheumatism of the ordinary type at some earlier period of life; that in some cases its commencement may be clearly traced to those conditions which are productive of acute inflammation; and that most of those who suffer from it are especially sensitive to vicissitudes of temperature and changes of season. The subjects of this affection are always more or less anæmic, but whether anæmia and debility are to

be regarded as anything more than predisposing causes in some cases, or as consequences in others, is exceedingly doubtful.

Morbid anatomy.—In rheumatoid arthritis the morbid processes are confined to the articular cartilages and synovial fringes. The central areæ of the cartilages, to a variable but gradually increasing extent, acquire a velvety or villous character, get worn down by degrees, and finally disappear, leaving the subjacent bone exposed, which then assumes an ivory-like compactness and smoothness. But while the central portions are thus disappearing, the margins form nodular outgrowths of extreme irregularity, in size, shape, and arrangement. The synovial fringes take part in the hypertrophic process, and form bulbous or pyriform excrescences. Moreover, in size, and often collected into clusters of more or less complex fibroma, are at first fibrous, but soon become the seat of cartilaginous growth; and both they and the ecchondroses tend to ossify, and often after a while get converted wholly into bone. These outgrowths in some cases blend with the osseous structure of the epiphysis, in some cases remain connected with it by fibrous cartilaginous pedicles, and occasionally break off. The gradual progress of the disease leads to the lateral expansion of the joint-surfaces and to extreme irregularity with nodular enlargement of the margins of the joint-ends of the bones, and to more or less dislocation, deformity, and immobility. All joints are liable to be thus affected—those of the hands and feet, those of the arms and legs, those of the jaws, and even those of the spine. The early changes which take place in the cartilages in this affection are enlargement and proliferation of the cartilage cells. In the central areæ of opposed cartilages, where they are subject to constant mutual pressure, the enlarging cavities which contain the multiplying cells communicate with one another in vertical linear series, and opening at the surface discharge their cellular contents into the synovial cavity; by this means the hyaline substance becomes honeycombed by vertical pits, or split into vertical columns, and thus acquires its characteristic velvety appearance. The proliferation, however, of the cartilage-cells at the periphery (where growth is less interfered with) and of the synovial fringes results in permanent overgrowth of these parts, and in that further development of them which has been described.

Symptoms and progress.—The symptoms of rheumatoid arthritis are mainly those which are due to the gradual advance of deformity, dislocation, and loss of mobility in the affected joints, and to a tendency to gradual implication of most or all of the joints of the body. But with these are associated: more or less pain and tenderness, rarely acute, in the affected parts, coming on at irregular intervals and attended with more or less febrile disturbance; wasting of the muscles connected with the diseased joints, with spasmodic cramp-like pains in them; and anæmia.

The disease, as has been stated, may follow immediately or remotely

on an attack of acute rheumatism; but in many cases it is chronic or sub-acute from the beginning. The patient complains, perhaps, of slight pain, tenderness and swelling in one or more of the joints, probably the knees, wrists, or fingers, and of slight feverishness; but ere long, with rest and confinement to the house, the symptoms subside. Then, after a short interval, the phenomena recur, probably with greater intensity; and possibly other joints besides those first affected now become implicated. Again, perhaps, the symptoms subside. These attacks, however, continue to recur for the most part at shorter and shorter intervals, to implicate a gradually increasing number of joints, and to leave them (in the intervals of subsidence) still swollen and tender, and water over them more and more useless. At length, after the lapse of some months, or it may be years, the patient becomes thoroughly crippled; most of his joints, or all of them, are swollen, distorted, and more or less rigidly fixed; all his muscles are wasted; and his arthritic and muscular pains, now never wholly absent, are liable to frequent exacerbations, especially in connection with changes of temperature.

Rheumatoid arthritis usually commences in the hands, and more especially in the metacarpo-phalangeal joints of the fore, middle, and ring fingers; the wrists and knees are also early implicated. The upper extremities as a rule suffer before the lower extremities; and although the metatarso-phalangeal joint of the great toe generally becomes affected in the course of the complaint, it is rarely or never the primary seat of attack. The articulations of the jaws and of the spine are for the most part implicated at a late period. The nodulated condition of the joint-ends of the bones is usually most distinctly marked in the finger joints, the ball of the great toe, the wrists, elbows, and knees; it is in these same joints, too, and in the hips, that imperfect dislocations most commonly occur. When the joints are rigid and fixed, they usually occupy the position of flexion; the thighs are flexed on the abdomen, the legs on the thighs, and the fore-arms on the upper arms—in the last case with some degree of pronation. The hands generally continue in a straight line with the fore-arms, or present some degree of tilting towards the ulnar side, but the fingers acquire various and strange distortions. The most frequent form is that in which, while the first and third phalanges are flexed, the second or intermediate phalanx is extended. The thumb is usually extended. Rheumatoid arthritis, though generally a progressive disease, and incapable of cure, occasionally remains limited to one or two joints, or becomes arrested in its progress, or even (so far as the structural changes which have taken place permit) undergoes a more or less perfect cure.

Pathology.—The relation between rheumatoid arthritis and acute rheumatism is not easy to determine. It is quite certain that acute rheumatism very seldom induces the characteristic morbid processes of

the former disease; and that rheumatoid arthritis is rarely attended with the profuse perspirations, the febrile urine, and the visceral complications which belong to acute rheumatism. On the other hand, rheumatoid arthritis is essentially an inflammation of the very structures which are mainly implicated in acute rheumatism; the joints become successively and symmetrically involved as they do also in the latter disease; and, with reference to the absence of sour perspirations and the like, it must not be forgotten that these may be entirely absent in cases of chronic, sub-acute, or muscular rheumatism; and as regards visceral complications, Trousseau shows that peri- and endocarditis are sometimes present in these cases, and that even cerebral mischief occasionally supervenes. Moreover, as Garrod points out, inflammation of sclerotic and other fibrous textures now and then attends rheumatoid arthritis. On the whole, we are inclined to regard it as a chronic inflammatory process, which is not necessarily, but is in a large number of cases, a sequela of acute rheumatism.

Treatment.—For the general treatment of rheumatoid arthritis we must refer to what has already been said in reference to the treatment of acute rheumatism. For the most part, however, we must trust to local measures and to constitutional treatment calculated to improve the general health of the patient. Locally, friction, counter-irritation, the inunction of the parts with preparations of iodine or mercury, the maintenance of the joints in one position by suitable apparatus, are all more or less important. Hot fomentations again are valuable, and especially perhaps (as recommended by Trousseau) the burying of the joint in sand heated up to 140° or 150°, keeping it there for an hour or two at a time, and repeating the operation three times a day.

III. GOUT. (*Podagra.*)

Definition.—Gout is a disease which is characterised by the deposition of urate of soda in a crystalline form in the cartilages and other textures of joints, and elsewhere among the fibrous tissues, and by recurrent attacks of articular inflammation. It is usually attended also with constitutional symptoms and grave lesions of important organs.

Causation.—Gout is mainly a disease of middle and advanced life, and of the male sex, and generally comes on between the ages of thirty and forty-five. It is sometimes, however, met with at or about the period of puberty, and has occasionally made its first appearance as late as the eightieth or even ninetieth year. In women it rarely shows itself until after the cessation of the menstrual flow. The influence of hereditary predisposition in the production of disease is probably nowhere more clearly evinced than in the history of gout; and, indeed, Dr.

Garrod's experience leads him to the belief that more than half the total number of gouty patients have clearly inherited the gouty proclivity from their parents. On the other hand, it is certain that gout is largely induced by habits of life, and that even where an hereditary taint exists, the influence of habits in accelerating the first attack or in postponing it, or even in preventing the occurrence of the disease, is still very considerable. As regards habits, it seems to be universally admitted that long-continued indulgence in alcoholic beverages, long-continued over-eating, especially of animal food and of rich dishes, and prolonged insufficiency of exercise, are (especially in combination) powerful agents in the causation of gout. It is, however, generally held that all alcoholic beverages are not equally injurious in this respect, that the distilled spirits are comparatively innocuous, that the light wines, claret, hock, moselle, and the like, are also fairly wholesome, but that the strong wines, sherry, and madeira, and above all port, and malt liquors, are all virulent gout-producers. But on what, it may be asked, do the injurious effects of alcoholic beverages depend? If, as seems reasonable to assume, they are due to the alcohol which they contain, how can we accept the statement that the distilled spirits are almost harmless, while bitter ale and porter are highly poisonous? If, on the other hand, the alcoholic constituent be acquitted, must we refer them to the comparatively simple matters which give to alcoholic beverages their respective flavours, their colours, or their body—matters which are, most of them, not special to such beverages, are most of them certainly not unwholesome, and individually form an insignificant percentage of the whole? We must confess our distrust of the evidence which, while accusing alcoholic drinks of causing gout, acquits the alcohol itself. On similar grounds we venture to submit, notwithstanding almost universal testimony to the contrary, that port is no more injurious than sherry or madeira, or other wines of equal strength. It is probably less in consequence of the port which they drink than of the association in their case of over-drinking, over-feeding, and want of exercise, that the higher classes suffer more frequently from gout than those who occupy a lower station of life. It must be added that fatigue, exposure, indigestion, and whatever impairs the health, and injuries inflicted on joints, are all apt to bring on attacks of gout in those who are liable to the disease. The impregnation of the system with lead appears to be peculiarly powerful in inducing a susceptibility to gout.

Morbid anatomy.—The morbid phenomena of gout are chiefly manifested in the joints and surrounding tissues. The earliest appearances are furnished by the superficial portions of the articular cartilages, which seem dusted, so to speak, with spots and patches of an opaque white colour. As the morbid process extends, the cartilages become more and more generally infiltrated, until they look like a mere mortary incrustation of the joint-surfaces of the bones. Later still, similar

mortary patches appear imbedded in the substance of the synovial membranes, and gradually involve them more or less completely; and at the same time, or later, masses (which eventually vary perhaps from the size of a pea to that of a filbert) accumulate in the substance of the soft tissues surrounding the joints, in the bursæ, and in the cancellous tissue of the subjacent bones. The changes do not end here. The infiltrated cartilages lose their vitality, become brittle, gradually eroded, and finally removed, exposing the bone beneath, which itself may sooner or later undergo destructive changes. The margins of the affected cartilages, on the other hand, not unfrequently become irritated into overgrowth, and form nodular enlargements like those of rheumatoid arthritis. The accumulations of mortary matter in the tissues about the joints, which constitute *chalk-stones* or *tophi*, gradually provoke erosion of the swollen and congested tissues which cover them, and finally an opening is formed from which they escape. The appearances above described are due to the deposition in the substance of the cartilages, and elsewhere where such deposits are found, of needle-like crystals of urate of soda, arranged for the most part in dense, opaque, stellate clusters. This deposition appears to commence within the cells, and although the needle-like rays extend thence into the surrounding intercellular substance, it is still to the cells that the crystalline formation is mainly confined. Gouty formations, as a rule, first manifest themselves in connection with the metatarso-phalangeal joints of the great toes, usually the right, and may remain thus limited for a considerable length of time. But gradually other joints (and for the most part with more or less symmetry of arrangement) become involved—the smaller ones, as a rule, first, the larger ones at a later period. Thus, after the metatarso-phalangeal joints of the great toes, the other toe-joints and the joints of the tarsus, fingers, and carpus, the sterno-clavicular articulations, the ankles and wrists, the knees and elbows, and finally the hips and shoulders, and other joints, become successively the seats of disease. The joints connected with the laryngeal cartilages also occasionally suffer. Gouty deposits, moreover, are apt to form along tendons, chiefly in the neighbourhood of gouty joints; beneath the periosteum of the tibiæ and other bones; in the course of the smaller vessels and nerves; and in connection with the perichondrium of the external ear, the tarsal cartilages, and the sclerotic coat of the eye. In the ear they mainly affect the convex edge of the helix; in the tarsal cartilages, those portions which immediately adjoin the edges of the palpebral orifice.

The ultimate effects of gout upon the joints are in most cases very serious. They get irregularly swollen, partly from inflammatory and gouty infiltration of the tissues which surround them, partly from the changes which have been going on in their interior. Accordingly the irregularity is not, as in rheumatoid arthritis, limited to the joint-ends of bones, but occupies the intermediate regions at least equally, and

probably in a still greater degree. The articulations become more or less fixed, generally in some inconvenient position, and may even be dislocated: these results being due in various degrees to the changes which have taken place in the soft tissues around, to uratic infiltration and loss of suppleness in the synovial membranes and ligaments, and to actual ankylosis, which sometimes follows the complete removal of the cartilages. Chalk-stones form more or less abundantly in the tissues external to the joint-cavities, adding to the apparent bulk of the joints and to their knotty irregularity, and finally become discharged through ulcerated openings, which, still secreting large quantities of chalky matter, may remain patent for years. The deformities and other ulterior changes here enumerated occur most frequently, earliest, and with greatest severity, in the joints of the hand; next in order in those of the feet; then in the wrists, elbows, ankles, and knees; and finally in the hips and shoulders.

It is rare to find in the necropsy of gouty persons that all other organs save those of which the morbid conditions have just been described are in a perfectly healthy condition. It could scarcely be expected, indeed, when one looks to the circumstances under which, as a rule, gout arises, that the internal viscera should escape those degenerative changes which so commonly follow long-continued persistence in bad habits, or attend that tendency to premature decay which some of us unfortunately inherit. It is not surprising, therefore, that gouty patients are liable to have degenerated arteries, valvular lesions and other morbid conditions of the heart, emphysema of the lungs, cirrhotic liver, and contracted granular kidneys. The last lesion, indeed, is so common in gout that it is not unfrequently termed the 'gouty kidney.' The kidneys of gouty patients, moreover, often present, especially in the cones, linear aggregations of a buff-coloured material, which is, in fact, a deposit of urate of soda, either in stellate crystals in the matrix, or in an amorphous form in the tubules. Concretions of the same material sometimes adhere to the mammillary processes. These precipitates are not, however, characteristic of gout, and are frequently found in persons who have no gouty tendency, and even in new-born children.

Symptoms and progress.—It has been distinctly shown by post-mortem examination that the gouty deposit takes place in the articular cartilages long before the joints become inflamed or give any clinical evidence of the nature of the process which is going on in them—a fact which is confirmed by the total freedom from inflammation and pain which usually attends the formation of those uratic concretions which are met with in connection with the aural cartilages and periosteum. And hence it may be assumed that at any rate a very large proportion of those who ultimately become distinctly gouty have been really gouty for a considerable time previously to the first considerable outbreak; and hence also it is easy to understand that in many cases

the first so-called 'attack' may have been preceded by premonitory symptoms, such as occasional pain or tenderness in one or both great toes, or other of the smaller joints, such as those of the fingers, wrists, ankles, or clavicles.

The first 'attack of gout' almost invariably comes on suddenly, with pain and swelling in the ball of one of the great toes, usually the right. Moreover it occurs for the most part early in the year, and almost without exception in the night time. The patient goes to bed probably in his usual health, but wakes about two or three o'clock in the morning with severe pain in the metatarso-phalangeal joint of one of his great toes. The agony is sometimes so intense that he dares not move the affected limb; he cannot bear the pressure of the bed-clothes, or even the slightest jar to his bed or the slightest movement in his chamber; his sufferings, too, are often aggravated by cramps and involuntary startings in the muscles of the leg; he becomes restless and hot, shivers, sometimes has repeated rigors, and, after tossing about for some hours, falls into a perspiration; and then, somewhere about the time when he should be thinking of getting up, he falls into a gentle sleep, from which, in the course of a few hours, he awakes, refreshed and comparatively easy, but with the great toe joint swollen, tense, and vividly red, and with the superficial veins of the foot, and probably some of those extending up the leg, unusually distinct and full. He most likely continues comparatively well throughout the day, and may even be able to limp about on his maimed limb; but with the advance of evening, or it may be in the early hours of the ensuing morning, he has a more or less severe recurrence of the local pain and febrile symptoms which marked the first attack, to be again followed after the lapse of a few hours by a second intermission. These nocturnal exacerbations, succeeded by matutinal remissions (lasting usually till evening), come on with comparative intensity for two or three successive nights, and then gradually diminish in severity, until at the end probably of a week or ten days all febrile symptoms and all acute suffering have passed away. But the affected joint probably remains swollen, weak, and tender for a week or two longer. During the attack, the ball of the toe becomes, as has been stated, tense, swollen, vividly red, generally more or less shiny and exquisitely painful and tender. Most of these conditions attain their maximum usually by about the second day, after which the pain and tenderness gradually subside, and the redness acquires a dusky hue; but the swelling probably still increases for a time, and even extends far beyond the limits of the seat of inflammation. Much of this, indeed, is now due to simple œdema, and the parts pit on pressure. The swelling disappears in its turn and desquamation follows. The febrile symptoms, from which the patient suffers during his attack, are, as has been indicated, of a remittent type, and attended not only with shiverings or rigors and perspirations, but frequently also with furred

tongue, loss of appetite, thirst, constipation, and a febrile condition of urine.

It occasionally happens: that even in the first attack of gout both great toes are simultaneously or sequentially implicated, or that not only the toes, but the ankles, knees, and other joints successively suffer, in which case especially the affection may present a close resemblance to acute rheumatism; or, again, that the first attack, instead of subsiding speedily, as it usually does, continues by a series of successive outbreaks of no great intensity in the same joint for weeks or months. It sometimes also happens that the first attack of gout is in the ankle, knee, or some other joint than that of the toe—a circumstance which in some cases is obviously due to an injury or some other lesion of the part.

The first attack of gout may also be the last. But far more commonly a second attack supervenes sooner or later, occasionally not for eight or ten years, sometimes after an interval of a few months only, most frequently at the end of a year or two. To the second attack other attacks succeed, at first separated from one another by intervals of probably about twelve or six months, but gradually approaching one another until at length the patient, though still liable to exacerbations, is perchance never actually free from suffering. Further, each successive attack, as a rule, implicates a larger and larger number of joints: those joints, however, which have been most frequently affected generally suffering most severely. Gradually these grow lumpy, deformed and rigid; the patient becomes more and more crippled; chalkstones form and discharge themselves through ulcerated openings; the general health deteriorates; and death, usually dependent on some visceral complication, finally ends the scene. The pain which attends the later attacks of gout, although more continuous, is rarely so acute as that of the earlier outbreaks; and, further, the degree of disorganisation of joints and the amount of urate of soda deposited in or about them are by no means necessarily related to the frequency or severity of the attacks of inflammation from which the joints have suffered.

The condition of the urine in gout, which has been carefully investigated by Dr. Garrod, has already been referred to. During the febrile paroxysms it is scanty and high-coloured, of high specific gravity, and generally deposits on cooling an abundant sediment. It contains relatively to its bulk an excess of urates; but the total amount of these passed in the twenty-four hours is absolutely less than in health. The urea is also probably somewhat diminished. In the chronic form of the disease the urine is pale, abundant, of low specific gravity, generally yielding no deposit, and presenting (as in the febrile stage) a diminished daily quantity of urates and urea. It often contains a small amount of albumen, with hyaline or granular casts. The condition of the blood has also been investigated by Dr. Garrod, who finds: that during

the inflammatory attacks of acute gout it contains urate of soda in relatively large abundance, while none can be detected in it previous to the occurrence of inflammation or after its subsidence; but that, when the gout assumes a chronic or inveterate character, urate of soda is present in the blood both during the exacerbations and in the intervals. He has also found in the blood oxalic acid, which he refers to the decomposition of the retained uric acid, and urea the presence of which is probably always dependent on associated renal disease.

In the foregoing account many of those phenomena which by some authors are regarded as among the most important in the history of gout have been passed over in almost complete silence. We refer, on the one hand, to the premonitory symptoms referrible to functional lesions of various organs, and, on the other, to the various sequelæ or complications which from time to time present themselves. Gouty persons are usually more or less dyspeptic; and it is not unnatural, therefore, that dyspeptic symptoms should in a large number of cases precede the gouty paroxysms, and persist more or less during the intervals between successive attacks. Among the premonitory symptoms, therefore, may be enumerated epigastric discomfort, pain, flatulence and eructation, with more or less constipation or disturbance of the bowels, palpitation, dyspnœa, headache, drowsiness, restlessness, moroseness, irritability and violence of temper. Such symptoms often attend the gouty outbreak, but, on the other hand, they are said frequently to be removed by it. The sequelæ and complications of gout are numerous, and may be considered *seriatim* in connection with the organs which are their seat. In connection with the nervous system occur vertigo, headache, convulsions, mania, apoplexy, anæsthesia, paralysis, hyperæsthesia, lumbago, sciatica, and various other neuralgic pains; in connection with the vascular system, palpitation, syncope, angina pectoris, and various forms of structural cardiac disease; in connection with the lungs, asthma, bronchitis, and emphysema; in connection with the gastro-intestinal tract, dyspepsia, gastralgia, irregularity of bowels, and hæmorrhoids. Further, the liver not unfrequently becomes indurated or cirrhotic, and jaundice, ascites, or melæna may ensue; the kidneys in a large number of cases get contracted, granular and incompetent, and the patient tends to suffer from the usual symptoms of chronic Bright's disease; the bladder becomes irritable or inflamed; concretions form in the urinary passages, or mucous or purulent discharges take place from them; and, lastly, skin affections often arise, especially perhaps chronic eczema and psoriasis. But none of the symptoms or lesions here enumerated is peculiar to gout; and their frequent co-existence with it is doubtless in large measure dependent on the fact that sufferers from gout are on the whole persons whose internal organs are in a greater or less degree in an abnormal condition, and whose bodily functions tend, therefore, to be imperfectly performed. It is asserted that, from exposure to cold

or other causes, gouty inflammation is apt to subside suddenly, and its subsidence to be followed by grave symptoms referrible to the stomach, heart, or nervous system. This metastatic affection, of which our knowledge is in every respect extremely unsatisfactory, is termed 'retrocedent gout.'

Gout is in general easy of diagnosis; it may, however, under various circumstances, be readily confounded with rheumatism or rheumatoid arthritis. We do not propose to rediscuss the many pathological and clinical differences which exist between these several affections. But we may recall to mind, as being specially distinctive of gout: its tendency to attack the smaller joints and lower extremities in the first instance, and mainly the metatarso-phalangeal joint of the great toe; the formation of chalk-stones, not only in connection with the joints, but with the perichondrium of the cartilages of the ear and eyelids, and in other superficial positions, where they may be easily recognised; and the presence of urate of soda in the blood. Superficial uratic concretions may be easily removed, wholly or in part, with the point of a lancet and submitted to microscopic examination, when they will be found to consist mainly of the characteristic needle-like crystals, more or less thickly aggregated. For the purpose of testing the condition of the serum of the blood, Dr. Garrod's method may be adopted. It is as follows:—about two drachms either of the serum furnished by the blood on standing, or of the fluid raised by a blister, are to be placed in a flat glass dish, somewhat larger than a watch-glass, to be acidulated with acetic acid, and to have laid in it an ultimate fibre from a piece of linen cloth; the prepared fluid is then to be allowed to stand until by evaporation it has been brought to the condition of a thin jelly, when, if there have been an undue amount of urates in the serum, the fibre will be found, on microscopic examination, studded with crystals of uric acid. To confirm the uratic character of the concretions removed from the surface of the helix, or of the crystals obtained from serum, recourse may be had to the murexide test, which consists in the development of a beautiful purple colour when a small quantity of the crystalline matter is heated with nitric acid on a porcelain surface and then treated with ammonia.

Pathology.—It is, we believe, now generally held that, although the deposition of urate of soda in the joints and elsewhere furnishes the only trustworthy evidence of the presence of gout, this deposition, the various premonitory symptoms, and the almost innumerable concurrent symptoms, complications, and sequelæ, are all traceable to the presence of urate of soda in the blood in undue quantity; that the disease, in fact, looked at from an earlier stage than the joint-affection, is to be regarded, not as a disease of the joints, but as an affection of the blood, a variety, as Dr. Murchison terms it, of 'lithæmia.' Dr. Murchison, who regards the presence of an excess of urates

in the blood as the consequence of functional disturbance of the liver, would naturally consider gout to have some such relationship to the liver as uræmic dropsy has to the kidney. Dr. Garrod, on the other hand, apparently inclines to the belief that the kidney, failing from some unknown cause to act efficiently in the separation of urates from the blood, is the organ mainly at fault. In the one point of view the excess of urate of soda in the blood would be due to simple retention, in the other to its excessive formation; but in either case the deposit of this substance in the joints would be regarded as eliminative, and in a sense curative. Dr. Garrod further regards the inflammation of gouty joints as destructive of urates in the blood of the inflamed part, and consequently indirectly in the whole mass of the blood. Against these hypotheses, several important considerations may be adduced. First, as Dr. Garrod clearly admits, urate of soda is occasionally present in the blood in large quantities and yet gout neither is present nor ensues; second, although the presence of this salt in the blood seems to have been universally detected during the inflammatory paroxysms of acute gout, and during both the exacerbations and the remissions of the chronic affection, Dr. Garrod points out expressly that it is not present during the intervals between the acute attacks, nor during that period prior to any attack of inflammation in the course of which its slow deposition is taking place in the joints. Surely these facts are more in accordance with the hypothesis that the urate is formed in the tissues affected and thence shed into the blood, than with that which refers the local lesions to the precipitation of this salt from the already overcharged blood. We must confess, indeed, that our own views as to the pathology of gout are very nearly those which have recently been advocated by Dr. Ord. We are inclined to look upon this disease as arising from a tendency to a special form of degeneration in certain of the fibroid textures of the body, derived by inheritance or acquired by habits of life—a degeneration characterised by the excessive formation of urate of soda in the implicated tissues, whence, on the one hand, it is discharged into the blood, on the other deposited here and there and especially in those parts (as cartilage) which are least well supplied with vessels and lymphatics. And we are disposed to regard the so-called 'attacks of gout' as being in some sense accidental—predisposed to by the gouty deposits which have already accumulated in the part, and determined by accidental injuries, exposure to cold, and generally any of those conditions which are apt to excite local inflammation. If this view be correct, we should expect to find a specially abundant formation of urate of soda in gouty tissues during the period of inflammation in them, and a specially abundant discharge of urates thence into the veins or lymphatics, or both. We know that Dr. Garrod's assertion with regard to the absence of urates from the serum furnished by blisters applied over the inflamed joints is apparently opposed to this

hypothesis; on the other hand, Dr. Ord quotes facts which are in its favour; and it must be added that the *experimentum crucis*, namely, the chemical examination of the blood obtained direct from the veins leading from the inflamed parts and its comparison with the blood of other vessels, has not yet been performed.

Treatment.—In reference to the treatment of gout we have to consider: 1. What should be done during the inflammatory attacks; 2. What should be done during the intervening periods.

As regards the first question, much difference of opinion prevails. Trousseau, following Sydenham, regards all attempts, local or general, to relieve pain or cut short the attack, as prejudicial. These views, however, are not generally held, at least in their integrity; and most physicians, we believe, would now rarely hesitate to make use of those remedies the efficacy of which has been proved by experience. The most valuable of these is colchicum, which may be given in any of the pharmacopœial forms, and of which the wine may be administered in doses of from 10 to 30 minims three times a day. Veratria has similar but less powerful anti-podagral properties. Quinine is another remedy the value of which in largish doses, 5 or 10 grains, three times a day, has been much lauded. Purgatives are often of great value; among such drugs colocynth is believed to be specially efficacious; and indeed the combination of quinine and colocynth forms the basis of some of the best known nostrums. Alkalies, again, mainly the carbonates or the salts formed by their union with the vegetable acids, are generally regarded as useful. The lithia-salts are especially recommended by Dr. Garrod in consequence of their solvent power over the uratic salts. When the inflammatory state of the joints assumes a chronic character, the above remedies may be employed, but with less vigour; and at this time guaiacum and particularly iodide of potassium are often of great value. Generally it may be said that, during the gouty paroxysm, the emunctories, above all the kidneys, should be encouraged to free action; the bowels kept freely open; perspiration promoted; and the flow of urine increased by the drinking of abundant diluents and the use of the alkaline diuretics. Dyspeptic symptoms should be counteracted, for which purpose the alkalies are again serviceable; and these, combined with colchicum, and either rhubarb or a vegetable bitter, will usually prove the most serviceable medicinal agents. As regards local treatment, the affected joints should be kept at perfect rest, and if necessary by mechanical means. Leeches and cold lotions are generally deprecated, and warmth is not always agreeable; still the investment of the joints in cotton-wool, or in bran poultices, sometimes gives comfort, and especially the application of blisters, or the wrapping of the parts in flannel or cotton-wool steeped in rectified spirit and covered with oiled silk or gutta-percha.

The question, however, of what should be done in the intervals to prevent the recurrence of the attacks is the more important one; and

the answer in general terms is not difficult. The patient should make an important reduction in the amount of alcoholic stimulants which he is in the habit of taking, or, if he can do so without detriment to his digestion and to his general health, give them up altogether; he should refrain from excesses in eating, and especially in eating flesh and rich articles of diet; he should, in fact, while not starving himself or so restricting his choice of viands as to render food unpalatable, reduce his daily consumption to the amount which nature requires, and restrict himself to what is wholesome and nutritious; he should take daily exercise, not, however, over-fatiguing himself, or insisting on overcoming by exercise the pain or tenderness of an already gout-stricken member; he should dress himself warmly, and guard against unnecessary exposure to vicissitudes of temperature; he should avoid as far as possible all overwork of the mind as well as of the body, and mental anxiety; if he suffer from indigestion or other ailment, this should be treated. Further, tonics, such as iron and quinine, and periodical baths, especially the hot bath, the hot-air vapour or Turkish bath, and change of air and scene, are often of the greatest value in the treatment of those who suffer from chronic gout. It must, however, be admitted that the rules here laid down cannot, unfortunately, always be enforced; and, further, that their strict enforcement will, in most cases, diminish only, but not eradicate, the gouty tendency.

It is in cases of this sort that recourse is constantly had to mineral waters. There can be no harm in taking alkaline or other waters provided the nature of the ingredients contained in them is compatible with the patient's requirements. The main benefits, however, which result from this practice are due to change of air and scene, to the bathing, and to the dietetic and other restrictions which are usually enjoined and submitted to.

IV. RICKETS. (*Rachitis.*)

Definition.—This is a disease of early childhood, characterised mainly by softening of the bones, with enlargement of the joint-ends of the long bones, enlargement of the liver and spleen, and coincident symptoms of general ill-health.

Causation.—Rickets rarely shows itself prior to the sixth month after birth, or later than the second year, and most commonly comes on between the twelfth and eighteenth month. Children, however, are said to have been born rickety, and in rare cases the supervention of the disease has been delayed to the fifth or sixth year. Many causes have been assigned for rickets, and yet it must be confessed that a good deal of mystery still enshrouds its ætiology. It does not appear to be

hereditary in the sense that rickety children are the offspring of rickety parents, or even of parents who are scrofulous or syphilitic; it nevertheless happens that rickets is often the appanage of certain families of children, and in such cases (according to Sir W. Jenner) it is common for the younger members to suffer exclusively, or more severely than their elder brothers or sisters. Poverty and hard living would seem to exert some influence in its production; at all events the children of the poorer classes are more frequent sufferers than those who are brought up in luxury and comfort. There is no doubt indeed that defective hygienic conditions generally favour it; but among these unsuitable feeding is probably by far the most efficient. A large proportion of rickety children have been brought up by hand, or have derived an insufficient quantity of inferior milk from inefficient nurses, and have at the same time been supplied with food which is more or less unsuitable, or even directly injurious. It is thus, probably, that delicate mothers are often indirectly answerable for the rickety state of their offspring. The influence of improper diet in the causation of rickets seems proved by the experiments conducted by M. Guérin on puppies. By removing these animals from the mother, weaning them and feeding them on raw flesh (a food unsuited for them at that early age), he rendered them, in the course of four or five months, unmistakably rickety. Rickets is said to be especially common in damp and cold climates.

Morbid anatomy and pathology.—The morbid changes which attend and characterise rickets are limited almost exclusively to the bones, liver, and spleen. And of these by far the most interesting and important are those which have their seat in the osseous system. The rickety process attacks all bones simultaneously, or nearly so, and in about an equal degree. It can, however, be best studied in the long bones. In these, the whole tissue tends sooner or later to become involved, but those parts in which growth is taking place most actively (namely the extremities and the outer surface) are primarily and principally affected. For the following account of the morbid anatomy of rickets we are mainly indebted to the investigations of MM. Cornil and Ranvier. In the normal process by which ossification extends from the already ossified shaft into the cartilaginous end, the line of advance is even and well-defined, the cartilaginous tissue immediately bounding it (to the thickness of about one-twentieth of an inch) presenting a peculiar bluish transparent aspect. In this bluish cartilaginous lamina the changes preliminary to ossification are going on actively. But it is in the layer of bone immediately subjacent to it that the earliest stage of actual ossification is to be recognised. In the former of these laminæ we find the encapsuled cartilage cells becoming larger and larger at the expense of the surrounding hyaline tissue, and giving origin to daughter-cells which themselves become encapsuled, but sooner or later yield large numbers of cells having the

embryonic character. During this process, the cavities in which the cells are imbedded increase in size, and presently communicate, more or less freely, with one another, thus forming branching channels or alveoli, full of embryonic cells, and separated from one another by areolæ formed of the surviving remnants of the hyaline matrix. In the latter of the laminæ above referred to, earthy matter is being deposited in the substance of these areolæ, and vessels are developing in the alveoli from the embryonic tissue or marrow which they contain. A little deeper, the concentric zones of earthy matter and stellate corpuscles which, by their presence within them, convert the alveoli into Haversian systems, may be recognised in process of formation.

In rickets, the bluish cartilaginous lamina becomes very irregular in form and thickness—the chief irregularity being manifested by its under surface, whence numerous processes extend into the subjacent [illegible] which also isolated patches of cartilage may sometimes be detected. The chief departure from health, however, is manifested by a lamina, corresponding in position to the second of the laminæ above described, which lies between the cartilage and the ossified extremity of the shaft (from which it is imperfectly divided), and the thickness of which varies, and in some cases is very considerable. This lamina has a marked resemblance to sponge, being, like it, both cavernous and elastic; but it is highly vascular, and its alveoli are filled with a sanguineous pulp. Microscopic examination shows the dependence of these abnormal appearances upon certain striking modifications of the process of ossification. In the bluish cartilaginous lamina, the mother-cells give origin to daughter-cells, which become encapsuled; but these, while still of large size, become encrusted with calcareous granules, which are also deposited in the intervening cartilaginous matrix. Their further proliferation is thus to a large extent impeded, and they fail to regulate the subsequent process of alveolation, which takes place as it were without discrimination—irregular cavities being formed here and there by the liquefaction of the hyaline cartilage and the contained calcareous capsules, and irregular trabeculæ resulting from the persistence of identical tissues in the intervals between them. The essential departure from health seems so far to consist in calcification of the capsules of the daughter-cells, and in the fact that the areolæ are formed, not of hyaline material only, but of this together with cartilage-cells, which are of large size, and encrusted with earthy salts. In the spongy tissue beneath the same process goes on; the alveoli (instead of getting smaller, as they do in healthy ossification, from the formation of concentric laminæ) grow larger and larger, the areolæ undergoing corresponding rarefaction and destruction, and becoming at the same time more and more calcareous. The medulla which occupies the cavities is richly provided with large delicate-walled vessels, and an abundance of ordinary granulation or embryonic tissue. The morbid processes which go on between the shaft and the

periosteum are (allowing for the anatomical differences between cartilage and periosteum) identical with those just described. The periosteum in the normal condition may be readily stripped as a membranous lamina from the surface beneath. In rickets a soft spongy formation, of variable and often considerable thickness, intervenes between them. This consists of refractive trabeculæ, formed partly of intercellular substance, partly of connective-tissue corpuscles, and infiltrated to a greater or less extent with earthy matter; and of intercommunicating medullary spaces filled with embryonic tissue and new-formed blood-vessels. It is obvious that the areolæ or trabeculæ, whether formed from cartilage at the epiphysal extremities of the bone, or from periosteum at its surface, and whether infiltrated with earthy matter or not, are continuous with the solid framework of the completely formed bone-tissue underneath; and that the embryonic or granulation tissue which fills their alveoli is continuous with the normal medullary matter occupying the Haversian canals, medullary spaces, and central cavity of the bone. And it may be added that if the rickety condition persist and extend, not only do the alveoli and the new-formed tissue enlarge at the expense of the trabeculæ between them, but the medullary tissue of the normal bone-cavities gradually acquires the embryonic character, the bone-tissue melts away around it, the Haversian canals and all other spaces consequently undergo enlargement, and general rarefaction ensues. Sir W. Jenner observes that the bones of healthy children yield about 37 per cent. of organic and 63 of earthy matter, whereas those of rickety children sometimes yield as much as 79 per cent. of organic and only 21 of earthy matter.

The general consequences of rickets are that the bones thicken (a change which is especially evident in the joint-ends of the long bones and in the edges of the flat bones of the skull) and become soft, and consequently liable to bend, or to break with the so-called 'green-stick' fracture. The enlargements are most obvious at the wrist and ankle joints, and at the elbows and knees; they are frequently also well shown at the junctions of the ribs with the costal cartilages, along the lines of the cranial sutures, and elsewhere where bones are in relation with epiphysal cartilages. The curvature of bones comes on somewhat later than their manifest thickening, and usually proceeds from the lower part of the skeleton upwards. Its direction is determined partly by that of the normal curvatures of the bones and partly by the direction and force of the mechanical influences which act upon them. The tibiæ and fibulæ usually bulge forwards and outwards, and the femora follow suit; when, however, the rickety condition appears late, the bending of the legs is often in the opposite direction, and the child becomes knock-kneed. The radius and ulna for the most part acquire a curve with the convexity facing backwards; but the shape of the humerus is often determined by the attachment of the deltoid.. The shoulders get narrowed by the shortening of the

clavicles due to exaggeration of their natural curves. The back becomes bent in the same sense, and often more or less twisted: by increase of the cervical curve the head tends to be thrown backwards, and the face to be directed upwards and forwards; exaggeration of the dorsal and lumbar curves is often attended with lateral deviation connected with rotation of the bodies of the vertebræ upon their axes. In children who have not yet walked the lumbar curvature is lost in that of the dorsal region, and both combine to form the segment of a circle with the concavity looking forwards. The shape of the chest in rachitic children becomes remarkably modified; the ribs sink in laterally, especially from about the third to the ninth, so that the transverse diameter is diminished in this situation, while the antero-posterior is correspondingly augmented, and the sternum thrown forwards. There is usually also in the same part of the chest a well-marked vertical groove running down on either side, just external to the junctions of the ribs and cartilages. The form of the upper part of the chest, however, is slightly or not at all altered; while the lower part, owing to the presence of the liver, spleen and other abdominal viscera, again expands, and its shape is pretty nearly normal. The changes which take place in the form of the pelvis are of great importance. The bones of the upper part get flattened and expanded by the pressure of the abdominal viscera upon them; but the weight of the spine above throws the sacrum forwards, and the pressure of the femora below causes an approximation of those parts of the pelvis which bound the acetabula; and the pelvic cavity consequently becomes contracted, and tends to assume a triangular form on transverse section. But the relative effects of these and other agencies are largely modified in different cases by a variety of circumstances, such as age, and the possession and use of the powers of walking, crawling, or sitting. The bones of the head and face share in the tendency to deformity. The fontanelles are slow to close, remaining open up to and beyond the second year; the head becomes large, flat on the top, elongated from before backwards, with projecting forehead, and unusual prominence of the frontal and parietal eminences. The teeth are late in appearing; and indeed if none have been cut by the age of nine months, it is a reason for at all events suspecting the presence of rickets. The teeth, moreover, are specially apt to decay and become loose. It will of course be understood that the deformities of the skeleton in rickety children are liable to innumerable variations from the types which have been enumerated; that in a large number of cases they never become serious, and are probably confined to the bones of the legs and perhaps some few others; while in some cases they assume such extreme proportions that they are not only a source of distress and misery, but are incompatible with the performance of some of the normal functions of life, or even with the maintenance of life.

After a time, which varies in different cases, the rickety condition ceases, and the bones regain their earthy elements and their strength. The bone tissue, indeed, gets unnaturally strong and dense. In some cases slight degrees of curvature slowly disappear in the progress of growth; more frequently, however, they are persistent, and there is more or less of permanent deformity.

Dr. Dickinson's observations seem to show: that a morbid process, in some respects like that in the bones, goes on in the kidneys and lymphatic glands, and especially in the liver and spleen of rickety children; and that these organs become enlarged and indurated, and the seat partly of interstitial development of fibroid tissue, partly of overgrowth of the glandular elements. The changes are transitory, and are accompanied by a deficiency of the earthy salts. They are quite distinct from amyloid or lardaceous degeneration.

Whatever the exciting cause of rickets may be, and however it acts, there can be little doubt that the morbid processes to which it gives rise in the several viscera which have been enumerated, and in the bones, have a close affinity with those of general subacute or chronic inflammation, and that the main incidents in them are an irritative overgrowth of the implicated tissues, and a modification or perversion of their normal nutrition.

Symptoms and progress.—In giving an account of the clinical phenomena of rickets, it is customary to enumerate a long series of precursory symptoms, the occurrence of which not only should excite suspicion of the impending danger, but is commonly regarded as a proof that rickets is the outcome of some more or less long-continued cachexia or dyscrasia. The constitutional origin of the malady can scarcely be denied; but it is certain that it must always have made some considerable progress before the deformity of the joint-ends of the long bones or of the chest, and the bending of limbs, become obvious, and that many, therefore, of the precursory symptoms belong properly to the earlier stages of the rickety process. Among these must probably be included catarrhal affections of the thoracic viscera, and gastro-intestinal disturbance. But by far the most important and characteristic are: first a febrile condition, manifested by restlessness at night or during the hours devoted to sleep, intolerance of the bed-clothes, which the infant continually throws off, and profuse perspirations, mainly limited to the head and upper parts of the body; and, second, general tenderness or soreness, due doubtless to general implication of the bones, and indicated by a gradually increasing unwillingness or fear to move or be moved, and a loss of enjoyment in the caresses and dancings which form so large a part of a healthy infant's life and happiness. The first distinct evidence, however, that the bones are undergoing serious change of structure is usually afforded by the enlargement of the lower extremities of the radius and ulna, and by the simultaneous or shortly subsequent enlargement of the

corresponding portions of the tibia and fibula, and of the knee and elbow joints. If the affection still proceeds, the shafts of the long bones and the spine acquire the alterations of form which have already been adverted to: the ribs fall in at the sides, the sternum protrudes anteriorly, and the pigeon-breasted condition results, in association with which the knob-like enlargement of the anterior extremities of the ribs on either side produces that characteristic appearance to which the name of the 'rickety necklace' or 'chaplet' has been applied; the pelvis gets distorted; and the bones of the head become affected, mainly, as has been pointed out, by thickening of their edges, protrusion of the parietal and frontal eminences, and unusual persistency of the fontanelles. But while all these deforming processes are going on with more or less uniformity and rapidity, the various symptoms which marked the beginning of the disease usually undergo aggravation; the fever becomes more intense, the pulse accelerated, the heat of skin augmented, the nocturnal perspirations more profuse, and the general tenderness more marked. Actual pain, indeed, often supervenes; and the fear or inability to move and the dread of being touched become much more apparent. In connection with these phenomena the appetite fails, there are thirst and irregularity of bowels, the urine is copious and contains a superabundance of phosphates, and the child undergoes rapid emaciation with disproportionate wasting and feebleness of the voluntary muscles; he assumes an anæmic or pallid, sad, anxious, wan aspect, and takes no interest in what is going on about him. During all this time the abdomen becomes relatively large, and the liver and spleen will probably be found to be distinctly hypertrophied; indeed, the enlargement of these organs is sometimes one of the earliest indications that the child is rachitic. Ascites is sometimes a consequence of the hepatic affection. The influence of rickets on dentition has already been fully considered. According to MM. Rilliet and Barthez, whose opinions are confirmed by those of Dr. H. Roger, a blowing sound is so commonly audible over the cranial sutures of rachitic children that it may almost be regarded as diagnostic of the affection.

When rickets proves fatal, it is usually either by gradual asthenia connected with the advance of the disease and impairment of the digestive functions, or by thoracic complications, such as collapse of the lung-tissue, lobular pneumonia, or bronchitis—the accession as well as the gravity of which are largely dependent on the weakness, deformity, and consequent inefficiency of the thoracic walls. Other causes of death in rickety children are laryngismus stridulus, convulsions, and chronic hydrocephalus. The duration of rickets commonly ranges between six and twelve months, at the end of which time the constitutional symptoms and those indicative of osseous inflammation subside.

Persons who have suffered from rickets in infancy not unfrequently

acquire great strength of limb (muscle and bone), but they usually remain of low stature; and the deformities which take place at the time of their malady only too commonly persist; while some (more especially those of the chest and pelvis) not unfrequently entail serious misery and danger in after-life.

In concluding this brief account of the symptoms of rickets, it is well to draw attention to the fact that the disease may be present in a slight degree—sufficient to cause manifest thickening of the wrists and ankles, and even bending of the tibiæ and thoracic walls—in children who maintain their vivacity and the aspect of almost perfect health.

Treatment.—It might be supposed, from the fact of the disappearance of phosphate of lime from the bones, that the administration of phosphate of lime is indicated in the treatment of rickets. Experience has not, however, confirmed this view, nor indeed does a correct interpretation of the pathological phenomena of the disease give it any sanction. The best medicinal remedies are tonics, such as iron and cinchona, and, above all, cod-liver oil. But these are valueless without the most careful attention to diet and hygiene. The diet should be at once nutritious, sufficiently abundant, and adapted to the age and circumstances of the child. For the young infant nothing can be more suitable than the healthy breast, or failing that, asses' milk, or else cows' milk, or preserved Swiss milk, properly diluted, and mixed, it may be, with a certain proportion of biscuit powder. At a later age a moderate quantity of well-cooked meat, comprised in a dietary which combines all the other essential elements of food, namely, sugar, starch, and fat, and composed therefore largely of milk and bread and butter, should be administered. The child should be warmly clad, should occupy an airy but sufficiently warm room, be regularly bathed and well rubbed afterwards, and taken out habitually into the open air. Change of air, and especially residence on the sea-coast, are often of essential service. But, in addition to the above measures, it may be desirable to treat the symptoms which are so apt to accompany rickets: to improve the condition of the stomach, to regulate the action of the bowels, to reduce fever, and to combat pulmonary and other complications. And especially it is of paramount importance to prevent as far as we possibly can the supervention of deformity. To this end, our measures must be regulated by the age and peculiarities of the patient. It is impossible to go into detail without becoming unsuitably diffuse. We may, however, point out that the child should lie on a soft well-made feather bed; that if it be quite young, it should not be allowed to sit up; and that if older (and the limbs give evidence of bending), it should be prevented from walking, and perhaps even from crawling. Mechanical appliances may be necessary to restrain undue locomotion.

V. MOLLITIES OSSIUM. (*Osteo-malacia.*)

Definition and causation.—This affection has in many respects a close resemblance to rickets. It occurs, however, not in children, but in adults. It has been recognised, indeed, only in women, and for the most part in women who are bearing children.

Morbid anatomy and pathology.—Mollities ossium is characterised anatomically by progressive softening of the bones, sometimes of those of the whole skeleton, occasionally of a limited number only. The minute changes appear to consist mainly in a progressive decalcification of the bony tissue, commencing from the Haversian canals and medullary spaces, and gradually involving the successive laminæ of bone which surround them, until finally decalcification is complete. A sharp line generally marks the limit of the morbid process, the bone external to it being still normal, whilst that between it and the canal or cavity is converted into a kind of fibroid tissue, in which the bone-corpuscles are scarcely distinguishable. At a later period the portions of tissue first affected soften and liquefy. Attending these changes the smaller vessels become dilated, and the contents of the various cavities red and pulpy. The enfeebled bones are liable to all those bendings and imperfect fractures which also characterise rickety bones; but, owing to the long duration of the malady and the extent to which softening takes place, the deformity which ensues is usually of the most aggravated kind and pretty universal. Trousseau maintains that mollities ossium is identical with rickets, allowance being made for difference of age and for the fact that the active processes of growth have ceased when osteo-malacia makes its appearance. The anatomical details, however, of the two processes are manifestly different. It is supposed by Rindfleisch and some others that the decalcification is due to the action of some acid (carbonic or lactic) contained in the blood. The appearances are, no doubt, much like those which might be thus caused. There is no direct evidence, however, to support this view; and indeed Virchow has ascertained the existence of a strong alkaline reaction in the gelatine yielded by fresh bones affected with this disease.

Symptoms and progress.—The symptoms of osteo-malacia, like those of rickets, are often very insidious; and the disease may first reveal its presence by the occurrence of deformity and fracture of bones. Its progress, however, is usually attended, even from the beginning, with febrile disturbance and copious perspirations, and with tenderness and pain in the course of the affected bones. The pains are, in the first instance, vague and wandering, and of a shooting character. But they gradually become more or less intense; affect not only the limbs, but the trunk, head, and face; and are greatly aggravated by move-

ment or pressure. They are often most severe in the joint-ends of long bones, and in the epiphysal ends of most bones. They may easily be mistaken at first for rheumatic or neuralgic pains. The urine contains an excess of phosphate of lime.

The progress of osteo-malacia is chronic; cases have been met with in which the disease has been prolonged for fourteen or fifteen years and upwards. Death usually takes place, however, at a much earlier date, and is generally due immediately to interference with the respiratory or circulatory functions. Recovery very rarely takes place, and never without persistent deformity. Trousseau has recorded a case or two of this kind.

Treatment.—No distinction need be drawn between the treatment of osteo-malacia and that of rickets. It was under the use of cod-liver oil that Trousseau's cases recovered.

CHAP. VIII.—DISEASES OF THE NERVOUS SYSTEM.

I. INTRODUCTORY REMARKS.

A. *Anatomy and Physiology.*

THE nervous system comprises the cerebrum and cerebellum, with the various ganglia and commissures which belong to these bodies, the medulla oblongata, the spinal cord, the sympathetic ganglia, and the nerves which, [illegible]ey m[illegible]inging from these several sources, are distributed throughout the o[illegible]ons and [illegible]. With the central organs are associated as important elemen[illegible]s which [illegible]vious membranes and other structures which serve for their su[illegible]volutions is, the[illegible]ion, and the arteries and veins which minister to their [illegible]with the localisation [illegible]ional activity.

1. *Membran*[illegible] has be[illegible] thrown [illegible]—The *cranial dura mater* is a thick, dense, inelastic [illegible]enquir[illegible]is n[illegible] W[illegible]e, which by its outer surface adheres firmly to the bo[illegible] the [illegible]he skull, by its inner surface, which is smooth and polished, constit[illegible]. Th[illegible]e parietal limit of the arachnoid cavity. It also forms certain se[illegible], [illegible]edless to specify, which intervene between the cerebrum and cerebe[illegible]em and their respective hemispheres. At the foramina for the exit of nerves at the base of the skull the dura mater becomes continuous on the one hand with the pericranium, on the other with the nerve-sheaths. The *spinal dura mater*, which, like the cranial, is dense, thick, and inelastic, is prolonged in the form of a loose bag from the margins of the foramen magnum, to which it is adherent, to the first or second sacral vertebra, where, blending with the filum terminale of the cord, it is continued onwards therewith to the lower end of the sacral canal. The spinal dura mater is separated on all sides from the bony cavity in which it lies by fat and areolar tissue. Its internal surface is smooth and polished, and presents along either side a double series of orifices of which each contiguous pair gives exit to the anterior and posterior roots of one of the spinal nerves. The dura mater is continued on each nerve as a tubular prolongation, within which the ganglion of the posterior root is contained. It then blends with the sheath of the nerve and becomes connected by fibrous processes with the margins of the intervertebral foramen.

The *arachnoid cavity* is usually regarded as a serous cavity. It occupies the interval between the dura mater, on the one hand, and the general surface of the brain and cord on the other, being perfectly continuous throughout. Its outer limit is represented by the smooth inner aspect of the dura mater; its inner limit is formed by a delicate transparent membrane which lies loosely upon the surface of the cen-

tral organs, never dipping into the sulci, and lying especially loosely upon the parts situated at the base of the brain, and upon the spinal cord. The inner and outer aspects become continuous by means of tubular prolongations wherever nerves or vessels pass from the protective organs without to the central nervous organs within.

The *pia mater* is the vascular membrane which closely invests the outer surface of the brain and cord, following all its inequalities. It is continuous by its applied surface with the connective web and vascular network which pervade the substance of the subjacent organs, and the neurilemma of the nerves appears to be derived from it. The pia mater within the skull is delicate and highly vascular, dips to the bottom of all sulci, and accurately fits the complicated arrangement of processes and depressions which exist at the base of the brain. It dips also into the great transverse fi[illegible] of the brain and into the somewhat similar fissure existing bet[illegible]tween the medulla oblongata and the cerebellum—forming in [illegible]ation a reduplication, the free margins of which are w[illegible]d, and constitute the bodies known as the chor[illegible]ia mater of the cord is much thicker, denser, a[illegible]han that of the brain, forms in front a duplicatur[illegible]h d[illegible] the bottom of the anterior furrow, and behind a thin vertical s[illegible] which occupies the posterior furrow.

The interval which exists between the pia mater and the visceral lamina of the arachnoid is known as the *subarachnoid space*; it is crossed by numerous delicate fibrous bands, and in the spinal canal on either side by the ligamentum denticulatum and behind by an incomplete vertical septum. It is the seat of the subarachnoid fluid which constitutes the great bulk of the cerebro-spinal fluid.

2. *Ventricles of brain and cord.*—The existence of the ventricles of the brain and cord (excepting the fifth) as distinct cavities is due in some sense to the failure already referred to of the pia mater at the great transverse fissure of the brain and at the posterior part of the fourth ventricle to follow the various diverticula or involutions which take their origin in these situations. The system of ventricles comprises: the lateral ventricles, which are continuous with one another and with the third ventricle in the interval, into which the velum interpositum extends, between the fornix above and the optic thalami below; the third ventricle, which communicates by means of the iter with the fourth ventricle; the fourth ventricle; and the central canal of the cord which commences above at the calamus scriptorius or posterior extremity of the fourth ventricle. The nervous boundaries of the ventricles are covered with a delicate membrane which is continuous with the neuroglia or connective web permeating the substance of the subjacent organs, is identical in structure with it, and is furnished with an epithelium. The ventricles form a continuous system, and have no communication with other cavities or spaces, excepting,

as was pointed out by Majendie, with the subarachnoid space through a small opening situated at the lower extremity of the fourth ventricle.

3. *Cerebral hemispheres.*—The cerebrum consists of two hemispheres, separated the one from the other above by the great longitudinal fissure, and united below mainly by means of the commissural fibres of the corpus callosum, by the fornix and certain other structures which need not be specified. It is composed of white and grey matter, of which the one forms a comparatively thin lamina on the surface, while the other makes up the great bulk of its mass. The surface of the organ, and with it of course the grey matter, is arranged in folds or convolutions, separated by fissures or sulci, the more important of both of which present a tolerably definite and regular arrangement. The superficial grey matter is doubtless the seat of the intellectual and emotional functions and the primary source of those various combined muscular actions which accompany and reveal their operation. The study of the convolutions is, therefore, a matter of interest, especially in connection with the localisation of function, on which subject important light has been thrown by modern pathology and recent experimental enquiries. We proceed to describe so much of the topography of the cerebral surface as bears directly on this subject.

a. Fissures.—The *fissure of Sylvius* (Fig. 3 and 10 *d*) commences on the base of the brain at the locus perforatus anticus, and, separating the middle from the anterior cerebral lobe, passes directly outwards until it reaches the lateral aspect of the hemisphere. Here it divides into two branches: an anterior short branch, which proceeds upwards and forwards, and a posterior long branch, which courses nearly horizontally backwards upon the outer surface, dividing the temporo-sphenoidal lobe below from the parietal lobe above. The *fissure of Rolando*, or *sulcus centralis* (Fig. 3 and 6 *c*), commencing above at the great longitudinal fissure a little behind the vertex, runs downwards and forwards over the outer surface of the hemisphere to near the point of bifurcation of the Sylvian fissure, separating the frontal lobe in front from the parietal lobe behind. The *inter-parietal fissure* (Fig. 3 and 6 *e*), originating in the angle contained between the fissure of Rolando and the posterior Sylvian branch, passes irregularly backwards towards the parieto-occipital fissure. The *parallel*, or *first temporo-sphenoidal fissure* (Fig. 3 *h*), running parallel to but below the posterior Sylvian branch, turns up behind its posterior extremity, and there loses itself in a group of convolutions which are limited above and behind by the posterior part of the inter-parietal fissure, and are known by the name of the gyrus angularis, or pli courbe. On the inner aspect of each hemisphere there are four fissures which call for special notice:—the first is the *fronto-parietal*, or *calloso-marginal* (Fig. 4 *f*), which, commencing in front, runs backwards parallel with the corpus callosum, forming the upper limit of the gyrus

fornicatus, until having arrived near the posterior edge of the corpus callosum, it turns up to reach the upper margin of the hemisphere a little behind the upper termination of the fissure of Rolando; the

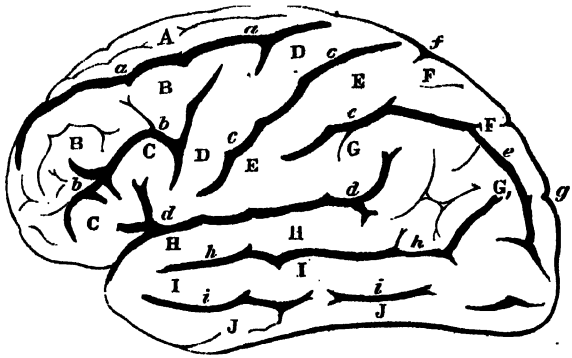


FIG. 3. Lateral view of brain, showing principal convolutions and fissures.

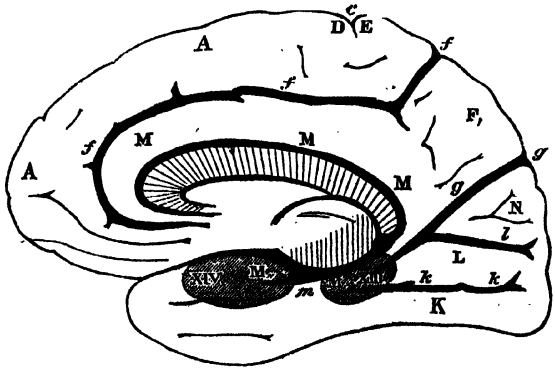


FIG. 4. Inner surface of hemisphere, showing principal convolutions and fissures, and Ferrier's centres of touch (XIII.), and of smell and taste (XIV.).

The several letters in the above figures, and also in figures 5, 6, 8, 9, and 10, refer to the same parts.

Fissures:—*a*, superior frontal; *b*, inferior frontal; *c*, fissure of Rolando; *d*, fissure of Sylvius; *e*, inter-parietal; *f*, fronto-parietal; *g*, parieto-occipital; *h*, first temporo-sphenoidal; *i*, second ditto; *j*, third or inferior ditto; *k*, occipito-temporal; *l*, calcarine; *m*, hippocampal.

Convolutions:—A, superior, or first frontal; B, second ditto; C, third ditto; D, ascending frontal, or anterior parietal; E, ascending parietal; F, superior parietal; F_1, præcuneus; G, supra-marginal; G_1, gyrus angularis, or pli courbe; H, first tempero-sphenoidal; I, second ditto; J, third ditto; K, fusiform lobule; L, lingual lobule; M, gyrus fornicatus; M_1, gyrus hippocampi; M_2, uncus gyri fornicati, or subiculum cornu Ammonis; N, cuneus.

second is the *vertical* or *parieto-occipital* (Fig. 4 *g*), which separates the occipital from the parietal lobe, and, commencing above on a level with the posterior extremity of the parallel sulcus, runs downwards and

forwards to unite at an acute angle with (third) the *calcarine fissure* (Fig. 4 *l*), which is nearly horizontal in position and corresponds to the hippocampus minor; the last is the *hippocampal fissure* (Fig. 4 *m*), which runs round the crus cerebri, and indicates the course of the hippocampus major.

b. Convolutions.—In front of the fissure of Rolando, and following its course from below upwards, runs the *ascending frontal*, or *anterior parietal convolution* (Fig. 3 and 6 D), and from the anterior aspect of this are given off in succession from above downwards the *first, second,* and *third frontal convolutions* (Fig. 3 and 6 A B C). The first of these runs parallel to the longitudinal fissure, and forms, indeed, the marginal convolution (Fig. 4 A) of that fissure; the second follows the same course as the first, but lies external to it; and the third, still more external, by its posterior part forms the upper and anterior boundary of the anterior branch of the fissure of Sylvius, and by its anterior part separates the second convolution above from the external orbital convolution below. The third frontal convolution of the left side is also called *Broca's convolution.* The *orbital convolutions* occupy that portion of the under surface of the anterior cerebral lobe which lies upon the floor of the skull. Parallel to the ascending frontal convolution, and separated from it by the fissure of Rolando, courses the *ascending parietal convolution* (Fig. 3 and 6 E E), from the posterior and outer margin of which two secondary convolutions, separated from one another by the inter-parietal sulcus, pass nearly directly backwards: the inner and upper one, the *superior parietal convolution* (Fig. 3 and 6 F), forming the margin of the longitudinal fissure in this situation, and ending behind at the parieto-occipital fissure; the outer and lower one, or *gyrus supra-marginalis* (Fig. 3 and 6 G), lying, at its anterior extremity, mainly between the inter-parietal sulcus and the posterior branch of the fissure of Sylvius, and further back between the inter-parietal sulcus and the posterior extremities of the Sylvian and first temporo-sphenoidal fissures. In the latter part of its course it is considerably curved, and receives the name of *gyrus angularis*, or *pli courbe* (Fig. 3 and 6 G). There are three *temporo-sphenoidal convolutions* passing nearly horizontally backwards from the anterior part of the temporo-sphenoidal lobe; the first (Fig. 3 H) is situated between the posterior branch of the Sylvian fissure above and the first temporo-sphenoidal fissure below; the second (Fig. 3 I) lies below the first temporo-sphenoidal fissure; the third is lower down, but parallel to the others. At the bottom of the fissure of Sylvius, at its point of bifurcation, and concealed by the overlying convolutions, lies the *island of Reil*, the grey matter of which is in close anatomical relation in front with that of the posterior part of the third frontal convolution, behind with that of the first temporal. On the internal aspect of the hemisphere, amongst other convolutions, may be observed the *gyrus fornicatus* (Fig. 8 M), which, com-

mencing in front beneath the genu of the corpus callosum, runs backwards over this body between it and the calloso-marginal fissure, then turns round its posterior extremity, being continued downwards and forwards under the name of the *gyrus hippocampi*, or *uncinate convolution* (Fig. 8 and 10 M), first between the hippocampal and calcarine fissures, and thence nearly horizontally forwards until it reaches the internal extremity of the fissure of Sylvius, where it forms the *uncus gyri fornicati*, or *subiculum cornu Ammonis* (Fig. 8 and 10 M). For an account of the remaining convolutions, to some of which we may subsequently allude incidentally, we must refer to anatomical works.

In connection with the subject of the convolutions it may be observed that M. Betz has recently shown that the surface of the cerebrum may be divided by microscopic peculiarities into two regions, of which the anterior, limited by and including the ascending parietal convolution, is characterised by containing, in greater or less abundance, giant-cells resembling those of the anterior cornua of the spinal cord, the posterior by an almost total absence of such cells.

4. *Ganglia at base of brain.*—Excepting the commissural fibres of the corpus callosum and fornix, and certain other commissures, which need not be enumerated, all the nerve-fibres from the grey matter of the convolutions converge to the group of ganglia situated at the base of the brain, namely, the corpora striata and the optic thalami, together with the corpora geniculata and corpora quadrigemina, and are thence continued (directly or indirectly) either through the superior cerebellar peduncles to the cerebellum or along the crura cerebri to the medulla oblongata.

Each *striated body* comprises three nuclei, separated from one another by white fibres. The first of these is the *caudate nucleus*, and is that portion of the body which is visible in the lateral ventricle. The second is the *lenticular nucleus*, which is placed in part external to and below the caudate nucleus, in part external to and below the optic thalamus: being separated from these bodies by a layer of white fibres, which forms the internal capsule, and on the outer side from the grey matter of the island of Reil by a similar white lamina, which is known as the external capsule. Imbedded in this last is the third or *tæniæform nucleus* or *claustrum*, which forms an exceedingly thin plate. The cerebral fibres which enter these nuclei and occupy the intervals between them come from all parts of the cerebral surface, but mainly from the anterior half; and those which emerge from them below pass mainly downwards and backwards to form the under portion or *crust* of the corresponding cerebral peduncle within which they become connected with an additional ganglion of the same system, namely, the *locus niger*. The further destination of the crust is twofold; it sinks into the anterior and upper edge of the pons, and there divides into two portions; of which one, according to Meynert, crosses among the

anterior fibres of the pons, and passes with these to the opposite half of the cerebellum, thus decussating with its fellow; while the other emerges from the posterior border of the pons as the anterior pyramid, which also decussates with its fellow, and is prolonged mainly to form the lateral column of the opposite side of the cord.

The *optic thalami*, *corpora geniculata* and *corpora quadrigemina* also derive fibres from nearly all parts of the cerebral surface, though mainly probably from the posterior and lateral portions; and, resting by their under surface upon the cerebral peduncles, are more or less directly continuous with their upper half or the *tegmentum*. This, which includes within it the *red nucleus*, divides like the crust into two portions. One of these continues backwards as the *processus e cerebello ad testes* and *valve of Vieussens* to form the superior peduncles of the cerebellum; and the fibres which constitute it for the most part decussate anteriorly to the posterior limit of the testes, and so reach the opposite sides of the cerebellum. The other continues downwards, in the substance of the pons and on the floor of the fourth ventricle, to become continuous mainly with the sensory tracts of the medulla oblongata and cord.

5. *Cerebellum and its peduncles.*—So little is known comparatively of the specific functions of different parts of the cerebellum that it is needless to consider here either its general form and arrangement or the names which have been given to its separate lobes and lobules. It may, however, be pointed out that, in addition to its superficial grey investment, it contains imbedded in its white medulla in the first place two ganglia (one on either side), the *corpora dentata*, and in the next place two other grey nuclei, the *roof nuclei* of Stilling, which lie below the central lobule of the superior vermiform process.

The cerebellum presents three pairs of peduncles or groups of white fibres, of which one comes from the cerebrum, one from the medulla oblongata, and the other is mainly transversely commissural. The first pair or the *superior peduncles* come almost exclusively from the tegmentum of the cerebral peduncles, comprise the *processus e cerebello ad testes* with the intermediate valve of Vieussens, and pass into the corpora dentata and thence to the convolutions. The second pair or the *middle peduncles* are constituted mainly by the transverse fibres which form the great bulk of the pons Varolii, but comprise the cerebellar fibres derived from the crust of the cerebral peduncles; of these the more internal pass into the roof nuclei, but the outermost, accompanied by the restiform bodies, reach the surface of the cerebellum without the intermediation of either of these ganglia. The third pair or the *inferior peduncles* are the restiform bodies.

It will thus be seen that the most direct, if not the only, communication between the hemispheres of the cerebrum and those of the cerebellum is effected by means of fibres which, taking their origin in the cerebral ganglia, pass backwards and lose themselves probably in the ganglia imbedded in the white substance of the cerebellum; that

of these some are derived from the crust, some from the tegmentum of the crura cerebri, and that all, according to Meynert, decussate in the course of their passage. It will also be gathered that both the cerebrum and the cerebellum send down strands of fibres to take part in the formation of the medulla oblongata. Those from the brain are continued from both layers of the crura cerebri; those from the cerebellum are the restiform bodies.

6. *Spinal cord.*—Before speaking further of the medulla oblongata it will be well to describe the spinal cord. This, which extends, in the adult, from the foramen magnum above to the lower part of the first lumbar vertebra below, presents an anterior and a posterior median fissure, and, on either side, two lateral furrows, which correspond to the successive points of emergence of the anterior and posterior roots. It is thus divided superficially, on each side, into posterior, lateral, and anterior columns. But, in addition to these, a slender median column, most obvious in the upper part of the cord, may be observed running along the edge of the posterior median fissure. On transverse section the grey matter of the cord will be found to occupy its central part, the white its periphery. The grey matter is arranged in the form of two lateral crescents, placed back to back, and united with one another in the middle by a transverse commissure, which crosses the narrow interval between the bottoms of the anterior and posterior fissures, and contains within it the ventricle of the cord. The posterior limb of each crescent constitutes the posterior horn of grey matter, the anterior limb the anterior horn. In the latter are situated distinct groups of large multipolar cells, which appear to be the nuclei of origin of the anterior or motor nerves, and from it the root of each nerve passes forwards through the substance of the white matter in several parallel bands. The posterior horn is tipped by the gelatinous substance of Rolando, from the whole transverse extent of which the fibres of each posterior root escape in wavy bands, some undulating through the substance of the adjoining posterior column previous to their appearance at the surface of the cord. At the root of the posterior cornu, on its outer side, is the group of cells which indicates the longitudinal tract to which Lockhart Clarke has given the name of *tractus intermedio-lateralis*; and in almost the corresponding situation, on its inner side, may be seen the sectional surface of the tract of cells which constitutes Clarke's *posterior vesicular column*. The grey matter varies in bulk in different parts of the cord, and is especially abundant in the cervical and lumbar enlargements, but the superficial white matter increases absolutely in quantity from below upwards.

7. *Medulla oblongata.*—At the upper part of the cord, where it merges in the medulla oblongata, considerable changes are presented in the distribution of its parts. These changes become more and more remarkable as we proceed from the lower to the upper part of the medulla oblongata, and are complicated by the appearance of additional

grey nuclei. The posterior fissure opens out and blends with the ventricle of the cord; the posterior pyramids are divaricated, forming between them the calamus scriptorius; and the remainder of the posterior columns, now constituting the restiform bodies, passes upwards and outwards to form the inferior peduncles of the cerebellum; in front of these is gradually developed, on either side, a grey column, due to the altered position of the gelatinous substance of Rolando; still further forwards we see the seeming blending of each lateral column with its olivary body, and in front the anterior columns, apparently continued upwards into the anterior pyramids. The arrangement of parts here is exceedingly complicated. But it may be stated generally: that the bulk of each posterior column of the cord passes upwards in the restiform body to the cerebellum; and that, according to Meynert, it has in its passage upwards a direct connection with the olivary body, and that in this region decussation of the tracts of opposite sides takes place, so that the relation between the cord and cerebellum becomes crossed; that the greater part of the white substance of each antero-lateral column decussates with the corresponding part of the opposite side at the lower extremity of the anterior pyramid; and that each pyramid is hence constituted mainly by the continuation upwards of the medullary matter of the opposite side of the cord to that on which it is itself situated, and then, passing through the substance of the pons Varolii, forms in front of it the larger bulk of the crust of the corresponding cerebral peduncle; and, lastly, that some portion of the fibres of the lateral columns, and most of the opened-out grey matter of the cord, pass upwards along the floor of the fourth ventricle and back of the pons Varolii, partly to form the tegmentum, partly to become associated with the grey matter of the iter and third ventricle.

8. *Cerebro-spinal nerves.*—The cerebro-spinal nerves, with only two exceptions, originate in the grey matter of the spinal cord, or its continuations in the medulla oblongata, along the floor of the fourth ventricle, and around the iter. They are of two kinds, motor and sensory. The motor spinal nerves have their immediate origin in the groups of large cells contained in the anterior cornua, and emerge at the surface of the cord in the furrow separating the anterior from the lateral columns; the sensory nerves originate apparently in the posterior cornua, and make their appearance superficially in the groove which divides the lateral from the posterior columns.

The cerebral nerves, in the main, arise according to their properties in the upward continuations of the motor and sensory tracts of the grey matter of the cord; in other words, the motor nerves spring from the upward continuation of that portion of grey matter which is anterior to the spinal ventricle, the sensory nerves from the upward continuation of that portion which is behind it. But these tracts, as has been shown, become modified in their relative positions in the medulla oblongata

and floor of the fourth ventricle; the motor tract gets superficial on either side of the median line in the course of the fasciculi teretes; the sensory tract, on the other hand, split into two halves, continues upwards on either side of the motor tract, occupying each lateral half of the floor of the ventricle, spreading out on either side along the inner aspect of the cerebellar peduncles towards the cerebellum, and at the anterior point of the fourth ventricle rising up and coalescing again, as in the cord, over the iter or tubular continuation of the ventricle.

The motor nerves, in their order from behind forwards, are the spinal accessory and hypoglossal, the portio dura, the abducens or sixth, the motor branch of the fifth, the fourth, and the third. The upper part of the spinal accessory arises from a nucleus situated in the lower part of the medulla oblongata, a little outside the central canal, and concealed by the posterior pyramid; and it becomes superficial as the lowermost member of the eighth pair at the lateral aspect of the medulla below the level of the olivary body. The nucleus of the ninth, or hypoglossal nerve, commences below in front of the spinal canal, in contact with the spinal accessory nucleus, and extends for a short distance along the floor of the fourth ventricle in the neighbourhood of the calamus scriptorius. Its superficial origin is between the olivary body and the anterior pyramid. The common nucleus of the portio dura of the seventh pair and abducens is situated just in front of the hypoglossal nucleus. The former nerve becomes superficial at the posterior margin of the pons, between the middle and inferior peduncles of the cerebellum; the latter in the groove between the anterior pyramid and the pons. The nucleus of the motor-root of the fifth pair is situated within the fasciculus teres, a little above, in front of, and external to that of the portio dura; the nerve becomes superficial by penetrating the lateral portion of the pons. The third and fourth pairs arise in common from a pair of nuclei, situated side by side in the floor of the iter. The fourth nerves encircle the iter in their course, and then winding round the outer side of the crura cerebri reach the base of the brain; each third nerve penetrates the subjacent locus niger, and makes its appearance on the inner side of the crus.

The sensory nerves, in their order from behind forwards, are the vagus and glosso-pharyngeal, the auditory, and the sensory portion of the fifth; to which may be added the optic and the olfactory. The nucleus of the vagus, connected with that of the spinal accessory below, appears on the floor of the fourth ventricle just above the calamus and external to the hypoglossal nucleus. Above, it appears to sink beneath the auditory nucleus. The glosso-pharyngeal nucleus, which is partly continuous with that of the par vagum, but higher up, is wholly concealed by the auditory nucleus, with which it is in some measure blended. These two nerves become superficial along the posterior border of the olive. The auditory nucleus is of large size; it involves the upward

continuation of the grey matter of Rolando, and, in part, the posterior pyramid and restiform body. It occupies the floor of the ventricle external to the fasciculus teres, and its outer part turns backwards with the restiform body to reach the cerebellum, some portion of it becoming connected with the dentate nucleus, some stretching across the roof of the ventricle to join its fellow. The nerve-fibres arising from this nucleus, taking various routes, combine to form the portio mollis, which has its superficial origin at the posterior margin of the pons, between the superior and middle cerebellar peduncles. The nucleus of the sensory portion of the fifth is, like the auditory, largely developed out of the upward continuation of the grey tubercle of Rolando, and also from that of the root of the posterior horn. It is situated in advance of the nucleus of the portio mollis, with which indeed it is, to some extent, connected behind, and extending upwards to the fossa, where the fillet meets the anterior fibres of the pons, arches backwards with the rest of the continuation of the grey matter from the cord towards the side and roof of the anterior part of the fourth ventricle and of the adjoining part of the iter. The superficial origin of the nerve is to the anterior and outer part of the pons Varolii. The optic nerves interlace in the chiasma, and thence each optic tract winds round the corresponding crus cerebri to reach the posterior portion of the optic thalamus, the corpora geniculata and the corpora quadrigemina of the corresponding side, which therefore may be regarded as its nuclei, or at all events as containing its nuclei; but, further, the optic tract in its whole extent is intimately connected structurally with the crus cerebri, and the chiasma with the grey matter lining the third ventricle. The olfactory nerve is really, as comparative anatomy has long shown, a lobe of nervous substance. It is formed of grey and white matter, and contains, according to Meynert, a central ventricle continuous with those of the cerebrum, which, however, according to Struthers, is absent in the adult; its so-called roots are connected respectively with the anterior and posterior extremities of the gyrus fornicatus, and some of the white fibres connected with it have been traced into the anterior commissure. It is an important fact that the fibres of the anterior commissure are connected with the occipital and temporo-sphenoidal lobes only, and that, hence the olfactory nerves, and it may be added, from their connection with the optic thalami and associated ganglia, the optic nerves, are both intimately connected with that portion of the brain with which, through the intermediation of the same ganglia, the rest of the sensory nerves are connected.

9. *Resumé of the relations of the different parts of the central nervous system.*—The anterior portion of the surface of the brain (all that in front of the fissures of Rolando, together with the ascending parietal convolutions behind those fissures, and certain other convolutions connected therewith) appears on sufficiently good grounds to be regarded

as the supreme organ of the cerebro-motor processes or impulses; and, indeed, as will presently be pointed out, pathological and experimental investigations have demonstrated that certain definite regions of this area are connected with certain special groups of combined movements. From all this extent of surface radiating fibres converge to certain parts at the base of the brain, namely, the caudate and lenticular nuclei of the corpora striata and the white matter (the internal capsules) which lies between these bodies and the optic thalami. Of these radiating fibres some pass without interruption through the internal capsules, while others enter the nuclei of the corpora striata. Below these nuclei, the fibres passing uninterruptedly through the internal capsules, together with others given off from the under surface of the corpora striata, form the crustæ of the crura cerebri, which, continued downwards through the pons Varolii, emerge from its posterior and lower border in the form of the anterior pyramids of the medulla oblongata. Hitherto the fibres derived from each cerebral hemisphere have travelled downwards and backwards on the corresponding side of the body; at the lower part of the anterior pyramids, however, decussation takes place, and the fibres of the anterior pyramid of one side are continued downwards, mainly along the anterior and lateral white columns of the opposite side of the cord. But, in addition to the corpora striata, with which bodies all the fibres passing from the cerebro-motor region of the brain have, in their passage downwards, a more or less intimate connection, there are, imbedded as it were in each lateral motor tract, a series of subordinate motor centres or nuclei, succeeding one another in close succession from the floor of the iter above to the termination of the cord below, each one of which gives origin to a motor nerve or to a certain number of fibres going to the constitution of a motor nerve.

It follows generally from the above account that complex motor impulses, originating in the hemispheres of the brain, are conveyed along the radiating fibres to the corpora striata, through the agency of which bodies, resolved as it were into their simplest elements, they are transmitted to the several subordinate cerebral and spinal nuclei which immediately govern the movements of those muscles, which in combination effect intended results. It follows generally also that impulses originating in one cerebral hemisphere act through the corpus striatum of the same side upon the spinal nuclei of the opposite side of the body, and hence upon the muscles of the opposite side of the body. It must be added that the same holds good of those motor nerves whose origins are situated above the decussation of the pyramids. There are, however, certain exceptions to these statements, due doubtless to the fact of the intimate connection by means of commissural fibres between the two hemispheres of the brain, and especially to the similar connection which subsists between the corresponding motor nuclei of opposite sides along the motor tracts. These exceptions are presented especially by the motor nerves of the eyes, and by the nerves concerned

in phonation, respiration, and other acts in which the corresponding muscles of opposite sides of the body habitually act in unison or concert. Further, it must not be forgotten that every subordinate motor centre has independent motor powers, which, if it retain its connection with its correlated afferent centre, are capable of being brought into action by reflex stimulation: that, if the cerebrum be removed, or its functions in abeyance, combined movements, to all appearance voluntary, may be effected through the immediate agency of the corpus striatum; that if the spinal centres be cut off from their connection with the higher centres, these also are capable of inducing reflex movements; and that under various conditions of health and disease the independent action of these various subordinate centres is a fact of more or less importance.

The afferent or sensory nerves, which near their entrance into the spinal marrow are furnished with ganglia, penetrate into the posterior cornua, and thus become connected with that portion of grey matter lying behind the central canal which constitutes the sensory region of the spinal cord. This sensory region occupies the whole length of the cord, and at the medulla oblongata becomes split longitudinally from before backwards, both halves passing upwards, one on either side of the now superficial motor nuclei of the medulla oblongata, to form the tegmenta and to become connected with the optic thalami, corpora geniculata, and corpora quadrigemina, and thus with the nuclei of origin of the optic nerves. From these ganglia radiating fibres proceed mainly to the grey cortex of the posterior portions of the cerebrum or to the true sensorium. Thus it appears that the posterior part of the cerebral surface has some such relation with the sensory functions as the anterior has with the motor functions, and the optic thalami and ganglia behind them some such connection with the same system as the corpora striata have with the motorial. And further, it seems probable (judging at all events by the analogies afforded by the organs of seeing and hearing) that complicated external impressions become analysed or disentangled, as it were, or reduced to their simplest elements by the organs which first receive them; to become again blended into a whole, so to speak, in their onward progress to the sensorium. Both experiment and pathology have shown conclusively that the sensory tracts decussate equally with the motor; and that the cerebral hemisphere and optic thalamus of one side are in direct relation with the sensory tract and nerves of the opposite half of the medulla oblongata and spinal cord. The decussation does not, however, take place in the pyramids or at any one spot; but each sensory nerve immediately after its entry into the grey matter of the cord decussates with its fellow of the opposite side, and its fibres of communication with the optic thalamus continue thenceforward to pass upwards on the same side as that body.

The relations of the cerebellum with the motor and sensory tracts as they traverse the base of the encephalon, and which are such that

(contrary to what obtains in the cerebrum) each lateral lobe is functionally connected with its own side of the body, have already been considered, and its connection with the posterior columns of the cord through the intervention of the restiform bodies has also been pointed out. It is further established that the posterior columns of the cord are in no sense the conductors of ordinary sensory impressions, as from their position was formerly supposed, but that whether afferent or merely commissural they are mainly subservient to the co-ordinating functions.

It is important to bear in mind: that at the base of the brain, and especially in the situation of the pons Varolii and medulla oblongata, the sensory and motor tracts of both sides become to some extent intermingled, that the nuclei of origin of many nerves of the highest interest and importance are crowded together into a very small space, and that hence disease affecting these parts is liable to be attended with complex, aggravated, and it may be added striking features; and that as regards the cord the sensory tracts although probably in part occupying the lateral white columns are mainly imbedded in its interior, while nearly all the white matter which forms its peripheral portion as well as the anterior cornua belong to the motor system, and that hence the sensory columns are specially protected from the influence of pressure or other injurious influences operating from without.

10. *Localisation of function.*—*a.* Pathological observation and recent experimental researches have combined to prove: that certain definite areæ of the grey surface of the cerebral hemispheres are the seats of special endowments; and that their stimulation is attended with certain specific consequences for the most part revealed by definite groups of movements, their destruction by equally specific consequences of an opposite or paralytic kind. It need scarcely be said that experimental investigation has been conducted almost exclusively on the lower animals, and that hence the determination of the areæ above referred to in relation to the human brain can only be regarded as approximative. The positions assigned to these areæ or centres by Dr. Ferrier are shown in Figs. 5, 6 and 4.[1]

It will there be seen: that the centre (v.) for movements of the lips and tongue occupies the posterior part of the third frontal and the lower part of the ascending frontal convolutions; and that in immediate relation with this are the centres, (vi.) for the depression of the angle of the mouth, (vii.) for its elevation—both seated in the ascending frontal—and (ix.) for its retraction with contraction of the platysma—occupying the lower part of the ascending parietal. At the upper part of the ascending frontal, encroaching however on the neighbouring ascending parietal and on the first frontal, is situated the centre (iii.)

[1] For much of what follows see Dr. Ferrier on the 'Functions of the Brain,' 1876.

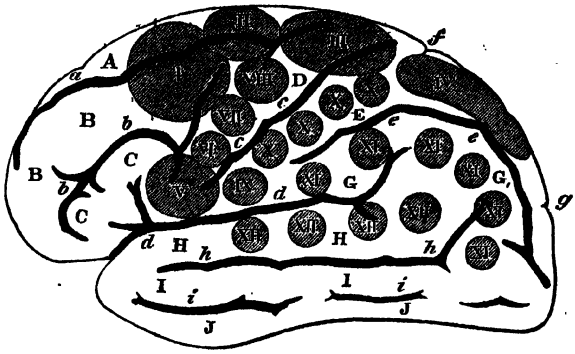


FIG. 5. Lateral view of brain, showing Ferrier's centres of movements.

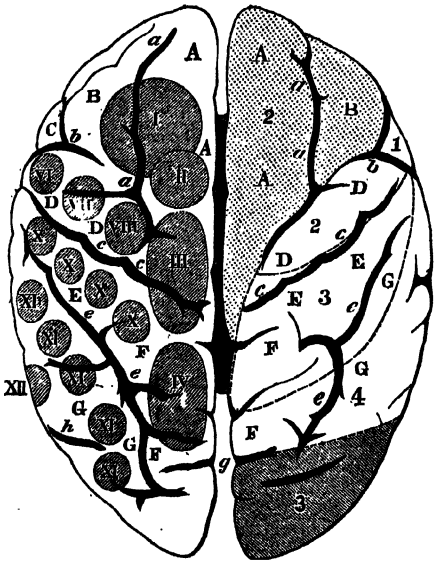


FIG. 6. Upper aspect of brain, showing principal convolutions and fissures; and on the left side Ferrier's centres of movements, and on the right the arterial area.

The Roman numbers in the above figures, and in Fig. 4, refer to Ferrier's centres.

i., lateral movements of head and eyes, with elevation of eyelids and dilatation of pupils; ii., extension of arm and hand; iii., complex movements of arm and leg, as in climbing, swimming, &c.; iv., movements of leg and foot, as in locomotion; v., movements of lips and tongue, as in articulation; vi., depression of angle of mouth; vii., elevation of angle of mouth; viii., supination of hand and flexion of forearm; ix., centre of platysma—retraction of angle of mouth; x., movements of hand and wrist; xi., centre of vision; xii., centre of hearing; xiii., centre of touch; xiv., centre of smell and taste.

for complex movements of the arm and leg, as in climbing, swimming, and the like; immediately in front upon the first frontal the centre (ii.) for extension of the hand and arm; and just behind it, and occupying partly the upper extremity of the ascending parietal and partly the superior parietal the centre (iv.) for movements of the leg and foot, as in locomotion. The centres marked (x.), occupying the greater part of the ascending parietal, are connected with movements of the hand and wrist; and that marked (i.), seated in the first and second frontal convolutions, is correlated with lateral movements of the head and eyes to the opposite side, elevation of eyelids, dilatation of pupils, and generally the look of surprise. It will be observed that all the above centres are included in that area of the surface of the brain which is in special relation through the corpus striatum with the motor tract, and which, according to M. Betz, contains giant-cells resembling those of the anterior cornua of the cord.

Below and behind the interparietal and Sylvian fissures is a series of centres which, though associated like the others with more or less definite movements, are really sensory centres; the movements due to their stimulation being excited reflectorially through the motor centres, and their destruction being unattended with loss of muscular power. The first of these (represented by a group of circles numbered xi.), which occupies the whole extent of the supra-marginal convolution and pli courbe, is the centre of vision. Its destruction causes temporary blindness of the opposite eye; the destruction of both causes permanent and absolute blindness of both eyes. Its irritation appears to evoke subjective visual phenomena in the opposite eye with turning of the eyeballs, and frequently of the head, towards that side, and contraction of the pupils. The second of these (xii.) corresponds to nearly the whole of the first temporo-sphenoidal convolution. It has a similar relation to hearing that the last has to seeing. Destruction of this part involves absolute loss of hearing on the opposite side; irritation causes sudden pricking of the opposite ear, and turning of the head and eyes in the same direction, with opening of the eyes and dilatation of the pupils. The third (Fig. 4, xiii.), situated in the hippocampal region, appears to be the centre for tactile sensation. Its destruction is attended with hemianæsthesia; its irritation with movements indicative of pain or uneasiness in the opposite side of the body. The last (Fig. 4 xiv.) is the centre of smell; intimately associated with which, though as yet impossible of exact localisation, is the centre of taste. Irritation of the centre of smell induces torsion of the upper lip and partial closure of the nostril on the same side as the centre; its destruction abolishes the sense of smell in the same nostril. Irritation of the part of the surface of the brain concerned in taste provokes movements of the lips, tongue, and cheeks; its destruction involves the abolition of the gustatory sense in the opposite side of the mouth. It is important to note that these sensory centres occupy

that part of the brain which is in special relation with the sensory tract, and which, as M. Betz shows, presents an almost total absence of giant-cells.

There are certain parts of the cerebral surface the effects of irritation of which are negative. These are especially, the internal aspect of each hemisphere including the gyrus fornicatus, the island of Reil, the occipital lobe, and the anterior parts of the frontal lobe including that part which overlies the orbit. But nevertheless Dr. Ferrier adduces plausible arguments: for believing that the occipital lobe has some definite relation to visceral sensation, and that its destruction is attended with abolition of appetite for food associated with depression and apathy, and, in general, speedy death; and for regarding the anterior part of the frontal lobe as being specially connected with the intellectual functions, its destruction being attended with apathy, dulness, disposition to sleep, and loss of the faculty of attentive and intelligent observation.

It should be added to the above summary that Drs. Dupuy and Burdon Sanderson have shown that the specific motor powers above considered (so far as they can be tested experimentally) do not reside absolutely in the grey matter of the convolutions; but that in most cases similar motor effects may be produced by exciting, after successive removals, each successive surface of that wedge of brain-substance of which the base corresponds to the particular superficial motor area, and the apex to a point in the corpus striatum. Dr. Sanderson, indeed, says that the movements are produced most distinctly when the irritation is effected directly upon the corpus striatum.

Finally, in reference to the surface of the brain, it must be pointed out that the posterior extremity of the third frontal convolution of the left side, or Broca's convolution, is, judging from pathological evidence, the centre of the faculty of articulate language. With this conclusion Ferrier's experimental results are reasonably accordant.

b. As might naturally be supposed, from its relations to the hemisphere above and to the medulla oblongata and cord below, destruction of the corpus striatum is attended with paralysis of voluntary motion of the opposite side of the body, excepting in so far as this is obviated by the intimate connections subsisting between those collateral spinal nuclei supplying opposite muscles which habitually act in unison. Irritation of this ganglion causes spasmodic contraction of the muscles on the opposite side of the body.

c. It seems clear that the optic thalamus has the same relation to sensation, including tactile sensation, sight, hearing, and taste (but probably not to smell) that the corpus striatum has to motion. Irritation of this body is unattended with motor manifestations, but its destruction involves hemianæsthesia of the opposite side of the body, including loss or impairment of taste and hearing, impairment of smell, due to anæsthesia in the domain of the fifth nerve, and blind-

ness. Dr. Hughlings Jackson records a case of destruction of the optic thalamus by disease, in which together with the phenomena above enumerated there was loss of sight in the half of each retina on the same side as the lesion. It has been maintained by many physiologists that the optic thalamus has not the connection with sensation here assigned to it, but that the posterior part of the internal capsule is the direct channel for the transmission of peripheral sensory impressions to the surface of the brain. There is no doubt that this is so far true that destruction of this part causes, like destruction of the thalamus, opposite hemianæsthesia; but this is due to the fact that the internal capsule is the medium of communication between the brain and thalamus.

d. Whatever other functions may belong to the cerebellum, at any rate this portion of the encephalon appears beyond all doubt to be the supreme centre for the regulation of 'the various muscular adjustments necessary to maintain the equilibrium of the body.' But the maintenance of equilibrium demands, not only a central organ, but a sensory or afferent mechanism by which the central organ may be kept informed of the condition of the body in relation to equilibrium, and an efferent or motor mechanism by means of which muscular adjustment may be effected. The former of these are the organs of common sensation, the eyes, and more important than all the semicircular canals with their afferent nerves; the latter are the motor nerves and voluntary muscles.

Experimental lesions of the cerebellum always induce disorders of equilibrium, but never impairment of sensation, or actual loss of voluntary muscular power. Without entering into any physiological explanation of the phenomena, we may briefly state that experimental evidence proves that destructive lesions of the anterior part of the middle lobe cause a tendency to fall forwards, of the posterior part of the middle lobe a tendency to fall backwards, of the right lobe a tendency to turn to the left (in the case of one of the lower animals, therefore, or of a person lying down, to roll from left to right, or towards the injured side), and of the left lobe a tendency to turn to the right; and that precisely converse tendencies result from irritation of the same parts. Attending these movements there is spasmodic contraction of the muscles of that side of the body towards which twisting occurs; and when the lateral lobes are affected the twist commences with spasmodic torsion of the head and neck. Usually also there is conjugate deviation of the eyes to the right, left, upwards or downwards, in accordance with the direction of the general bodily movements, or more or less nystagmus. Lesions of the structures connected with the cerebellum are also attended with disturbance of equilibrium: division or destruction of the middle peduncle of the cerebellum on either side causing the same symptoms as destructive lesions of the corresponding cerebellar lobe; and

irritation and injury of the corpora quadrigemina equally inducing incoordination of movement. It must be added that affections of the semi-circular canals produce the same consequences as regards equilibrium as affections of the cerebellum: affections of the superior vertical canals being equivalent to affections of the anterior part of the middle lobe; affections of the posterior vertical canals to those of the posterior part of the middle lobe; and affections of the horizontal canals to those of the corresponding lateral lobes.

e. The corpora quadrigemina are through the corpora geniculata brought into immediate relation with the optic tracts, and, indeed, there is no doubt that these bodies are the subordinate centres of vision, and have reflex connections with the motor nerves of the eyes. In the lower animals destruction of one optic lobe causes blindness of the opposite eye and more or less immobility of the pupil; irritation induces sudden starting backwards of the animal as if in alarm, turning of the eyes and head to the opposite side, dilatation of the pupils, and more or less spasmodic contraction of the facial muscles with trismus and opisthotonos. It is important to note that the pupils are completely paralysed only when the destruction of these bodies is bilateral; and that when irritation causes dilatation of the opposite pupil, dilatation of the pupil of the sound side speedily follows. In man destruction of one of the anterior tubercles of the corpora quadrigemina appears to cause hemianæsthesia in both eyes on the same side as the lesion. There are good reasons for believing that the testes are particularly connected with some forms of emotional expression. We have already referred to the relation of the corpora quadrigemina to equilibrium.

f. From the anatomical facts which have been detailed it is obvious that the medulla oblongata, including all that region from which the cerebral nerves arise, is the chief centre of many important functions which are more or less essential to the maintenance of life. It is clearly established, indeed, that even in warm-blooded animals all the centres above the medulla oblongata may be removed without destroying life; and that with the medulla oblongata remaining respiration and deglutition are still capable of performance. The medulla is in fact the coordinating centre of the respiratory acts, of phonation, of articulation, of facial expression, and of the acts of sucking and deglutition. Moreover, it is the centre of inhibition and acceleration of the action of the heart, and the centre of innervation of the blood-vessels. The coordinating centre of respiration is placed by Flourens in the angle of the calamus scriptorius.

g. As regards the cord all that we need add to statements already made is: that like the medulla it is a centre, though subordinate centre, of reflex action; that cut off from its connection with the parts above it is still capable through its afferent and efferent connections of producing coordinated movements; that under such cir-

cumstances irritation of the ends of sensory nerves generally causes reflex movements of the part with which the irritated nerves are in relation; that if the irritation be extreme or the cord unnaturally irritable the influence instead of remaining limited becomes diffused horizontally and perpendicularly throughout the cord, so that reflex phenomena, instead of being confined to a particular district, become more or less widely distributed; that the tone of muscles, and consequently the action of the sphincters, is due to reflex action in constant automatic operation; and, lastly, that the nutrition of muscles and probably of other tissues, and secretion, are largely influenced directly or indirectly by the spinal cord.

h. We only deem it necessary to remark in conclusion upon the olfactory and the optic nerves. There is good reason to believe, partly on anatomical, but mainly on pathological, grounds, that the

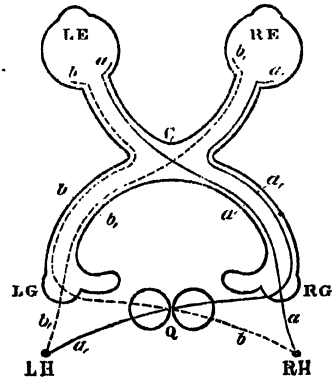


FIG. 7. Scheme of decussation of optic tracts.

L E and R E, left and right eyes; C, chiasma; L G and R G, left and right geniculate bodies; Q, corpora quadrigemina; L H and R H, left and right centres of vision; *b* and *a*, nerve-fibres from left and right sides respectively of left eye; *b*, and *a*, corresponding fibres from right eye.

olfactory nerves, unlike all others, do not decussate, or at any rate that the olfactory nerve of each side is connected chiefly with the supreme centre of smell of the same side. The arrangement and course of the optic nerves are peculiar. But experiment and clinical observation demonstrate facts in relation to them which anatomy alone does not teach us. We have shown that destruction of the supreme centre of sight on one (say the left) side causes total blindness of the right eye, a result which equally follows destruction of the right optic nerve; whereas destruction of the optic tract, corpora geniculata, or corpora quadrigemina of one side causes hemiopia of both eyes. A reasonable explanation of these phenomena is offered in the accompanying diagram, for which we are indebted to Prof. Charcot.

11. *Sympathetic system.*—The sympathetic system of nerves appears to have its supreme centre in the medulla oblongata, or rather on the floor of the fourth ventricle; but it is intimately interwoven with the spinal system, and, as is well known, each spinal nerve receives branche from, and transmits branches to, a neighbouring sympathetic ganglion. We need not consider the anatomical details of this system; it is sufficient to point out that it presides over the shortening and lengthening of the organic muscular fibres wheresoever situated, that it determines the dilatation and contraction of the blood-vessels, and therefore the amount of blood supplied to various parts, and in some degree the rapidity of its flow through them, and that it thus regulates to some extent the nutritive and other functions of the different parts of the organism and their temperature. There is some reason also to believe that special branches are supplied to the secreting cells of some, if not of all, of the glandular organs, and that hence a direct influence is exerted by it over the physiological processes which go on in these organs.

12. *Arteries of brain.*—The meningeal arteries are derived mainly from branches of the external carotids; but a minute branch is furnished also by each vertebral immediately after its entrance into the skull. They have no connection with the arteries which supply the brain and its vascular membrane, the pia mater.

The proper arteries of the brain are derived from the internal carotids and the vertebrals, which, after entering the skull and giving off certain branches, to some of which we shall presently again refer, form between them that remarkable anastomosis known as the circle of Willis. Each internal carotid artery having first given off the ophthalmic and then the posterior communicating artery, divides into two branches, namely, the anterior and the middle cerebral. The *anterior cerebral* (figs. 6, 8, 9, and 10), which is the smaller of the two, anastomoses after a short course with its fellow by the anterior communicating artery. Its trunk then turns round the anterior edge of the corpus callosum, and runs backwards along the upper surface of that body. It divides into three principal branches, of which the first is distributed superficially to the orbital convolutions below, and to a small portion of the inner aspect of the hemisphere in the neighbourhood; the second is distributed to the first frontal convolution, to the greater part of the second, to the upper extremity of the ascending frontal, and to all that part of the inner aspect of the hemisphere which lies between the area of distribution of the first branch and the ascending limb of the fronto-parietal fissure, including the anterior two-thirds of the corpus callosum; the third branch supplies that area of the inner surface of the hemisphere which lies between the ascending limb of the fronto-parietal and the parieto-occipital fissure, and also the posterior part of the corpus callosum. The *middle cerebral* artery (figs. 6, 8, 9, and 10) divides in the fissure of Sylvius into four branches,

which, radiating in conformity with the convolutions of the island of Reil, and supplying them with vessels, emerge on to the outer surface of the brain, and are thus distributed:—the anterior or first branch ramifies over the third frontal convolution exclusively; the second is distributed to a portion of the second frontal convolution and to almost the whole of the ascending frontal; the third supplies mainly the ascending parietal and superior parietal convolutions, the posterior and

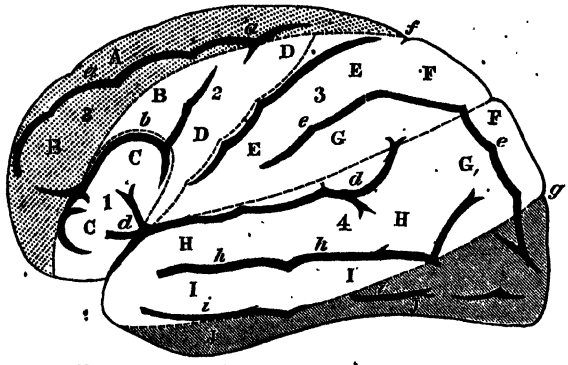


FIG. 8. Lateral view of brain, showing arterial areæ.

FIG. 9. Inner surface of cerebral hemisphere, showing arterial areæ.

In the above figures, and also in Figs. 6 and 10, the dotted surfaces correspond to the anterior cerebral artery; the clear surfaces to the middle cerebral; the shaded surfaces to the posterior cerebral. The sub-divisions of these surfaces made by dotted lines indicate the areæ supplied by the principal branches of the above arteries, and the Arabic numbers attached to them the order of the branches from before backwards.

lower limit of its distribution being indicated partly by the inter-parietal fissure, and partly by a horizontal line drawn from this to a point on the upper margin of the hemisphere midway between the fronto-parietal and parieto-occipital fissures; the fourth or posterior branch is distributed to the first and second temporal convolutions,

and to the gyrus angularis, its posterior limit being determined by a line drawn from the posterior extremity of the second temporal sulcus to the parieto-occipital. The *posterior cerebral* arteries (figs. 6, 8, 9, and 10) result from the division of the basilar; each sends branches into the brain-substance of the locus perforatus posticus, is then joined by the posterior communicating artery of the same side, and finally gives off three principal branches, which are distributed to all those parts of the cerebral surface which have been hitherto unaccounted for: the anterior to the uncinate convolution; the middle to the third temporal and the fusiform or lateral occipito-temporal convolutions, and to the hinder part of the gyrus fornicatus; and the posterior to the median occipito-temporal convolution, to the cuneus, and to the occipital lobe.

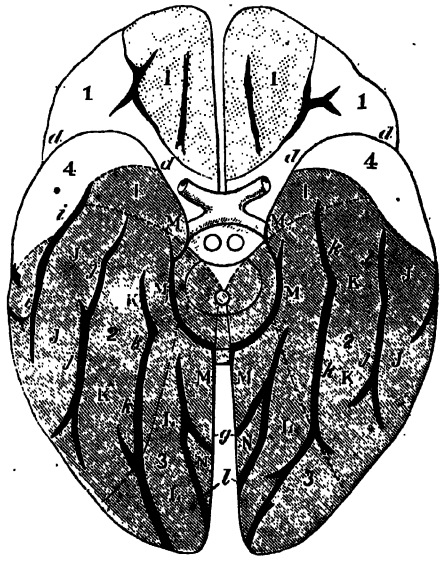


FIG. 10. Under surface of brain, showing principal convolutions and fissures and the arterial areæ.

The distribution of the arteries to the ganglia at the base of the brain is not less important than that upon the surface of the organ. All three pairs of cerebral arteries for the most part take a share in supplying these bodies. The anterior cerebral gives small branches to the anterior extremity only of the caudate nucleus, and not unfrequently none at all. The middle cerebral, on the other hand, has a comparatively wide and a very important distribution. It gives off many branches of somewhat large size, which, entering at the locus perforatus anticus at right angles, or nearly so, to the trunk, supply the whole of the lenticular nucleus of the corpus striatum, the whole

or greater part of the caudate nucleus, the internal capsule, and the anterior and outer part of the optic thalamus. They may be divided into two groups: an internal group, consisting of comparatively small vessels, which are distributed to the internal portions of the lenticular nucleus, and to the adjoining portions of the internal capsule; and an outer group of vessels of considerably larger size, which course mainly over the outer aspect of the lenticular nucleus, and supply the outer part of that body, and also, according to their position, the caudate nucleus or the optic thalamus. One of these branches, called by Charcot 'the artery of cerebral hemorrhage,' is pre-eminently large, and, after penetrating the outer part of the lenticular nucleus, and traversing the internal capsule, enters the substance of the caudate nucleus, and passes from behind forwards in it to its most anterior part. The posterior cerebral arteries give branches to the choroid plexuses and ventricular walls, and supply also the tegmentum, the corpora quadrigemina, and the posterior and inner parts of the optic thalami. The branches which they give to the last-named bodies may be divided into internal and external. The former supply the inner aspects of the thalami, and their rupture is apt to be followed by the profuse escape of blood into the ventricular cavities; the latter supply the outer parts of the thalami, and since before they enter them they pass through the cerebral peduncles, their rupture is apt to be attended with effusion of blood into the substance of these bodies.

The vertebral arteries unite to form the basilar, which divides in front into the two posterior cerebrals. The vertebrals, besides supplying meningeal and spinal branches, give off on either side a posterior inferior cerebellar artery, which is distributed to the hinder portion of the lower aspect of the cerebellum and to the choroid plexuses of the fourth ventricle. The basilar, in addition to sending a branch to each internal ear and other branches to the substance of the pons, gives off also a right and a left anterior inferior cerebellar artery to the anterior part of the under surface of the cerebellum, and a right and a left superior cerebellar artery, which are distributed over the whole of the superior surface of the cerebellum, and supply the valve of Vieussens and partly the velum interpositum.

It is necessary to bear in mind—for, indeed, it is this fact which makes an accurate acquaintance with the details of the cerebral circulation important—that, save at the circle of Willis, little or no communication takes place between the branches of the cerebral arteries even down to their finest ramifications, excepting by means of capillary vessels; and that hence, if any artery become obstructed, the region to which it leads almost necessarily suffers in its whole extent. Thus, if the middle cerebral be blocked, the nutrition of the whole region to which it is distributed becomes impaired; if one of its primary branches be obstructed, the limitation of morbid change is equally definite; and if a secondary or even smaller vessel be alone involved,

secondary changes will be limited to correspondingly minute districts. It is further important in reference to this point, to know that the arteries on the surface of the convolutions give off long and short branches, which are quite distinct from one another; and of which the short are distributed to the cortical grey matter, the long enter the white substance, and are limited in their distribution to it. It is scarcely necessary to add that the ultimate arteries supplying the ganglia are equally distinct from both.

It is a matter of no slight practical importance that the ophthalmic artery arises from the same trunk as that which gives off the anterior and middle cerebral arteries; and that it supplies not only the eyeball itself, but the contents of the orbit including the lachrymal gland, and gives off branches to the eyelids and contiguous parts of the forehead and nose, and to the ethmoidal cells. The arteries of the internal ear again are mainly derived from one of the intra-cranial arteries, namely, the basilar.

13. *Veins of brain.*—The veins distributed over the surface of the cerebrum and cerebellum open into the several sinuses to which they are respectively contiguous. Those situated within the lateral ventricles converge to the venæ Galeni, by means of which they empty themselves into the straight sinus. It is needless to enumerate or trace the several sinuses. There are, however, two or three points in connection with the venous circulation of the brain, which are important. These are:—first, that the cerebral and cerebellar veins all converge, directly or indirectly, to the lateral sinuses, and that hence all or nearly all the blood from these parts is returned by the internal jugular veins; second, that the ophthalmic vein, which has almost exactly the same distribution as the ophthalmic artery, empties itself into the cavernous sinus on the one hand, and on the other anastomoses with the branches of the facial and other veins; and, third, that the longitudinal sinus communicates with the veins on the exterior of the skull through the parietal foramen, and the lateral sinuses with those of the head and neck through the mastoid foramina.

B. *Pathology.*

Most diseases of the nervous system may affect any part of that system; and hence, although in many cases producing symptoms indicative of their specific nature, they tend also to evoke symptoms referrible to the particular regions of the nervous organism which they involve, and to the degree and manner in which they involve them. As regards the last point, it is obvious that here as elsewhere the functions of parts may be impaired, exalted, or perverted. And as regards locality, it is clear that disease may involve some portion of the motor tract, some portion of the co-ordinating tract, or some portion of the sensory tract; that it may be seated either in the peripheral nerves, in the cord, or in the intra-cranial centres; and that

the symptoms will vary accordingly. Further, if the supreme centres be involved, there will be not only pathological sensory or motor phenomena, but also phenomena referrible to the intellectual and emotional functions. We proceed to discuss some of the more important phenomena which are dependent on the situation of the parts affected, and on the degree and manner of their involvement.

1. *Motor Paralysis. Paresis.*

By the term "paralysis' is meant the more or less complete loss of that power which the different motor centres should exert over the movements of the muscles. The term 'paresis' is often used of the slighter forms of this condition. Paralysis of the voluntary muscles, to which alone we now confine our attention, may vary from the slightest degree of impairment of voluntary power over them to that condition in which every trace of such power has disappeared, and the part affected is absolutely motionless and incapable of motion. The quality, also, of this paralysis varies in different cases. In some, as in general paralysis of the insane and disseminated sclerosis, the enfeebled muscles become tremulous under the attempt to use them; more commonly, as in most cases of ordinary hemiplegia, their movements are slow, weak, and halting, but uniform. In some cases the paralysed muscles retain their normal bulk, in some they waste. Sometimes they are flaccid, sometimes they preserve their natural tonicity, sometimes they are rigid and perhaps contracted. In some cases, again, they more or less completely lose the power of reacting to faradism or other forms of irritation; while occasionally their contractility remains unimpaired or even becomes increased. And, lastly, in different cases the electro-sensibility of the affected muscles becomes weakened or exalted, or remains unaffected.

a. Cerebral paralysis—i. *General paralysis* seldom occurs excepting in association with insanity, and is then due, as might be supposed, to some general impairment of the surface of the brain. It is for the most part slight in degree, and indicated by feebleness, not only of the muscles of the limbs, but of those of the trunk, head, and neck, and of those of expression, mastication, and deglutition. Further, as has already been pointed out, the muscles are usually slightly tremulous when put into action.

ii. *Hemiplegia*, or paralysis limited to the distribution of the motor nerves of one side of the body, is due as a rule to disease of the opposite cerebral hemisphere, corpus striatum or crus. Its most common cause probably is disease implicating the corpus striatum or the white matter immediately external to it; and it is in such cases that hemiplegia presents its typical characters. The paralysis, as has been observed, is limited to the opposite side of the body; but it does not affect the whole side uniformly; for while some nerves are almost always affected in a greater or less degree, others almost invariably

escape or suffer very lightly. Those which escape are such as act in association with corresponding nerves of the opposite side, whose combined actions we cannot voluntarily restrain, and whose nuclei are probably (as Dr. Broadbent suggests) more intimately connected with one another than are the nuclei of other symmetrically placed nerves, and are hence influenced in a greater degree than these by motor impulses which descend from the other side of the brain. The third, fourth, and sixth nerves seldom if ever suffer, so that the motions of the eyeball on the affected side continue, for the most part, perfect. Again, the motor-root of the fifth nerve suffers, as a rule, but little. The portia dura, on the other hand, generally is distinctly though slightly and unequally involved; thus the face is usually more or less blank on the affected side, the muscular wrinkles more or less effaced, the mouth drawn to the opposite side, the eye a little more open than its fellow, and winking somewhat laggingly performed; nevertheless the eye can generally be perfectly closed, and some power of movement remains in the whole of the side of the face, but more especially in its upper half. The hypoglossal is almost invariably markedly involved, and the tongue consequently is protruded with its tip pointing towards the paralysed side. The motor fibres of the par vagum, and the motor roots of the spinal nerves going to the muscles of the head and neck and trunk, suffer but little; and hence the patient as a rule has no difficulty in deglutition, in phonation, in maintaining the due position of his head, in respiration, or in acts needing the employment of the muscles of the abdomen or back. The nerves of the arm and leg are always chiefly affected. If the case be severe both limbs are alike motionless; but it is a curious fact that if there be a difference between them it is generally that the leg retains a greater degree of motor power than the arm, that it is the last to fail, the first to recover. But the distribution of paralysis is liable to variation, and occasionally the leg escapes wholly, occasionally it is affected in a higher degree than the arm.

Disease situated in the substance of the hemisphere is also generally attended with hemiplegia; especially disease occupying the frontal or parietal lobe. And it is in such cases that our diagnosis of the seat of mischief and the cause of mischief may be aided by the facts which have already been discussed, with regard to the localisation of function in the grey matter of the convolutions, and the areæ of distribution of the cerebral arteries. If the hemiplegia be attended with aphasia we may assume that either the posterior part of the third frontal convolution, or else the wedge of white matter extending thence to the corpus striatum, is involved.

When the lesion is situated in the crus cerebri, together with hemiplegia of the opposite side of the body, there will probably be paralysis of the third nerve of the same side.

b. Bulbar paralysis.—When paralysis arises from disease of the

medulla oblongata or pons Varolii, it is obvious, from the abundance and close proximity of important nerves and nerve-nuclei in these organs, and from the fact that the sensory and motor strands from both cerebral hemispheres here meet and blend, that such one-sided limitation of paralysis as occurs in hemiplegia is scarcely likely to be present, and that if there be general paralysis it must differ largely in its details and in its danger to life from that which has before been adverted to. It is mainly in such cases that what is called *cross paralysis* is met with—paralysis, that is to say, of one side of the body and of the opposite side of the face. It is in such cases, again, that we sometimes find paralysis of both arms and legs, or of one arm and both legs, or the converse. And, moreover, it frequently happens, for obvious reasons, that there is more or less paralysis of the muscles of one or other or both eyeballs, or of one or other or both facial nerves; or that there is difficulty of articulation, phonation, mastication, deglutition, or respiration, or of control over the rectum and bladder; or that a greater or less number of these paralyses occur in combination. It must be recollected, in reference to these cases, and equally in reference to diseases involving the under surface of the brain, that, together with the opposite or hemiplegic paralysis due to involvement of nerve-tissue above the nerve-nuclei, we are always apt to have paralysis, generally of the same side, due to the direct implication of nerve-nuclei, or of nerves after their emergence from their nuclei. It is by this circumstance that cross paralysis is to be explained. The great danger to life which, as is well known, attends disease of the parts now under consideration is due mainly to paralysis of the nerves supplied to the organs of deglutition, respiration, or circulation, which is almost invariably present in a greater or less degree.

c. Spinal paralysis. Paraplegia.—When paralysis is due to disease of the spinal cord, it generally goes by the name of paraplegia, and is specially characterised by the fact that the paralysis involves only the muscles supplied by those nerves which are given off from the cord at and below the seat of disease. The symptoms will of course vary, both with the situation and with the extent of the lesion. Thus if it involve the whole thickness of the cord high up in the neck above the origin of the phrenic nerves, there will be complete motor paralysis of all parts seated below—of the arms and legs, as also of the diaphragm and other respiratory muscles. If it be situated at or above the cervical enlargement, the movements of the diaphragm will be unaffected, but the arms and legs will be paralysed as in the former case. If the dorsal region of the spine suffer, the arms will necessarily escape, and the paralysis will be limited to the lower extremities and to just so much of the lower part of the trunk as is supplied by nerves given off below the seat of mischief. In all such cases there is more or less interference with the functions of micturition and defæcation. If the disease be seated high up, as in the cervical or upper dorsal region, there is

difficulty in the act of micturition, owing to spasm of the sphincters; if, on the other hand, the disease be situated in the lower dorsal or lumbar region, the sphincters are paralysed, and the urine runs away. The bowels are usually constipated, and defæcation is performed involuntarily. It need scarcely be added that in complete paralysis sensation as well as motion is annulled.

But paralysis below the seat of lesion is not necessarily complete. In many cases where it is due to pressure, or to disease of the surface of the cord, or of the structures which surround it, sensation remains perfect, or nearly so, while motorial power is wholly lost. In many cases, again, the paralysis, though involving all parts below equally, involves them only to the extent of impairing their power of motion. Further, many cases are met with in which the disease implicates unsymmetrically certain defined tracts only of the cord. The consequences are often very remarkable. If one lateral half be diseased in its whole horizontal extent, but to a limited extent vertically, complete paralysis necessarily involves all the motor nerves given off from the cord on the same side as the lesion, but below it, in consequence of the lesion having cut off all direct connection between them and the brain above. But, inasmuch as the decussation of the sensory nerves takes place in the cord itself immediately after their entry into the cord, it follows that the sensory nerves associated with the paralysed region remain unaffected, while those of the corresponding region of the opposite side of the body share the fate of the motor nerves of the diseased side. Hence arise: paralysis with retention of normal sensation on the one side; anæsthesia, with perfect power of motion, on the other side; and in some cases a more or less distinct line of anæsthesia forming, on the side of lesion, the upper limit of the region of motor paralysis. Perfect unilateral limitation of disease is of course rare; it is more usual to find one side involved in a portion only of its horizontal extent, or both sides involved more or less, and in unequal degrees; under which circumstances the resulting paralytic phenomena are of course less typical, and irregularly distributed.

It is a curious fact, which will hereafter be more fully considered, that certain forms of disease have a remarkable tendency to involve particular regions or strands of the cord, and to be limited to them. The parts to which particular reference is here made are the posterior columns, the lateral columns, and the groups of large or motor cells in the anterior cornua. When disease affects the posterior columns only, or, as Charcot points out, the outer bands of these columns which abut directly on the inner aspects of the posterior cornua and the roots of the sensory nerves, the condition known as locomotor ataxy, or in other words, loss of co-ordinating power, and not ordinary motor paralysis, involves the voluntary muscles of all those parts which are below the seat of disease. In a large proportion of cases the legs alone suffer, but the arms and even parts above the arms are all liable to become

implicated. Inco-ordination is shown: partly by loss of the muscular sense, in virtue of which the patient is unable to judge of the amount of force needed to accomplish definite results, and unable therefore (especially if his eyes be closed) to determine the position of his affected limbs in relation to other parts of his person or to surrounding objects; and partly by want of control over his voluntary movements, which are consequently more or less violent than necessary, and involve a larger or smaller number of muscles than are suitable for their execution. There is not, however, any necessary loss of muscular strength, and the affected limbs sometimes retain extraordinary power. When the lateral columns only are the seat of disease, or more particularly the white matter which lies behind the horizontal line drawn laterally through the median canal, motor paralysis ensues in all those parts which are situated below the seat of lesion; but under these circumstances, according to Charcot, the muscles of the affected limbs tend to get, not only paralysed, but at first tremulous and ultimately more or less rigid and contracted. If the groups of large cells in the anterior cornua are diseased, then only the nerves which take their origin in them, and those muscles which these nerves supply, suffer: the muscles become paralysed, and in a large number of cases speedily lose their faradic contractility, and waste.

d. Nerve paralysis.—In the foregoing account we have considered more especially those forms of paralysis which are due to disease occurring above the nuclei of origin of the paralysed nerves. We have, however, referred here and there to the fact that paralysis may be caused by disease involving either these nuclei, or the nerves after their emergence from them. We have, indeed, in considering paralysis due to disease originating within the brain or cord, been almost compelled to advert to the fact that, when certain parts, more especially the pons, medulla oblongata, base of the brain, and spinal cord, are affected, the paralysis which ensues is necessarily apt to be compounded of paralysis due to the cutting off of the connection between nerve-nuclei and the higher centres, and of that dependent on direct implication of nerve-nuclei or nerves. Paralysis from destruction of a nerve or of its nucleus of origin, is necessarily of very limited distribution; it affects a single muscle or a group of muscles, as for example the external rectus of one eye, or the superior oblique, or the other muscles of the eyeball together with the levator palpebræ, or the muscles of expression of one side of the face, or certain muscles of the head and neck, trunk, or extremities. It also tends soon to become absolute. It is not, of course, denied that other varieties of paralysis are often absolute; but, as we have pointed out, in ordinary well-marked hemiplegia certain nerves appear to escape implication, and certain others, such as the portio dura, become involved only to a slight extent. In primary paralysis, however, of the portio dura, the paralysis of the muscles which it supplies is for the most part general

and complete. Further, the paralysed muscles usually rapidly lose the power of responding to the faradic stimulus, and at the same time grow flaccid, and waste.

e. Disease of the cerebellum.—Diseases of this part are, no doubt, often attended with loss of sight, and with hemiplegia; but these phenomena are accidental accompaniments of cerebellar lesions, and due either to the direct implication of some neighbouring part, or to pressure exerted by the diseased cerebellum on the adjoining quadrigeminal bodies or subjacent medulla oblongata. The usual and natural result of cerebellar disease is a staggering gait like that of a drunken man, or in extreme cases a total inability to stand or walk; in consequence, not of muscular debility or mere inco-ordination of the movements of the lower extremities as in tabes dorsalis, but of a general impairment or loss of the power of maintaining equilibrium. Nystagmus and parallel deviation of the optic axes are also liable to occur in affections of the cerebellum.

f. Condition of muscles in motor paralysis. i. *Tone.*—In some cases of paralysis the muscles retain their normal tonicity; in some they get limp and flaccid; in some they become rigid and contracted. The normal tonicity is preserved in a large number of cases of both cerebral and spinal paralysis. It is essential, indeed, for its conservation that the connection between the muscles and the cord remain intact. Limpness of muscles not unfrequently attends those cases of paralysis of the same centres in which the affection which causes paralysis is sudden in its onset and extensive; it generally also soon becomes developed in those muscles whose nerve-nuclei are directly implicated, and in those whose connection with these nuclei is interrupted. Rigidity or contraction of the muscles in cerebral or spinal disease is often the consequence of some irritation, inflammatory or other, going on at the seat of disease. It is then to be regarded as an acute condition, and generally comes on early. But rigidity, with more or less contraction, is apt to ensue gradually in cases of old paralysis; sometimes, in the case of atrophied muscles, from their gradual and slow longitudinal contraction; more frequently, perhaps, in consequence of secondary degenerative changes going on in the lateral columns of the cord.

ii. *Contractility and irritability.*—The contractility of muscles under the influence of the faradic stimulus remains unimpaired in many cases of paralysis. In some cases, however, it becomes exalted, in some diminished, or annulled. Contractility is for the most part retained both in cerebral and in ordinary spinal paralysis; but in both, and more especially in the latter, it is not unfrequently exalted, so that the paralysed muscles are more readily thrown into contraction by a weak current than are the muscles which are still healthy. Loss of faradic contractility usually takes place to some extent in the muscles of paralysed limbs which have been long disused, in consequence simply of their disuse; and when atrophic or degenerative changes are

going on in muscles their contractility is necessarily impaired at least proportionately to the amount of injury or destruction that has taken place. The most remarkable instances, however, of such loss are those in which the paralysis is due to disease involving either the nerve-nuclei or the nerves connected with the affected muscles. In some of these cases the loss of faradic contractility is marked and rapid, and not unfrequently it becomes totally abolished in the course of from five to ten days. It is important to note, however, that the galvanic current acts in a very different way to the faradic current; that when slowly interrupted it acts far more powerfully on many paralysed muscles than on healthy muscles; that in the cases just adverted to, attended with rapid loss of faradic contractility, readiness to respond to the slowly interrupted galvanic current increases with the disappearance of the former; and that at length even a feeble galvanic current will often readily evoke contraction in muscles which have become wholly irresponsive to faradism.

iii. *Faradic sensibility.*—This condition (provided complete anæsthesia do not attend the muscular paralysis) is generally augmented with augmentation of muscular contractility, and diminished with the diminution of that property. But occasionally, as for example in hysteria, contractility remains when muscular sensibility has disappeared; while, on the other hand, it now and then happens that sensibility continues after the muscles have almost entirely ceased to contract.

iv. *Nutrition.*—In a considerable number of cases paralysed muscles retain their bulk and texture, or at most become slightly impaired in these respects, as even non-paralysed muscles are apt to do, from mere disuse, and hence remain in a condition to take on active duty so soon as the cause of paralysis disappears. This is generally the case when the cause of paralysis lies above the nuclei of origin of the paralysed nerves. In these cases, also, the muscles generally retain their tone and faradic contractility little or not at all impaired. When, however, the motor nuclei or nerves emanating from them are the seat of disease, rapid muscular emaciation usually takes place concurrently with loss of faradic contractility. To this subject we shall subsequently recur.

v. *Reflex action.*—Involuntary movement of paralysed muscles, in obedience to reflex irritation, is a phenomenon of common occurrence. The most striking forms of reflex action occur in cases of spinal paralysis, in which some circumscribed lesion cuts off all nervous connection between the brain and paralysed limbs, leaving the portion of cord with which those are connected in a healthy condition. In such cases, sometimes under the influences of defæcation or micturition, sometimes from the irritation of bed-clothes, but more strikingly from touching or tickling the soles, the paralysed limbs may be made to execute violent and repeated movements. When one sole is irritated the corresponding limb may be made by successive efforts to become powerfully flexed at the hip, knee, and ankle-joints, while the toes are widely

separated and extended. In most cases these reflex movements are limited to the irritated member; but in some instances both limbs become involved, and occasionally the muscular contractions are still more widely distributed. Similar reflex phenomena may generally be excited in the paralysed limbs of hemiplegic patients. It is obvious that reflex phenomena can arise only where the connection between the paralysed muscles and the nerve-centres of the cord are maintained; and that hence they can never be present in parts whose paralysis is due to the destruction of nerves or nerve-nuclei. A phenomenon referrible to the same class is sometimes observed in patients suffering from cerebral paralysis, namely, the sudden movement of a paralysed limb or of certain groups of paralysed muscles under the influence of emotional excitement.

2. *Anæsthesia.*

Anæsthesia, or impairment or loss of sensation, may, like motor paralysis, exist in various degrees, and occupy various regions of the body. It may be limited to the skin or muscles, or may involve the whole thickness of the part affected. The last variety is the most common. In its slighter degrees it is often attended with more or less tingling, pricking, formication, or sense of numbness; and the affected part, in relation to things with which it is brought into contact, feels to the sufferer as if protected or covered by some thick soft texture. If his hands be the seat of anæsthesia, they seem as if clothed with thick gloves; if his feet, as if he were walking on cotton-wool or other soft yielding material. In extreme cases the skin and subjacent parts are wholly insensible to external impressions, and admit of being pricked, cut, burnt, or otherwise injured without the knowledge of the patient. In most cases abeyance of ordinary tactile sensibility is attended with similar abeyance of the capacity for distinguishing painful impressions, and heat and cold; but this is not always the case, for now and then the capability of recognising these latter impressions appears to survive in some degree when the capability of distinguishing the former is wholly lost—a fact which has led some physiologists to believe that these different forms of sensation travel to the sensorium by different routes. Muscular sensibility is sometimes impaired or lost in cases of paralysis while the cutaneous sensibility remains unaffected; and occasionally in hysterical cases muscular insensibility, with or without cutaneous anæsthesia, goes along with unimpaired muscular contractility.

There are a few other points connected with anæsthesia which claim attention. The first of these is the fact that persons suffering from anæsthesia often experience subjective sensations referrible to the anæsthetic regions, and not unfrequently complain of neuralgic and other pains in them; the second is that sometimes, when sensation is

greatly impaired, the patient does not take cognisance of impressions made on the affected part until after the lapse of a few seconds, or it may be as much as half a minute—the impression appears to be delayed in its transmission to the sensorium; the third is the fact that under similar conditions there is often a peculiar difficulty in distinguishing between the characters of different impressions; the last to which we shall refer is the circumstance that, inasmuch as it is through the sensory or afferent nerves that reflex motor phenomena are excited, it is obvious that if the disease causing anæsthesia exist in the course of the nerve or in its nucleus, no irritation of its extremity can evoke reflex action, while if it be due to spinal disease, the probability is that irritation of the extremities of the anæsthetic nerves given off below the seat of disease will excite muscular action in the corresponding muscles. Similar phenomena to the last, but of a more complicated character and higher order, are not unfrequently produced through the agency of the nerves of sight and hearing.

Anæsthesia, equally with motor paralysis, may depend on disease of the brain, disease of the medulla or other parts at the base of the brain, disease of the cord, or disease of nerves.

a. Cerebral anæsthesia.—i. *General impairment of sensibility* may attend the general paralytic condition which is associated with a special form of insanity.

ii. *Hemi-anæsthesia* may arise, like hemiplegia, from disease of one of the cerebral hemispheres or the ganglia or crus immediately connected with it. It is, however, of much less common occurrence than hemi-paralysis, and rarely occurs independently of it. Experiments on the lower animals and pathological observation concur to prove: that the optic thalamus has the same kind of relation to sensation that the corpus striatum has to motion; and that lesion of this part is attended with anæsthesia of the opposite side of the body. As a matter of fact, however, hemi-anæsthesia is generally due to lesion, not of the optic thalamus, but of the external capsule in relation with the optic thalamus, or of that portion of the commencement of the corona radiata which contains the fibres of communication between the optic thalamus and corresponding cerebral hemisphere. Cerebral hemi-anæsthesia, unlike cerebral motor paralysis, is for the most part universal throughout the affected side: and in connection with it there is frequently if not generally anæsthesia of certain of the nerves of special sense—deafness of the corresponding ear, loss of taste on the corresponding side of the tongue, and especially loss of sight in the corresponding eye. As regards loss of sight in the eye opposite the cerebral lesion it may be observed that this seems to occur when the lesion implicates the internal capsule. A case of Dr. Jackson's would seem to show that disease of the optic thalamus itself tends rather to cause hemiopia of both eyes. True smell is probably lost on the same side as the cerebral lesion; but tactile anæsthesia of the nostril occurs on the anæsthetic side.

Theory, and to some extent experience, would lead us to believe that disease affecting the temporo-sphenoidal and occipital lobes, and the posterior part of the parietal lobe, would be more likely to be attended with anæsthesia, than with motor-paralysis, of the opposite side of the body. And in connection with this subject it is important to recollect the position of Ferrier's centres of vision, hearing, smell, taste, and touch.

Affections involving the tegmentum of the crus would of course be attended with more or less marked hemi-anæsthesia of the opposite side.

Hemi-anæsthesia is in some cases absolute—the patient feels nothing. But more frequently it is incomplete—he feels generally in some degree; or he retains more or less sensation in certain parts, especially the palm or sole, or certain areæ of the leg or forearm; or, as occasionally happens, the affected side is irregularly studded with anæsthetic patches. Not unfrequently patients who are incompletely hemi-anæsthetic experience considerable pain when their paralysed limbs are moved, and refer the sensation due to pricking and other forms of pain inflicted on them to a joint or some other part remote from the seat of injury; and at the same time wholly misconceive the character of the impression.

b. Bulbar anæsthesia.—When disease involves the pons or medulla oblongata, some degree of anæsthesia is very apt to be associated with motor paralysis, and like it to be of irregular distribution. Of course all the sensory nerves which take their origin in these parts are, like the motor nerves, liable to be implicated.

c. Spinal anæsthesia.—Anæsthesia from spinal disease, like that from cerebral disease, is far less common than the corresponding paralytic affection, and is rarely met with apart from it. There are several reasons for this: the sensory tracts of the cord are limited almost exclusively to the posterior cornua, and the rest of the grey matter behind the central canal, so that they are apt to escape pressure and the other consequences of disease occupying the periphery of the cord or the surrounding tissues; moreover, it seems to be proved by experiment that a narrow thread of grey matter is sufficient to maintain an effective connection between the sensory tract below and that above. Disease limited to the central region of the cord, or rather to its posterior part, might conceivably induce anæsthesia without paralysis. We have previously pointed out the important fact that disease of one lateral half of the spinal cord, interrupting the longitudinal continuity of the fibres, causes anæsthesia of the opposite side of the body.

d. Nerve-anæsthesia.—Local anæsthesia, like local paralysis, may arise from disease affecting either a sensory nerve or its nucleus, and thus necessarily occupies the same side of the body as that on which the lesion exists. Such anæsthesiæ are not unfrequent in the area of distribution of the fifth pair or some of its branches, or in that of one

or other of the spinal nerves. The anæsthesia which forms the upper boundary of the paralysed region in cases of unilateral injury or disease of the cord is a typical example of this condition. The nerves of special sense may suffer equally with those of common sensation.

3. *Convulsions. Spasms.*

In speaking of paralysis, we have referred to the facts, that associated with this state it is not uncommon to observe tremulousness of muscles arising especially during voluntary efforts, and that paralysed muscles occasionally become rigid and contracted. It may be added that convulsive movements of various kinds are not unfrequently associated with paralysis. They more frequently occur, however, independently of it.

Convulsions may affect single muscles or portions of muscles, groups of muscles, a limb, the head and neck, one half of the body, or the whole of it. They may be intermittent or continuous, and may vary in intensity from a scarcely perceptible flickering of the muscular fibres, to contractions of such violence and strength that the muscles become ruptured. Intermittent contractions are termed *clonic*, persistent contractions *tonic*.

The slighter forms of convulsions are exemplified in the tremulous, more or less rhythmical, movements which are observed under many various conditions, and generally cease during sleep. Some of these attend efforts at voluntary movement only, and are then usually regarded rather as evidence of debility than as convulsions in the true sense of the term, on the ground that they depend on the intermittent transmission of voluntary impulses only. In true convulsions there is a similar intermittent transmission, but the force is exerted independently of and beyond the will. This distinction is useful to be borne in mind, but it is one that is not always available in practice, if indeed the two conditions do not frequently run into one another. Among the convulsions which on the above view might be referred to debility are the fibrillar tremblings of the lips and tongue of patients suffering from general paralysis during their attempts to speak; and the irregularly rhythmical movements of the limbs which not unfrequently attend the voluntary efforts of those labouring under disseminated sclerosis of the nerve-centres; among those which are truly convulsive may be enumerated that flickering of the orbicularis palpebrarum which is known by personal experience to all, the general tremulousness which attends exposure to cold and febrile rigors, the subsultus tendinum of patients in the typhoid condition, the more or less general rhythmical tremors of paralysis agitans and chronic mercurial poisoning, and the tremors which occasionally attack the lower extremities of paraplegic patients.

Another form of convulsion is that to which the term choreic may be applied, and of which chorea furnishes the most typical example.

In this, as in the former series, the movements cease during sleep, and as in the paralytic form of trembling are greatly aggravated during voluntary efforts and under mental excitement. Choreic movements are characterised mainly by their abruptness and irregularity, and by the fact that when they are engrafted on any voluntary movement they interrupt its progress by a series of grotesque contortions and diversions which are not then necessarily limited to the limb or organ which is making the effort. Choreic movements are not unfrequently hemiplegic or even limited to a limb. Related in some degree to chorea are the grimaces and other tricks of movement to which some persons acquire an uncontrollable impulse; the peculiar rotatory and other rhythmical or irregular motions to which hysterical females are occasionally addicted; and especially perhaps the redundant and awkward movements of locomotor ataxy. A peculiar form of convulsion also related to chorea has been described by Dr. Hammond under the name of 'athetosis.' It is characterised mainly by continued slow movements of the fingers and toes, and inability to maintain them in any position. It generally ceases during sleep, and supervenes on hemiplegia, in which more or less complete recovery of motor power has taken place.

The clonic convulsions of epilepsy and epileptiform conditions are characterised by more or less violent and rapidly repeated alternating movements—rapidly repeated alternate flexions and extensions of the arms or legs, jerkings of the head and neck, or similar movements of the muscles of the face or eyes. These, like choreic convulsions, are not unfrequently unilateral, but they may be general, or limited to a single limb or to part of a limb.

In tonic spasm or convulsion, muscular contractions take place, which are more or less enduring. It can readily be understood that the terms clonic and tonic can be employed only in a relative sense, and that clonic and tonic spasms pass into one another by insensible gradations. Tonic spasms are exemplified in the cramps which occur after fatigue in the muscles of the calf, or in various muscles in Asiatic cholera; in the contraction of the limbs which takes place in the course of some forms of paralysis; in the folding of the thumb into the palm, the gradual drawing up of the arm, or other comparatively slow and strong contractions of muscles, which are usually the earliest of the convulsive phenomena of the epileptic fit; and in the violent attacks of muscular rigidity which by their repetition constitute the characteristic sign of tetanus and strychnia-poisoning.

It is not always easy to localise the seat of the diseases causing convulsions. In reference, however, to this point, it must be recollected that all those parts which by their destruction cause paralysis of certain regions, are necessarily likely under irritation to cause motor phenomena in the same regions. Thus, since hemiplegia is determined only by destructive disease of the opposite cerebral hemisphere, corpus

striatum or crus cerebri, it may be taken for granted that convulsions affecting generally one side of the body must be caused by disease of the same parts. It is probable on this ground (but not on this alone) that choreic and epileptic convulsions, which are frequently unilateral, are of cerebral origin. Again, since paraplegia depends on disease affecting the cord, there is reason to suspect that convulsive affections presenting a similar arrangement are of spinal origin. Tetanus and strychnia-poisoning are cases in point, although it must be admitted that in both cases the lesion involves the medulla oblongata even more importantly than the cord. When a single muscle or group of muscles is affected, we must look to the origin of the nerve or nerves which supply it; and it is clear that theoretically the lesion might be referred, either to the nucleus of the nerve, or to a limited spot in the corpus striatum or in the grey matter of the cerebral convolutions in direct linear continuity with the nerve-nucleus. It may be observed, however, that the simpler and more restricted in area such limited convulsion is the more likely is it to be due to the influence of the nerve-nucleus; the more complicated and so to speak purposive, the more likely is it to be traceable to the action of the corpus striatum or cerebral convolutions.

The recent observations with regard to the intimate association between certain areæ at the surface of the brain and certain groups of muscular movements have a special importance in relation to the localisation of cerebral disease attended with convulsions. It will be recollected that irritation of each of these centres provokes specific combinations of convulsive movements; and these facts, together with Dr. Hughlings Jackson's, and (since his) other pathological observations on the same subject, demonstrate that we may in many cases determine the exact seat of cerebral lesion by paying minute attention to the details of convulsive attacks.

An important fact in connection with unilateral convulsive affections, such as chorea and epilepsy, due presumably to disease of the opposite cerebral hemisphere or corpus striatum, is that the convulsive movements are generally unilateral only in those parts, such as the arm and leg, which are chiefly paralysed in hemiplegia; while in those parts which are only slightly affected, or escape wholly, in hemiplegia, convulsive movements are not only present but are associated with similar movements of the corresponding parts on the opposite side. This fact confirms Dr. Broadbent's hypothesis (already referred to) with respect to the more or less intimate connection subsisting between the corresponding nerve-nuclei of opposite sides. For that intimate connection which in hemiplegia would allow motor influences descending from the opposite healthy hemisphere to be in certain regions diffused horizontally and thus to counteract paralysis, would necessarily equally allow abnormal motor impulses descending from the irritated hemisphere to influence in the same regions the motor nuclei of both sides, and thus to cause bilateral convulsions.

4. *Hyperœsthesia. Dysœsthesia.*

Augmented or perverted sensibility has the same relation to the sensory part of the nervous system that spasms and convulsions hold to the motor, and indeed the two conditions are not unfrequently associated. Hyperœsthesia means strictly exalted sensibility—a condition in which the various organs of sense are more readily affected than they should be by impressions which are made upon them, or in which the sensorium is more appreciative than natural of the impressions which are conveyed to it from the organs of sense. Practically, however, exalted sensibility is scarcely if ever distinct from painful sensibility. The hyperœsthetic eye cannot bear bright light, the hyperœsthetic ear is affected painfully by powerful, high, or discordant sounds, the hyperæsthetic skin shrinks from the slightest pressure or from mere contact. Hyperœsthesia in this sense is not uncommon; it is frequently observed in hysteria, sometimes in the early period of febrile disorders, occasionally in inflammatory and other affections of the central nervous organs. It is a common feature in hemi-paraplegia, in which case not only is the paralysed side generally still sensitive, but its sensibility often becomes painfully acute; it is common, too, in inflammatory affections involving the skin.

Under the general term dysæsthesia may be included a large number of abnormal sensations, referrible to the ordinary sensory nerves, to the nerves of special sense, and to the sympathetic system, or at all events to the afferent nerves connected with the visceral organs. Among perverted sensations referrible to the skin may be included sensations already adverted to as frequently indicating the advance of anæsthesia, namely, numbness, sense of coldness, tingling, formication, and the like; as also itching, burning, cutting, stabbing, crushing, shooting, aching, constrictive and other pains, which are so common and arise under so many various conditions that it would be a waste of time to endeavour to enumerate them all. True neuralgic pains are usually of a shooting character, flash with momentary intensity along the fibres of the affected nerve, and occur in paroxysms composed of momentary shocks following one another in rapid succession. Other varieties of dysæsthesia are those which are manifested in relation to visceral organs, among which may be included the 'want of breath,' which attends asthma and cardiac disease; the agony of angina pectoris; painful thirst or craving for food; gastralgia, enteralgia; and various indescribable sensations referrible to different parts of the body, of which nervous and other patients complain, or which constitute many of the varieties of the so-called epileptic aura. Dysæsthesia of the organs of special sense may be indicated by the appearance of subjective phenomena referrible to these organs: of the eye, by the appearance of sparks or flames, or other objects, which may even pre-

sent definite forms, be endowed with motion, and assume the visible attributes of living objects; of the ear, by the perception of sounds, such as humming, buzzing, singing, the ringing of bells, violent explosions, and even words and actual conversation; of the nose, by the perception of odours; of the taste, by the perception of flavours.

5. Influence of Nervous Diseases over the Nutritive Processes.

a. Sympathetic system.—There is no doubt that the sympathetic system is largely concerned in morbid processes. We know how importantly constriction of the bronchial tubes is connected with asthma; what an essential part contraction of the vessels plays in the production of the phenomena of angina pectoris, and of the anæmia of the brain which, as a rule, precedes the epileptic attack; how largely dilatation of vessels is concerned in inflammation; and also that diabetes and various other disorders have been attributed to dilatation of the vessels of the liver or other organs with consequent hyperæmia of these parts. We need not, however, consider in detail the various pathological phenomena which are rightly or wrongly attributed to the influence of the sympathetic nerves. It is sufficient to say that these, so far as the vessels are concerned, solely determine variations in diameter; that, if contraction take place, less blood reaches the tissues, which then suffer in their functional activity, and even in their nutrition; that if, on the other hand, dilatation take place, the tissues become hyperæmic and the various physiological processes proper to them stimulated into unwonted activity. Still, however much the affected parts suffer, temporarily or permanently, in their functional attributes, it does not appear that their nutrition necessarily gets checked or perverted beyond the limits of health; the anæmic tissues do not necessarily fall into degeneration or decay, the hyperæmic tissues do not necessarily pass into inflammation or pathological overgrowth. It must nevertheless be admitted that the hyperæmic tissues when exposed to irritation are more liable to become inflamed than healthy tissues are.

b. Cerebro-spinal system.—Admitting fully, however, the essential part which the sympathetic system plays in the regulation of the nutritive processes both in health and in disease, and admitting also the little obvious influence which the spinal system of nerves exerts over the same phenomena in health, it seems certain that it is to the spinal rather than to the sympathetic system that we must refer certain localised lesions which are apt to take place in the course of nervous disorders, and which we are now about to discuss. We refer more particularly to certain affections of the muscles, certain affections of the joints, certain affections of the skin and subjacent tissues, and certain affections of the viscera, especially the kidneys and bladder.

i. *Muscles.*—We have already shown that in many cases of motor

paralysis the implicated muscles retain their healthy texture, their bulk, and their contractility, under the influence of stimuli, and, it may be, retain these qualities scarcely impaired for an indefinite period; only after a while wasting to a slight extent from mere disuse, and possibly undergoing some secondary degenerative change. Other cases of motor paralysis, however (cases for the most part of acute onset), are attended with rapid loss of faradic contractility and concurrent acute wasting of the paralysed muscles. These consequences may follow: first, on lesions of motor nerves; second, on affections of the cord; and, third, but much less frequently, on cerebral disease; but they do not follow indifferently on all forms of disease or injury of these several parts. It is doubtful if simple destruction of nervous tissue, however complete, is sufficient to induce them; nor have they relation to the completeness of the paralytic phenomena. They appear to be due mainly to disease or injury causing irritation of the nervous tissue which is its seat, and especially to disease or injury involving the motor nerves or motor centres. If, therefore, muscular emaciation and loss of contractility be the consequences of lesion of a nerve, that lesion is almost certainly not a clean cut, but the result of pressure, stretching, bruising, laceration, inflammation, or implication in some growth; if they accompany spinal disease they are due to disease, probably irritative, involving the anterior cornua, and particularly those groups of giant cells which are in direct relation by means of motor nerves with the affected muscles. No doubt many different forms of spinal affection are apt to be followed by muscular atrophy. Locomotor ataxy, disseminated sclerosis, and disease limited in the first instance to the lateral white columns, may each by horizontal extension involve the anterior cornua at certain points, and so induce irregularly distributed atrophic changes of the muscles; and, similarly, caries of the vertebræ, fracture of the spine, and tumours, may each of them, by pressure or otherwise, implicate the grey matter in their immediate vicinity and hence cause atrophy of the muscles supplied by the nerves emanating from the seat of lesion. Further, diffused inflammation, and hemorrhage into the substance of the cord, both of which conditions may involve the central parts of the cord in a considerable length, may result in widespread muscular lesion. But the spinal affections which are the most common causes of muscular atrophy are those which induce infantile paralysis and equivalent conditions in the adult. In these the disease, which appears to be of an inflammatory nature, originates apparently in the groups of large cells occupying the anterior cornua, and even if distributed throughout the whole length of the cord may still be limited to them. When acute atrophy of muscles occurs as a sequela or consequence of cerebral disease, it is probably always directly referrible to some secondary descending lesion of the cord implicating the motor nuclei. Post-mortem examination seems to show that, in the particular form of wasting of muscles here discussed,

the implicated nervous tissue from the seat of lesion downwards undergoes (mainly in its connective-tissue elements) inflammatory proliferation; and that the initial change in the muscles is similarly an inflammatory hyperplasia of the connective-tissue elements and of the nuclei of the sarcolemma, inducing a kind of cirrhosis, to which the diminution in size of the muscular fibres is secondary. These fibres, though gradually becoming more and more attenuated, seem rarely, if ever, to lose their striation or to become fatty.

ii. *Joints and bones.*—Irritative affections of nerves are sometimes followed by inflammation of joints or periosteum, which may terminate in disorganisation and necrosis. But joint-affections are also occasionally developed, in the course of diseases of the cord or brain, in the members which are paralysed. Charcot divides these joint-affections into two groups. In the one the attack is acute or subacute, and attended with tumefaction, redness, and often more or less severe pain. In the other the attack commences suddenly, with diffused swelling of the limb, is attended with little or no pain, but involves the rapid erosion and disappearance of the cartilages and joint-ends of bones. The former of these affections has been met with in paraplegia from injury to the spine or from caries, and then most frequently in the knee. It has been observed also in hemiplegia, and mainly in hemiplegia due to softening; it then occurs usually in the joints of the upper extremity, which it attacks as a rule from two to four weeks after the occurrence of paralysis and at the time when chronic contraction is taking place. This affection is essentially synovitis. The other form of the disease has been most frequently observed in persons suffering from locomotor ataxy, and for the most part at the onset of the symptoms of inco-ordination. It usually occurs in the knees, shoulders, or elbows, and is attended with much effusion into the joints, rapid destruction of the joint-surfaces, and not unfrequently dislocation. The processes here adverted to are not unlike those which occasionally attend rheumatism. The diagnosis, however, between joint-affections of nervous origin and those due to rheumatic inflammation is generally easy, if attention be paid to the limitation of the disease to the paralysed limbs, and to the concurrence of other trophic changes belonging to the same class. It must be especially noted that the joint-affection is by no means unfrequently associated with rapid muscular atrophy, and that there is ample reason for believing that it is due to irritation of the same nerves and the same part of the cord whose iritation causes the muscular lesion.

iii. *Skin.*—Various inflammatory and other nutritive changes in the skin and subjacent tissues have been traced to affections of the nerves and nervous centres. It has long been recognised: that, after division of the branches of the fifth pair distributed to the eye and conjunctiva, inflammation, leading to more or less rapid destruction of these parts, is apt to occur; and that in cases of paraplegia attended

with total abolition of sensation, inflammation, ulceration, and gangrene are extremely liable to supervene in the paralysed parts. It has been proved, however, by experiment on the lower animals, and by the results of careful attention to patients suffering from such lesions, that these inflammatory changes are not usually due to the withdrawal of any direct conservative influence which the healthy sensory nerves exercise over the parts to which they are distributed, but to the fact that the loss of sensation prevents the sufferers from recognising the presence of mechanical irritants or other injurious influences and so from avoiding or counteracting their operation. Various affections, however, more or less closely related to these in their characters, are undoubtedly referrible to the direct operation of irritative affections of the sensory nerves, cord, or brain. The most important of them may be ranged under the heads of erythema, vesicular eruptions, bullous eruptions, atrophic changes, and gangrene.

Cases are not unfrequently met with in which mechanical or other injuries to sensory nerves, not necessarily attended with anæsthesia, but often, as might be supposed, with neuralgia, are followed by erythematous redness limited to the areæ of distribution of the nerves, which redness may proceed to vesication, pustulation, ulceration, or gangrene. Such results have been observed in cases of tumour or inflammation involving the fifth nerve, and also in cases of injury or disease of certain of the sensory or mixed nerves of the arm or leg. One of the most interesting examples is furnished by herpes or zona, limited to the distribution of a single sensory nerve. Another example of great interest is afforded, as Charcot has pointed out, by anæsthetic leprosy—one of the special features of which affection is the excessive development of cellular elements in the course of the nerves, between the nerve-tubules. This overgrowth leads to the gradual destruction of the nerves and consequently to both anæsthesia and motor paralysis; but it leads also to atrophic changes in the muscles, and (what specially concerns us now) to erythematous patches on the skin, on which vesicles or bullæ become developed or which undergo atrophic changes, and in some cases to gangrenous destruction of the skin and subjacent soft tissues, and even of the bones.

A peculiar atrophic alteration of the skin following injuries of the nerves of the extremities has been described under the name of 'glossy skin,' by Mr. Paget and Dr. S. Weir Mitchell. In well-marked cases the skin becomes smooth, hairless, almost devoid of wrinkles, glossy, pink or ruddy, or blotched as if with permanent chilblains. The nails, too, become fissured and have a tendency to separate from their matrices. The skin thus altered is frequently the seat of an eruption having a close resemblance to eczema, and of a form of neuralgia which varies from the most trivial sense of heat to agonising pain. The skin, moreover, becomes extremely tender.

Trophic affections of the skin are equally apt to take place in con-

nection with lesions of the cord. In locomotor ataxy, for example, according to Charcot, eruptions are occasionally developed, more especially during periods of exacerbation of the disease, and in connection with the occurrence of neuralgic pains; and he points out that the eruption is not unfrequently limited to the parts to which the suffering nerve is distributed. Among special forms of skin disease thus arising he mentions lichen, urticaria, zona, and ecthyma or impetigo; and we may add to the list erythema nodosum of unusual distribution. In this case Charcot attributes the cutaneous affection, as he does the pain, to the involvement, in the disease of the posterior columns, of the nerve-fibres passing through the outer part of these columns, previous to their emergence from the cord. Again, erythema in patches—which may go on to the development of vesicles (herpes), and thence to the formation of large bullæ (pemphigus)—is not uncommonly a consequence of that affection of the membranes of the cord termed 'pachymeningitis,' which during its progress compresses and irritates the cord itself and the roots of the sensory nerves. A similar general development of bullæ is sometimes met with in vertebral caries. Erythematous, vesicular, or pustular eruptions are occasionally developed in hemiplegic patients upon the paralysed side of the body.

The most important, and on that account the most interesting, of the cutaneous lesions consequent on paralysis are the patches of gangrene which are commonly known as 'bed-sores.' These are apt, of course, to form in many patients, whether paralytic or not, who are confined to bed, partly from the constant pressure to which prominent parts are under such circumstances exposed, partly from the effects of the patients' secretions, which accumulate beneath them, and in no small degree from the neglect of nurses. But there are certain paralytic cases in which bed-sores form with remarkable rapidity—in the course of two or three days, it may be, from the commencement of the illness—and this without exposure to undue pressure, or to the irritation of urine or fæces, and in spite of the most watchful care on the part of the attendants; moreover, the bed-sores appear on the paralysed part, and on that alone, even if the unparalysed parts have been specially exposed to pressure. The bed-sores here referred to commence as patches of erythema, with more or less inflammatory infiltration and congestion of the subjacent tissues, sometimes including the muscles and the bones. In a short time vesicles or bullæ appear upon them, and superficial sloughs form. These gradually extend in surface and depth, and may thus eventually occupy a wide area, and involve muscles, bones, and even implicate subjacent cavities. Such bed-sores may occur either in hemiplegia or in paraplegia, and on any part of the paralysed surface, but they arise more especially on such parts as are exposed to pressure. In hemiplegia they rarely appear except about the centre of the buttock. In paraplegia they

specially involve the sacral region, and are hence situated on a higher level than those occurring in hemiplegia, and occupy a more central and symmetrical site. Moreover, in paraplegia they are apt to appear also on the heels, inside the knees, and upon the hips. The form of hemiplegia which acute bed-sores tend specially to complicate is that due to hemorrhage. The spinal affections in which they more particularly occur are those in which inflammation or hemorrhage involves a pretty considerable extent of the central portion of the cord. They may hence follow fractures and other injuries of the spine and exacerbations or acute complications of chronic diseases. The formation of these acute bed-sores must always be regarded as a phenomenon of serious import; but occasionally, of course, the morbid process becomes arrested, and convalescence may supervene.

The special seat in the cord of the lesions on which the various skin affections which have been enumerated depend has not been so accurately determined as the seat of those lesions which evoke affections of the muscles and joints. There are good grounds, however, for the conclusion that the posterior cornua and central regions of the grey matter have the same trophic relation to the skin as the anterior cornua to the muscles.

iv. *Viscera.*—The visceral affections of chief importance referrible to spinal lesions are inflammations of the bladder and kidneys, with the discharge of purulent, bloody, ammoniacal, and fetid urine. In most cases of paraplegia these conditions are apt to supervene after a time in consequence of the constant retention of urine in the bladder, and the irritation to the mucous surface which results from its accumulation and decomposition. But there are certain cases of paraplegia in which the occurrence of these phenomena is as early as that of bed-sores, and in which, indeed, they take place simultaneously. The change in the quality of the urine and the inflammation of the kidneys and urinary passages cannot then be explained by simple paralytic retention or by spontaneous decomposition of the urine; and there is consequently little doubt that they also are referrible to the direct influence of the diseased spinal cord.

Recapitulation.—It may be convenient, by way of summary, to remark: first, that the pathological influence of the spinal nerves, of the spinal cord, and more remotely of the brain, upon the production of morbid changes in the muscles, bones, skin, and viscera is associated only with those lesions which are irritative in their effects or inflammatory; second, that the lesions which immediately determine muscular and arthritic mischief are situated either in the course of the motor nerves or in their nuclei of origin; third, that the lesions which immediately determine cutaneous, and probably also visceral, inflammations involve either sensory nerves or the grey matter of the cord posterior to the central canal, or the immediately adjoining portions of the posterior columns of the cord, or possibly the ganglia at the

base of the brain; and, fourth, that although the variously situated spinal lesions and their respective pathological consequences have been separately considered, they are necessarily not unfrequently associated.

6. *Ascending, Descending, and Collateral Lesions.*

It is a fact of considerable importance, and one to which we have already more than once adverted, that circumscribed lesions of the brain, cord, and nerves tend to the production of degenerative changes either in the nervous tissue above them or in that below them, or in both; and, further, that in the case of the brain or cord there may be horizontal extension. Thus apoplectic or other destruction of some portion of one of the cerebral hemispheres, and still more certainly similar destruction of the corresponding corpus striatum leads to the occurrence of degeneration, which, commencing at the seat of disease, gradually extends downwards in a band-like form along the motor tract, first involving the corresponding crus and anterior pyramid, and thence passing to the opposite side of the cord and downwards mainly along the posterior part of the lateral white column. Again, when lesions occupy the lower part of the cord, they are apt to induce degenerative changes which gradually ascend in the posterior columns of the cord, and more especially in those parts of them which lie immediately on either side of the posterior median fissure. Further, lesions of intermediate regions of the cord are liable to be followed by both ascending and descending degenerations; the former, as in the last case, limited to the posterior columns; the latter, as in the first case, to the lateral columns. Occasionally also foci of disease involve secondary changes which extend from them in the horizontal direction; and similar extension would seem now and then to start from either the ascending or descending secondary lesions, so as to involve more and more of the thickness of the cord, and especially the anterior cornua and their motor nuclei.

When the anterior root of a spinal nerve is divided, its peripheral portion (at any rate its white substance) undergoes degeneration; while, if the posterior root be divided, all the peripheral part—that which still retains its connection with the ganglion—remains healthy, while that which enters the cord degenerates. It may, however, be observed that, consecutively to amputation of limbs, the large cells of the anterior cornua in relation with them have, after a considerable time, been found atrophic.

It is obvious that the various secondary changes above described, and others which occur but do not as yet admit of being referred to any general rule, must necessarily, in many cases, induce special clinical phenomena, complicating more or less seriously those due to the primary lesion.

7. *Central and Reflex Consequences of Lesions of the Nerves.*

Not only are central lesions efficacious in the production of peripheral lesions, but lesions of sensory or centripetal nerves are capable of inducing central mischief, or by reflex action mischief in the area of distribution of centrifugal nerves. It is thus apparently: that injury to sensory nerves induces that irritable condition of the spinal cord which forms the pathological basis of tetanus; that intestinal irritation and the irritation of dentition cause convulsions in young children; that certain uterine or ovarian conditions are instrumental in the production of the various psychical and motor phenomena which characterise hysteria; that renal affections occasionally lead to paraplegia; and that injury to the frontal branch of the fifth nerve is apt to cause amaurosis. Brown-Séquard, who has devoted great attention to this subject, attributes indeed to reflex influence almost all the consequences which are also caused by affections of the nervous centres; among others, various forms of paralysis, anæsthesia, deafness, loss of taste and smell, convulsions, delirium, and coma, together with cutaneous eruptions and wasting of muscles. It is perhaps needless to add that he, as well as others, attributes many internal inflammations to the influence of irritation acting reflectorially.

8. *Headache.*

Headache is a frequent attendant upon cerebral lesions, but it is still more commonly due to affections of remote organs, such as the stomach, or to neuralgic, rheumatic or other such conditions. In whatever cause the pain originates, or in whatever part of the head the cause operates, it seems pretty obvious that the pain must be referred to the peripheral distribution of the sensory nerves—that is, of those sensory nerves which have their apparent origin at the base of the encephalon, and which are distributed to the integuments, bones, and membranes of the brain. Thus, some headaches are limited to one half of the forehead and probably to the corresponding eye—to the area of distribution of the first branch of the fifth—some occupy both sides of the forehead, some affect the vertex, some the occipital region. Other headaches, again, appear to occupy the temples, and shoot from one side to the other, others are confined to the neighbourhood of the ear, and others seem to be generally diffused. Headaches vary in character, and are variously described. Sometimes they are shooting, sometimes aching, sometimes throbbing, sometimes likened to a weight upon the top of the head, sometimes to a sense of constriction. They are not unfrequently associated with intolerance of light and sound, visual spectra, tinnitus aurium, vertigo, nausea and sickness, drowsiness or wakefulness, and sometimes (even if the affection be superficial

and wholly independent of brain disease) with more or less delirium. It is in most cases exceedingly difficult to refer headache to its proper cause, unless our diagnosis be aided by the presence of distinctive associated phenomena. Thus pain, almost accurately resembling in all its characteristics that of megrim, may be induced by the simple pressure of an unyielding hat upon the frontal branches of the fifth pair, and immediately cured by the removal of that pressure; rheumatic neuralgia of the forehead, from simple exposure to a blast of cold air, is not unfrequently attended with a peculiar sense of drowsiness; and in either case the pain may be so intense and so distracting as to lead the patient or his doctor to suspect serious disease of the internal parts. Dr. Hughlings Jackson remarks that frontal headache is generally referrible to abdominal affections, headache at the vertex to cerebral disturbance, and occipital pains to disorders of the circulation, and more especially to anæmia. However that may be, it is certain that pain due to cerebral disease may, especially in the case of cerebral tumours, be referred to all parts of the head, and that it may exactly simulate those which are of less serious origin. It may be slight or intense, continuous or paroxysmal, and may be attended with tenderness of the scalp, or with various of the symptoms which have already been referred to as frequent accompaniments of headache. When the pain is intense, and especially if it be paroxysmal, it frequently causes the patient to scream out, and to support his head with his hands. The most intense pain, which is then usually very limited as to its seat, is induced by the pressure of intra-cranial tumours or abscesses upon sensory nerves.

9. *Vertigo.*

Vertigo, or swimming in the head, is that condition in which a person suffers from a sense of failing equilibrium, of falling or of rotating, and in which not unfrequently surrounding objects appear to swim or oscillate before his eyes. It has been attempted to distinguish between that form of vertigo in which the patient refers the vertiginous phenomena to his own person, and that in which he refers them to surrounding objects. The distinction is, however, obviously inadmissible. Vertigo may vary from a mere uncomfortable sense of oscillation, such as one feels on landing after a sea-voyage, to a condition in which the patient is quite unable to maintain his equilibrium, and either falls to the ground, or is compelled to support himself by clutching some fixed object. It may be momentary or of long duration, and in the latter case is commonly attended with exacerbations. It is generally more pronounced when the patient is standing or walking, but may come on while he is lying down, and even has his eyes shut. The proximate cause of vertigo is probably multiform. Vertigo is often, and probably accurately, referred to variation or disturbance

of the cerebral circulation; in proof of which view it may be observed that it not unfrequently attends syncope, anæmia, and loss of blood, on the one hand, and cerebral congestion and inflammation on the other. It is not uncommonly the consequence of an unhealthy condition of the blood, or of the presence in it of poisonous matters: as appears from its frequent occurrence in inflammatory diseases and in the specific fevers. It attends epilepsy, eclampsia, and organic lesions, such as effusions of blood and tumours. It is frequently of eccentric origin, referrible, for example, to dyspepsia or other functional derangements of the stomach. Physiological experiments have, as we have already observed, demonstrated that the cerebellum is the centre of equilibration, and that injury or irritation of this organ or of the parts immediately connected with it, such as the pons Varolii, corpora quadrigemina, or crura cerebri, in the lower animals, is followed by vertiginous movements. It is probable, therefore, that in the human being vertigo is referrible in large proportion to functional disturbances and organic lesions of the same parts. It is certain that affections of the cerebellum are generally if not always attended with failure of the power to maintain equilibrium. Affections of the eye and ear, and even of the spinal nervous system, are also capable of causing vertiginous phenomena. In the case of the eye, vertigo depends, for the most part, on affections involving the muscles, such as loss of power in one or more of the recti of one eye, in consequence of which a convergent or divergent squint is produced; or on the presence of nystagmus. Vertigo referrible to the ears is usually associated with deafness, and immediately due, according to Menière's researches, to disease of the semicircular canals, injury to which has been shown by experiment on the lower animals to be followed, equally with injury to the crura cerebri, by vertiginous movements. In reference to affections involving the spinal nerves, it may be observed that the inco-ordinate movements of locomotor ataxy, and the oscillating movements of disseminated sclerosis and of paralysis agitans, are not unfrequently attended with the subjective phenomena of vertigo, especially if their influence be not counteracted or neutralised by sight or hearing. Vertigo is frequently associated with headache, functional disturbance of the eyes and ears, sickness, and other phenomena. The recognition of the exact cause of vertigo in any case must be based less upon the simple vertiginous phenomena than on the accompanying symptoms.

10. *Impairment or Loss of Power of Speech.* (*Aphasia. Aphemia. Amnesia.*)

We employ the above terms in their widest sense, and as including, therefore, not merely defects referrible to, or manifesting themselves in, the organs of articulation, but defects relating to reading and writing. Paralytic affections of articulate speech may be divided into four

classes:—first, that in which the motor nerves of the organs of speech are paralysed in a greater or less degree, and where, therefore, the defect of speech is simply the result of inability to use these organs; second, the class in which the co-ordinating centre of the movements of articulation is affected, and where the patient, having complete control over the movements of his lips and tongue for other purposes, is yet unable to utter articulate sounds; third, the class in which the impairment of speech is central, where there is loss of memory of words, or *amnesia*, and other losses of mental attributes; and, fourth, a complex class, including all those cases in which the conditions characteristic of the second and third classes are combined.

a. The *first class* comprises a well-defined but rather wide range of cases, among which may be enumerated those of right or left hemiplegia, general paralysis of the insane, general spinal paralysis, locomotor ataxy, disseminated sclerosis, chorea, glosso-laryngeal palsy, and lesion of one or more of the motor nerves of the organs of speech. In left hemiplegia and in paralysis of one portio dura or hypoglossal, the defect of speech is often scarcely appreciable, and rarely amounts to more than a little thickness of utterance. In general paralysis there is usually a little tremulousness of the tongue and lips when the attempt to speak is made, a little hesitation and thickness or inexactness of utterance, which become especially marked when the patient speaks with vehemence. In locomotor ataxy and disseminated sclerosis the defect of speech may present some degree of variety; in some cases there is more or less slowness and tremulousness; in others the slowness is attended with exaggerated efforts on the part of the lips and tongue to effect their purpose; in others the syllables are unnaturally divided, and there is a tendency, as it were, to scan the sentences; but in all, even though separate letters may be accurately pronunciated, the more complex their combinations in words the more clumsy and inexact does their pronunciation become, and the latter parts of long sentences, or of a sustained conversation, always contrast unfavourably in these respects with the beginning. In glosso-laryngeal paralysis, the early stages of defective articulation resemble those observed in general paralysis, but gradually the lips, tongue, and soft palate lose almost entirely their capacity for movement, and the patient loses not only the power of articulation, but that of retaining the saliva in his mouth, and that of swallowing. The character of the defective articulation of chorea need not now detain us.

b. The *second class* of cases corresponds to the group to which Dr. Bastian endeavours to limit the use of the word *aphemia*, adopting the word from Broca, who, however, has employed it in a different and far wider sense. Typical cases of this kind are very rare. In them, patients recovering from an attack of unconsciousness are found to be entirely speechless, and to remain speechless for days, weeks, or even months, notwithstanding that they may have regained the use of every

other faculty which might be supposed to have any, the remotest, connection with speech: that is to say, notwithstanding that they can hear, understand everything that is said to them, read, converse by means of writing, and use the lips and tongue with the utmost precision for every purpose excepting speech. Now in such cases as these it is obvious that the patient retains all his mental faculties, and that he thinks (as is probably usual) with the aid of words, which he still retains the power of expressing by means of writing; but which he cannot utter, not because he has lost the use of his muscles of articulation, but because the wish to speak does not evoke the combined automatic movements on which speech depends. In ordinary conversation the words which express our thoughts flow automatically from our lips; the complicated combinations of movements on which their utterance depends are executed momentarily and with the utmost precision, without any attention whatever being, as a rule, bestowed upon the movements themselves.

Looking to the extreme complexity of these movements, it seems certain that that part of the brain in which words are transformed into ideas, and are revived in thought, acts, in the process of transforming them again into articulate speech, upon the centres of origin of the various nerves of speech, through the intermediate agency of a special co-ordinating centre. This centre is probably situated somewhere in or below the corpus striatum; and within it, on the receipt of the message from above, the various telegraphic communications with the nerve-origins below are automatically so manipulated as to cause, through these latter, the organs of speech to execute the necessary combined movements. Words are practically innumerable. The elementary articulate sounds, however, which by their combinations produce articulate language, are probably less than fifty in number, and this comparatively small number therefore also represents all the groups of simultaneous combined movements which the tongue and lips can be called upon to execute. It seems probable, partly on these grounds, partly from the consideration that language (apart from the mere mechanism by which it is uttered) is a mental function, and partly from the consideration that the function of a co-ordinating motor centre is to regulate or combine groups of movements, that the duty of the assumed co-ordinating centre of speech must simply be to preside over that essential but comparatively subordinate department of speech which consists in the production of the elementary articulate sounds. If this view be correct, it is easy to understand how some lesion involving this centre or cutting off the direct communication either between it and the intellectual centre of language above, or between it and the nerve-nuclei below, might result in dumbness, while at the same time the command of language might in all other respects be retained perfectly, and the power of executing the most delicate movements with the lips and tongue remain intact. It is easy also to understand how,

in such cases as this (considering that all articulate sounds are merely the results of certain mechanical arrangements of the speech-organs) the patient who has lost the power of speech might be taught to copy these mechanical arrangements, and thus again to speak, exactly as deaf mutes are taught. The morbid anatomy of this class of cases has not been yet investigated. The patients in whom aphemia has been observed have had fits, epileptic or apoplectic, from which they have recovered with or without temporary paralysis.

v. In the *third class* of cases there is *amnesia*, or loss of memory of words. In typical examples of this kind the patient, with perfect power of utterance, is yet incapable, from want of words, of joining in conversation; with perfect vision he is unable to read even to himself; and with (it may be) entire command over his arm and hand, he cannot make himself understood by writing, or even write. In most if not in all of these cases, however, there is not merely forgetfulness of words, but there is more or less inability to recall facts, to concentrate the thoughts, and to pursue any train of reasoning. An amnesic patient, when he attempts to speak, commences perhaps with one or two words correctly uttered, then hesitates for a word, probably uses a wrong one, notices that he is wrong, tries to correct himself, perhaps repeats the words that he first uttered, stumbles a little, and then, with a gesture of annoyance, comes to a stop. If his speech be carefully observed, it will generally be noticed that his vocabulary is limited to a very few words, and that he tends to repeat certain of these, and especially to repeat certain combinations of them; and indeed he often appears to recall phrases more readily than single words. If asked to name even the most common things he fails in very large proportion, and fails probably to remember words which he has been taught to utter only a minute or two previously. Yet he seems to understand everything that is said to him; he at once distinguishes the right name from the wrong when submitted to the test; and he can articulate readily every word which is dictated to him. It is very interesting to note that uttered words entering by the ear are by a voluntary effort at once and perfectly reproduced by the organs of speech, and at the same time recall for the moment to his mind the ideas which properly attach to them. Such a patient may often be seen with a newspaper or book, over which he pores as if he derived the greatest interest from its perusal; but on asking him to read aloud he will probably indicate his inability to do so, and not even make the attempt; or possibly he may pick out a word here and there which he recognises, and which he pronounces with more or less approach to accuracy. It might be supposed that, although he cannot translate written into vocalised words, yet that written words convey to his mind through the eye their proper meaning, and that hence he really understands what he reads. This, however, is generally not the case, for if he be examined by leading questions he fails to show that he has any know-

ledge of what he seems to have been reading about. He will, however, not unfrequently point out here and there words, or even phrases, which he recognises and perhaps utters. He seems, indeed, much in the condition of a child poring over the pages of a book written in a foreign language, which he has just begun to learn. If now asked to name letters he probably fails, just as he previously failed with words; and, again, if asked to point out letters as they are named to him, his inability is equally marked. In fact, just as he has forgotten the names of things he has forgotten the names of letters, and consequently their value; and he fails, partly on this account and partly from the complexity of the mental process which it involves, to attach any sound or any meaning to the various combinations of letters which stand for words. When he recognises printed words, it is probably as a whole that he generally recognises them: thus, he will sometimes point out his own name, though unable to point to, or designate, a single letter that it contains.

A similar difficulty exists in regard to writing. If his hand and arm be not paralysed, or only slightly thus affected, he can execute all accustomed delicate movements with them, and indeed can employ the hand as a mere machine just as well, probably, as ever he did. If he could draw, he can probably still draw, and he can copy the forms of geometrical figures, and therefore the forms of letters. He can write and print from a copy. If, however, he tries to write (and he is not unfrequently fond of writing) he either makes a series of unmeaning up and down strokes, manifesting even here a dim recollection of the art of writing, or he begins a word, perhaps his own name, correctly, and after writing a letter or two repeats them and then stops, or passes on into unmeaning strokes. If words are dictated to him he writes them even more incorrectly than those which he writes voluntarily, and probably writes letter-characters which are dictated to him as faultily as words. Yet not unfrequently, if he be set to copy from a printed page, he will translate the printed words (letter by letter) into their written equivalents as well and as quickly as if he were in perfect mental health, and this without being able to name or to understand the printed words and letters, or those which he himself forms. It is curious to observe here the correspondence that exists between the eye and the hand: the patient sees the printed word, and by an effort of the will reproduces it automatically in written characters, yet neither the word he sees, nor its written equivalent, nor the act of writing it, brings to his mind, even for an instant, any glimpse of its meaning. An amnesic patient who is unable to write from dictation will often put down figures from dictation, and, further, perform simple arithmetical sums upon a slate with tolerable correctness. He may even perform sums in addition of money; and very curiously he will sometimes, while adding up, miscall the figures which he writes down correctly.

Now the degree in which any one or all of the above peculiarities may be present in any case varies of course within very wide limits; and so also does the degree in which the patient's memory of facts and power of concentrating his thoughts, and of reasoning, are retained. But the deficiency of his mental powers is not to be measured by the degree of his loss of memory of words. Many of those patients in whom the amnesic condition is extreme take such a lively interest in all that is going on around them, play at simple games of skill so cleverly, are so quick in their movements and in the use of their senses, and display such quickness of perception, that they obviously possess considerable intelligence. We are apt indeed to give them credit for much more intelligence than they really possess. It seems probable that, in proportion to their inability to recall facts and words by voluntary effort, they live more and more, as it were, in the objects which present themselves to their senses, and in the evanescent ideas which they evoke.

But many amnesic patients present peculiarities which do not quite accord with the above description—these peculiarities being due either to the degree in which the patient is affected or to the fact that other forms of sensory, motor, or mental derangement are superadded. In some cases the aphasic condition is revealed only by the occasional misuse of certain words, or by the omission of certain words or letters in speaking or writing, or by the occasional employment of wrong endings or beginnings to words, or by the transposition of syllables or letters, or by the use for the word intended of some other word having a phonetical relation with it, or some analogy to it either in its meaning, its appearance, or the ideas it evokes; in other cases the patient's vocabulary is limited to one or two sounds or words, such as 'yes' or 'no,' or to a phrase or two, such as 'damn it,' or 'can't afford it,' which he utters whenever he makes the attempt to speak, and sometimes without appearing to recognise that his language is in any degree peculiar. In other cases the patient does little more than repeat words which are dictated to him, and these he repeats over and over again until a newly dictated word displaces the former one from his memory. In other cases, again, he makes inarticulate sounds, which he utters volubly and with emphasis, and which, if carefully attended to, seem divided into lengths. These sounds, indeed, may have some obvious phonetic relation with words, and as the patient's condition improves become resolved into articulate speech. It may be added that amnesic patients not unfrequently utter an unexpected oath or phrase under the influence of emotional excitement, just as paralysed patients under similar circumstances are apt to move limbs over which they have no voluntary control; and, further, that amnesic patients who have but few words at their command, when asked to repeat from dictation things that they have learnt, such as 'the Lord's Prayer,' the numerals, or the alphabet, will often, instead of repeating the word or sentence

actually dictated, utter the word or sentence which immediately follows, and possibly continue their recitation until they become confused and mumble unintelligibly, or repeat themselves. It is this third group of cases, together with the fourth group (to be considered presently) to which the investigations of M. Broca and others chiefly relate. It is in these cases that there is almost invariably right hemiplegia dependent on some lesion of the left cerebral hemisphere, occupying, roughly speaking, the district which the left middle cerebral artery supplies, namely, the corpus striatum and the wedge of nervous substance extending outwards, towards and including in its base the island of Reil with some of the neighbouring convolutions—more precisely (according to M. Broca) the posterior third of the third frontal convolution. It is this district in which the effects of cerebral embolism are most frequent.

d. The *fourth group* of cases includes all those in which amnesia is associated with aphemia, or with both aphemia and paralysis of the organs of speech. These cases are very numerous, and present great varieties of symptoms, according to the degree in which each of the above-mentioned conditions is present, absolutely or relatively. In typical cases of this class the patient, after an attack of right hemiplegia, loses absolutely the power of speech, or at most utters some one or two inarticulate sounds, and perhaps has some difficulty in using the tongue and lips; but he apparently understands everything that is said to him, and when asked to point out words and letters on a printed page, probably points them out correctly. So far the symptoms are those of aphemia. But presently the patient gradually or suddenly recovers the power of articulate utterance, and it is then found that he is suffering from amnesia in addition to aphemia, that he has in a greater or less degree forgotten the names of things, perhaps his own name.

In conclusion it may be suggested that it seems convenient still to employ the word 'aphasia' in that general sense in which it has been used by Trousseau, as inclusive of all difficulties of speech which come under the second, third, or fourth of the above groups; and that, inasmuch as the aphasic condition thus defined includes two perfectly distinct clinical phenomena, which, though often combined, may exist separately, it seems also convenient to have a distinct name indicative of each of them, and applicable to those cases in which one or other of them occurs separately. The terms 'aphemia' and 'amnesia' may be thus employed.

We may here call attention to the facts: that articulation and phonation are distinct elements in spoken language; than phonation in some degree survives in all cases of aphasia, and that not unfrequently aphasic patients who can utter only one or two words can yet hum tunes with facility; and that loss or impairment of phonation is usually the result of disease involving directly the nerves of the intrinsic

muscles of the larynx, or of hysterical and other such functional disturbances.

11. *Mental and Emotional Disturbances.*

It may be pointed out in conclusion that all forms of mental disorders are apt to attend not only brain-diseases, but a large number of affections in which the brain is only secondarily or remotely implicated. This subject is much too vast to admit of separate discussion here. It may, however, be observed that patients may suffer in feeling, intelligence, and will, either conjointly or separately, and that these may be exalted, perverted, or impaired. Thus, as regards feeling, he may be excited (angry, boisterous, merry), depressed (melancholy, anxious, fearful), or suspicious, mischievous, or sullen; as regards intelligence, his ideas may flow rapidly and with vivacity, he may have delusions, his reasoning powers may be perverted or impaired, his memory may fail, or there may be incoherence or general mental imbecility; and, as regards will, he may show abnormal obstinacy or tenacity of purpose, extreme vacillation, or utter listlessness and apathy, or incapacity for exertion. The various forms of delirium—the low muttering, the busy or garrulous, and the maniacal—are all common in different forms of disease. Insanity in all its varieties is apt to attend or supervene upon a large number of acute or chronic disorders, whether these affect the brain or other parts. And, lastly, coma—the abeyance of all mental phenomena, the condition in which the patient lies as in a profound sleep and insensible to every external influence—if not the primary disorder, constitutes the common fatal termination of most of the other mental affections which have been enumerated.

C. *Electricity in Nervous Diseases.*

The employment of electricity is so important not only in the treatment of nervous diseases but for the purposes of diagnosis, that a few remarks in reference to its mode of application and uses will not be out of place.

Two forms of electricity are employed in medicine; one the *continuous* or *galvanic* current, the other the *induced* or *faradic* current. The former is the kind of electricity which is developed by chemical decomposition, and is usually obtained from one or more similar cells, arranged in a series or circuit. For medical purposes a battery of from twenty to fifty cells is usually employed. The latter is the kind of electricity furnished by magneto-electric and other induction machines. The galvanic current is characterised: first, by constantly flowing in one direction, namely, from the positive pole or rheophore to the negative pole; second by its comparatively low intensity, and

considerable quantity—its intensity, however, depending on the number of cells employed, its quantity on the size of the elements; third, by its possession of powerful chemical and thermal properties which are specially observable at the point of application of the negative rheophore: and fourth, by its comparatively little influence in causing muscular contraction. The faradic current is of instantaneous duration, occurs only at the moment of making or breaking contact, and takes place alternately in both directions. It is remarkable for its high intensity, and for the powerful effect it has in causing contraction of muscles, and in acting on motor and sensory nerves. But it has no thermal or chemical influence whatever.

The rheophores, or instruments by means of which electricity is applied, are of various kinds; but they should always be furnished with insulating handles, so that they may be freely and safely manipulated by the operator. The larger ones generally consist of a sponge fixed in a metallic cup; the smaller ones are usually an ovoid metallic knob covered with wash-leather.

If the electricity is to be limited in its action to the skin the rheophores must be used dry, and it is well even to dust the skin with a little dry powder; but if, as is usually the case, muscles or nerves are to be acted on, they should be well moistened with hot salt and water, as also should the surfaces to which they are applied.

There are three different ways in which galvanism or faradism may be used to influence muscles. First, superficial muscles may be acted upon individually by placing the rheophores immediately over the belly of each muscle which it is desired to affect. In this case the rheophores should always be situated within a short distance of one another. Second, one rheophore may be applied over the trunk-nerve leading to a group of muscles, and the other successively over the different muscles supplied by this nerve, each of which will thus in turn be caused to contract. Third, one rheophore may be fixed on some indifferent part of the surface, as for example on the nape of the neck, while the other is placed over the nerve leading to the individual muscle or group of muscles which it is intended to influence. By this method, which is sometimes termed the indirect method, many deep muscles which could not otherwise be reached may readily be made to contract. It need scarcely be said that the successful employment of this method requires accurate knowledge of the situations at which muscular nerves are most accessible. In this case the first rheophore should be of large size, but the second or that to be applied to the nerves should be small and ovoid.

The same methods of procedure may be employed for galvanising or faradising nerves.

In *employing electricity for diagnostic purposes* it is important:—first, that if possible corresponding healthy and diseased parts in the same individual should be compared; second, that the patient should

be as far as practicable at absolute rest, and especially that the parts to be examined should be placed under exactly similar conditions; third, that the rheophores should be the same and at equal distances apart, and the strength of the current identical, in each parallel series of observations; and fourth, that as a general rule, in testing the diseased side, the feeblest current capable of affecting the healthy side should be employed.

Faradism has, as has been pointed out, a very powerful influence in causing contraction of healthy muscles. Galvanism in the same case causes contraction only at the moment of breaking circuit. Its peculiar properties are best manifested when the interruptions are slow. The effects of electricity on paralysed muscles are variable. In hemiplegia there is no necessary change in this (most; but in the early period, and at times when inflammation or other degeneration is present, there is often some increase of re-action to both kinds of stimulus; while later, sometimes from mere disuse, sometimes in connection with secondary degenerative nervous lesions, this undergoes diminution. In paraplegia the electrical phenomena are much the same and obey the same rules, but here both faradic and galvanic irritability are in the early stage of the disease more commonly and more markedly increased than in hemiplegia. In destructive lesions of motor nerves or of their nuclei of origin the consequences which ensue are remarkable and for the most part distinctive. At first the reactions of the paralysed muscles both to faradism and to galvanism are normal. Then both varieties of irritability may undergo some degree of diminution. At the end, however, of a week or from this to the fourteenth day, faradic contractility diminishes considerably, and in the course of a few weeks probably becomes wholly lost. But while faradic contractility is disappearing, galvanic contractility increases, and in the course of a short time the affected muscles probably react to a current half as strong as that which is required for producing the same effect on the healthy muscles. At a late period the response of the diseased muscles to galvanism in its turn diminishes and disappears.

As regards nerves it appears that for a few days after their division there is a slow increase of both galvanic and faradic irritability. But soon gradual decrease takes place; and finally all irritability ceases to both forms of electricity. The electric irritability of nerves once lost is very slow to return, and it is an interesting fact that the recovering nerve sometimes allows the passage of the mental stimulus while it is still wholly irresponsive to electricity. It is suggested by Erb that this phenomenon is dependent on thickening of the neurilemma. Onimus considers that the failure of paralysed muscles to respond to faradism while they still react to galvanism is due to changes having recurred in the intra-muscular nerves. It need scarcely be added that when motor nerves have lost their electric irritability, it is impossible to stimulate muscles by the indirect method.

Electric sensibility may be diminished or increased. In general, diminution attends ordinary anæsthesia, and increase accompanies hyperæsthesia. But it may be observed that variations of muscular and cutaneous sensibility are not always in relation with one another. Muscular sensibility is usually impaired when electric contractility is diminished, and augmented when the latter is excessive. Muscular sensibility is increased in cases of muscular rheumatism, and often diminished in hysterical paralysis even when the muscles contract strongly under the electric stimulus.

It may be added that the condition of things in hysteria presents much variety. But in hysterical paralysis, especially when the muscles have wasted, there is often considerable, but equal, loss of both faradic and galvanic irritability. This is frequently associated with diminution of cutaneous electric sensibility.

Therapeutical uses of electricity.—For a full consideration of this important subject we must refer the reader to special treatises.[1]

Meanwhile, however, we may direct attention to a few points in regard to it. Besides its caustic and cauterising effects, (for which galvanism is often special use to the surgeon, and as a counter-irritant) electricity possesses stimulant and sedative properties which are of great therapeutic value. Its stimulant properties are specially serviceable in the treatment of paralytic conditions; its sedative and anodyne properties are useful in assuaging various forms of spasms, but are chiefly valuable in the relief or cure of neuralgic affections.

Faradism is in general the more powerful stimulant of muscular and nervous tissues; and it is usually applicable to all paralytic or paretic cases in which any trace of faradic irritability remains, whether it be used for restoring parts which have lost their powers, or for preventing the wasting of disused or disabled muscles. The slowly interrupted galvanic current is also a stimulant, but in most cases a less powerful stimulant than the other. It possesses, however, in addition to the power of exciting muscular contractions, a special influence over the nutrition of the parts to which it is applied. It is better adapted than faradism for the treatment of muscles which have lost their faradic contractility, and it is often employed by preference in the treatment of those which, in addition to being paralysed, are wasted. Faradism and galvanism are alike useless in paralysed muscles which retain, or have reacquired, their normal electrical reactions.

For sedative purposes faradism is sometimes employed. But it only diminishes spasm or contractility by causing fatigue, and therefore, generally, is not best suited for this purpose. The most valuable sedative for muscular spasm is the continuous galvanic current. This is the only form of galvanism that should be used in the treatment of

[1] See especially Dr. Poore's 'Text Book of Electricity.'

pain. It is usually advisable to include the painful region between the rheophores in this case, and in neuralgia to apply the one pole over the spinal column above the point of origin of the affected nerve, and the other in turn to the several painful spots.

In the habitual use of faradism or galvanism for the above purposes, it is important: in the first place, to employ no greater strength of current than is absolutely necessary to effect the intended result—if to cause muscular contraction, the weakest current capable of causing contraction, if to relieve pain or spasm only such a current as produces a slight degree of tingling; in the second place, that each sitting should not, as a rule, be continued beyond five, ten, or at most, fifteen minutes; and in the third place, that during this period, every affected muscle or painful point should be brought successively under treatment, and that, in the case of the galvanic current, the negative pole should never be kept for any length of time on one spot.

II. INFLAMMATION OF THE CEREBRAL AND SPINAL DURA MATER. PACHYMENINGITIS.

Causation.—Inflammation of the dura mater is either traumatic, or the consequence of the extension of disease from parts external to it, or it is of idiopathic origin. With traumatic inflammation the physician has little to do. Inflammation from extension may be secondary to erysipelas or other such affections of the surface of the head, but is mostly traceable to caries of the petrous or mastoid portion of the temporal bone, or to similar disease of the frontal plate of the ethmoid or adjoining parts of the sphenoid or orbital parietes, or to syphilitic or other like affections of the bones of the skull, or to caries of the vertebræ, or to sacral bed-sores.

Morbid anatomy. 1. *Cerebral dura mater.*—When inflammation extends from the bones of the skull to the dura mater this membrane becomes thickened and softened, and its connection with the subjacent bone more or less loosened. Not unfrequently a false membrane forms upon its free aspect, and may cause it to adhere to the corresponding surface of the brain; or suppuration takes place which may either be limited by adhesions, or become widely diffused in the cavity of the arachnoid. Further, inflammatory overgrowth or actual suppuration often takes place between the skull and the dura mater, and in the latter case the dura mater is apt to get perforated, and the pus to be discharged into the cavity of the arachnoid. When inflammation occurs in the neighbourhood of the sinuses, these are liable to get involved and to become the seat of thrombosis or suppuration, or the source of pyæmia. This event is especially common when the

meningitis is due to disease of the temporal bone, in which case the lateral, petrosal, and cavernous sinuses may either or all of them suffer.

2. *Theca vertebralis.*—The inflammatory products which are developed during the progress of vertebral caries tend sooner or later to accumulate in the neighbouring part of the spinal canal, between the bones and dura mater. In the majority of cases, according to M. Michaud, this accumulation takes place in the first instance between the back of the bodies of the vertebræ and the vertebral ligament, which gradually undergoes erosion and perforation. The theca vertebralis then becomes involved in the inflammatory process, the outer surface of its anterior portion undergoing proliferation, and possibly forming a kind of caseous button, which, when the cord becomes compressed in this disease, constitutes for the most part the agent of compression. Inflammation of the dura mater, secondary to vertebral disease, may of course occur in any part of its length. In patients who are suffering from extensive bed-sores of the sacral region, it frequently happens that the sacral and coccygeal bones become exposed and eroded. In some of these cases the sacro-coccygeal ligament gets destroyed, and hence the inflammatory process extends into the vertebral canal, or, in consequence of perforation of the theca vertebralis, into the cavity of the arachnoid. Occasionally, consecutively either to vertebral disease, or to the condition last mentioned, or from some idiopathic cause, the theca vertebralis becomes inflamed throughout, or in great part of its extent, and suppuration takes place on either side of it. The pus which forms externally first accumulates in the spinal canal, and then (if the case be of sufficiently long duration) escapes with the nerves through the intervertebral foramina, and follows their primary ramifications: forming it may be a longitudinal series of abscesses behind on either side of the vertebral spines, and a similar series in front on either side of the bodies of the vertebræ, of which those in the abdomen possibly constitute multilocular psoas abscesses. The pus which is effused from its inner aspect distends the cavity of the spinal arachnoid, and may spread thence to the base of the brain.

Whenever the pus which is diffused throughout the arachnoidean cavity is derived from gangrenous sources, or from areæ of disease communicating with the external atmosphere, it is fetid, greenish in hue, and dirty-looking, and on post-mortem examination the surface of the brain or cord in relation with it is generally found stained to a greater or less depth by imbibition. This peculiarity is most frequently observed in meningitis due to perforation of the theca vertebralis by bed-sores, and in that which takes places consecutively to chronic ear-disease, and is occasionally met with in other varieties of caries of the skull or vertebræ.

3. *Pachymeningitis* is the name given to a peculiar form of chronic

inflammation of the dura mater. This may be the consequence, as are the varieties of meningitis just discussed, of injury or of subjacent disease. But it is more commonly of spontaneous origin. *In the head* it commences, for the most part in the area of distribution of the middle meningeal artery, with the formation over a greater or less extent of surface of a delicate adherent film, which consists partly of embryonic corpuscles, but mainly of large irregular thin-walled capillaries. Other similar films become developed in slow succession one upon the other over the diseased area, until the adventitious formation attains considerable thickness: the deeper-seated laminæ meanwhile becoming denser, more fibrous, and less vascular. Owing to the large size and extreme delicacy of the newly-formed blood-vessels, rupture, with extravasation of blood, is of frequent occurrence. For the most part the hemorrhages are minute and numerous, and result in the precipitation of crystalline and other forms of blood-pigment; not unfrequently, however, they are abundant, and form large accumulations between the laminæ, giving, it may be, to the whole growth the aspect of a mere clot. *Pachymeningitis of the theca vertebralis* usually takes place in the neighbourhood of the cervical enlargement of the cord. The dura mater becomes greatly thickened by the formation of a series of concentric fibroid laminæ, successively developed upon its inner aspect. All of them, even the most recent, are dense and tough, and little vascular or inclined to bleed, and thus differ from those occurring in the cerebral dura mater. In the progress of the disease the pia mater is apt to become involved, and sooner or later the cord gets compressed, and the nerves in their passage to the intervertebral foramina also implicated.

Symptoms and progress.—The symptoms which attend inflammation of the dura mater are necessarily vague, unless the inflammation be suppurative, or have extended to the pia mater, blood-vessels, or subjacent nervous matter, or involve the compression of the nervous centres, or of nerves. They are especially vague, if not trivial and misleading, in the earlier stages of the chronic forms of the disease. If suppuration take place, febrile disturbance with rigors is likely to ensue; and, as has often been observed, the fever is then apt to assume an irregularly remittent or even intermittent type, and thus the patient's illness may for a time have no little resemblance to an attack of ague. If the disease go on to the effusion of inflammatory products into the cavity of the arachnoid, or to the involvement of the pia mater or substance of the brain or cord, or of nerves, special symptoms referrible to these several parts will of course be developed. We proceed to consider in detail the symptoms of the different varieties of inflammation of the dura mater, the morbid anatomy of which we have already passed in review.

1. *Acute inflammation of the cerebral dura mater*, as met with in medical practice, is almost always due to chronic disease of the ear.

Recent otitis, however intense, is rarely followed by it. The patient, who may be of any age between early childhood and advanced senility, has suffered probably for years, perhaps nearly all his lifetime, from deafness, attended with more or less constant, more or less copious, and more or less offensive aural discharge, and occasional attacks of earache.

The supervention of meningeal mischief is induced sometimes by exposure to cold, sometimes by a blow on the affected side of the head or on the jaw, and not unfrequently seems to occur spontaneously. Very often it is preceded by or attended with sudden diminution or cessation of discharge. The patient is usually attacked with intense pain in the affected ear or its neighbourhood, or possibly with severe headache referrible to some other part of the head. This, which is generally more or less constant, is attended with exacerbations which are often so violent that he writhes and groans or grinds his teeth and even shrieks out. Not unfrequently it continues as long as the patient retains consciousness; but it often remits or disappears, and in some cases is wholly wanting from first to last. It is probably for the most part referrible to the disease of the ear rather than to that of the internal parts. Sometimes a paroxysm of convulsions is the earliest specific indication of meningeal mischief; sometimes vomiting; sometimes an attack of vertigo, incoherence, or rambling; sometimes a rigor. The disease is subject to remarkable variations both as to duration and as to the phenomena which attend it. Assuming it to prove fatal, the patient may die in the course of two or three days; more commonly he survives for two or three weeks; but his life may be prolonged for several months. In the last case especially intervals of apparent restoration to health probably occur; thus, in some instances, the patient has a convulsive attack, attended perhaps with vomiting, from which he recovers, and a second attack which proves the precursor of fatal symptoms does not take place for some days or weeks; in some instances he has strabismus and double vision, which may disappear from time to time, but are finally associated with graver phenomena; in some he suffers from a combination of symptoms threatening speedy dissolution, from which, nevertheless, he emerges, but only to become sooner or later the victim of a relapse.

The symptoms of the established disease comprise, in addition to headache localised in the ear or occupying the forehead, vertex, occiput, or other parts of the head, vertigo, intolerance of light and sound, hyperæsthesia, neuralgic pains in the head, neck, and limbs, nausea and vomiting, sleeplessness, restlessness and irritability, muttering busy or maniacal delirium, convulsions, local or general, occurring at rare intervals or following one another in rapid succession, paralysis limited to certain of the cerebral nerves or hemiplegic, drowsiness and coma, together with febrile symptoms. But these are not all necessarily present in the same case. Occasionally the patient, after suffer-

ing from severe pain in the ear, and possibly indefinite symptoms of brain-affection, falls into a state of collapse; sometimes he suffers mainly from convulsions, which are attended or succeeded by paralysis and coma; sometimes paralytic symptoms are the main feature of his malady—he becomes hemiplegic or has paralysis of some of the muscles of one of the eyeballs or of the portio dura, or he has difficulty in speech or deglutition, or he loses the sight of one or both eyes; sometimes he suffers mainly from mental derangement; sometimes he has frequent and severe rigors, coming on at more or less regular intervals, with other febrile symptoms, such as coated tongue, heat and dryness of skin alternating with perspirations, rapid pulse, and the like. In other cases the skin is cool, the pulse of normal rate, and there is total absence of febrile reaction.

The differences of symptoms which different cases present are no doubt in great measure attributable to differences in respect of the depth or superficial extent to which the inflammatory process extends within the skull. If the inflammation be limited to the dura mater, even should this become sloughy and pus accumulate between it and the bone, the symptoms are in a large number of cases undistinguishable from those due to otitis alone, and may be uncomplicated with fever. Again, if the inflammation reach the free surface of the dura mater, and especially if pus escape into the arachnoidean cavity, it is natural that aggravated brain-symptoms should be suddenly excited, that more or less fever should be developed, that some of the nerves at the base of the brain should become implicated, and that some of the symptoms of cerebro-spinal meningitis, such as retraction of the head and pain on moving it, should be experienced. Further, if abscesses form in the contiguous brain-substance, symptoms due to their presence are likely to arise. When inflammation of the dura mater involves thrombosis of the sinuses which are contained in its laminæ, escape of blood from contiguous parts of the brain may be impeded. The most interesting phenomena, however, are those which are referrible to the veins of the face and neck which are in continuity with the obstructed sinuses. Thus it sometimes happens that the veins in the eyelids and conjunctiva of the affected side get preternaturally distended with blood; or that inflammation of the internal jugular in the neck occurs, with formation of deep-seated abscesses in that situation. Optic neuritis, too, is often present. Lastly, pyæmia is not unfrequent; and, although rigors may be caused by local suppuration or by effusion of pus into the arachnoid, they are often an indication that the affection of the ear has become complicated with purulent infection.

It may be added: that the pulse is liable to great variations, that it may be accelerated, or of normal rate throughout, that it is sometimes preternaturally slow, and generally, when death approaches, becomes very rapid and feeble; that the skin is sometimes hot and dry, but often perspires profusely, especially towards the close, and

that generally during the course of the disease Trousseau's 'tache cérébrale' can be elicited; that the tongue differs in its character, is often natural, but tends to become coated, and with the approach of death dry and brown; that the evacuation of urine and fæces is often performed unconsciously; and that in the course of the disease symptoms are sometimes relieved by the sudden discharge of pus from the ear or even from the nose.

Death is usually due to collapse or to coma; it may, however, be caused by asphyxia, or be traceable to the effects of pyæmia.

2. *Pachymeningitis of the cerebral dura mater.*—The symptoms which attend this disease are exceedingly vague, and none the less so that it usually affects aged persons in a state of imbecility or dementia. It has also been observed, according to M. Lancereaux, in cases of chronic alcoholism and chronic pulmonary phthisis. The symptoms include pain in the head, vertigo, failure of the mental powers, and gradually increasing hemiplegia, with occasional epileptiform or apoplectic attacks, in one of which the patient probably dies.

3. *Acute general inflammation of the theca vertebralis*, such as results from its perforation by a bed-sore, or the extension of inflammation occasionally following fracture or caries of the spine, is sometimes attended with marked symptoms, but is often extremely obscure in its indications. The symptoms which may be looked for are: pain in the course of the spine, sometimes of an exceedingly intense character, and for the most part liable to aggravation by any movement, voluntary or involuntary, of the limbs or of the trunk or of the head and neck; more or less rigidity of the muscles, with perhaps twitching; and at the same time more or less loss of motor power, and probably of sensation, and of control over the bladder and rectum. To these, cerebral symptoms are apt to be superadded, more especially delirium, convulsions, and coma. Further, there may be tenderness in the course of the spine, due partly to the disease within it, partly, perchance, to the extension of suppuration into the muscles of the back. In cases of sufficiently long duration and sufficient intensity, it is possible that psoas abscesses may be discernible by palpation in the neighbourhood of Poupart's ligament. When the inflammation is due to the extension of bed-sores which have become developed during the progress of paralytic or other diseases, attended with impairment of the mental faculties, its presence is almost certain to be overlooked. Some degree of febrile disturbance will probably always be present.

4. *Caries of the vertebræ*, even when it is attended with considerable displacement, does not of itself usually cause paralysis. The paraplegia, indeed, which so commonly attends the disease, is almost invariably due to the extension of the inflammatory process to the membranes of the cord and to the cord itself, and to pressure caused by the accumulation of inflammatory products.

Among the early symptoms of involvement of the nervous contents

of the spinal canal (in addition to local pain and tenderness, and possibly angular curvature, indicative of the bone affection) must especially be noticed burning pains in the course of some of the nerves springing from the implicated portion of the cord. These pains, according to the situation of the disease, may involve the nerves of one or both shoulders or arms, or one or both great sciatic nerves, or certain of the intercostal nerves or of those of the abdominal walls. They are liable to come and go, and when continuous are often attended with exacerbations; moreover, there may be more or less hyperæsthesia in the area of their distribution. The sense of constriction, often likened to the feeling as of a cord drawn tightly round the chest or abdomen, which is so commonly complained of by paraplegic patients, belongs to the same category. These morbid sensory phenomena are due to involvement of the sensory roots of spinal nerves, which generally occurs before the cord itself suffers; and it not uncommonly happens at this period that erythematous or vesicular eruptions or pemphigus becomes developed in the area of distribution of the affected nerves, or even more extensively. It need scarcely be said that motor branches may also be involved, and that limited motor paralysis and atrophy of muscles may ensue. If the disease occupy some considerable length of the spinal canal, or a part in which nerves only are present, the several phenomena due to implication of nerves alone may become pretty widely distributed. Thus, if (as not unfrequently happens) the disease occupy the situation of the cervical enlargement, there may be hyperæsthesia, burning pains, and cutaneous eruptions, involving one or both arms, with flaccidity and wasting of the muscles, and rapid loss of faradic contractility, followed after a time by anæsthesia and complete motor paralysis of the same parts, but without any involvement whatever of the lower part of the body; if it be in the situation of the cauda equina, one or both lower extremities will probably suffer in the same manner as the arms in the former case. In either case there will be partial or total absence of reflex movements in the affected limbs.

After a time, which varies in different cases, symptoms due to involvement of the cord come on. These consist, in the first instance, in numbness, tingling, or formication in the affected limbs, together with some impairment of muscular power. The latter generally increases more or less rapidly until complete motor paralysis is established. The impairment of sensation, on the other hand, for the most part remains stationary, or undergoes amendment, or varies from time to time. But it may of course go on to absolute anæsthesia. As a rule, the muscles below the seat of disease, even if there be total abolition of sensation and motion, retain their natural tonicity and plumpness; their faradic contractility remains normal or increases; and reflex movements may be much more readily induced in them than in health. Such movements, indeed, are often provoked by the contact

of the bed-clothes, or the passage of evacuations. Occasionally, however, there is marked diminution or even abolition both of reflex action and of faradic contractility. If the involvement of the cord persist, the ordinary ascending and descending lesions take place, the former along the posterior median columns, the latter along the lateral columns. The progress of these complications is attended with: aggravation of reflex phenomena, and especially the occasional occurrence of tremulous movements, lasting for a few seconds or even for many minutes at a time, in the affected limbs; the supervention from time to time of clonic or tonic spasms; and gradually increasing rigidity of the muscles, which in the first instance goes along with extension of the limbs, but at a later period with flexion.

The symptoms which attend the form of paraplegia under consideration present considerable differences, in dependence partly upon the situation of the spinal caries, partly upon the degree in which the cord is involved. Thus, when the disease is in the dorsal or lumbar region, one lower extremity only may be involved, or both may suffer in different degrees, or there may be cross paralysis, with loss of motion on one side and impairment of sensation on the other; when the disease is in the neck the arms are commonly affected prior to the legs, and they may be involved unequally; and, even when legs and arms are all implicated, the paralytic phenomena in each may present differences both in degree and in kind. Thus, also, while the rectum and bladder are often little if at all compromised when the lumbar or lower dorsal region is affected, want of control over these viscera is usual when the disease involves the upper dorsal or cervical region; and, indeed, in the latter case this want of control occasionally precedes all other paralytic symptoms. Further, when the cervical spine is the seat of disease, various phenomena, of more or less interest or importance, are apt to be superadded to the simple paraplegic symptoms, among which may be enumerated affection of the pupils of one or both eyes—in the first instance dilatation, at a later period, and more commonly, contraction; cough, difficulty of breathing and of speech; difficulty of deglutition, hiccough, vomiting and gastralgia; epileptic attacks; and permanent slowness of the pulse, with frequent tendency to faint.

The prognosis of paraplegia dependent on caries of the vertebræ is, so far as the paralysis is concerned, not unfavourable: that is to say, presuming that the patient is not carried off by the effects of long-continued suppuration or of degenerative changes in internal organs, by rapid extension of inflammation throughout the cord, or by pulmonary phthisis or other complications, there is always good reason for anticipating, in a case that comes early under observation, a more or less complete restoration of motion and of sensation. Cures have often been effected in patients who had been completely paralysed for one or two years, or even longer. And, indeed, it has been shown by

anatomical evidence that substantial recovery has occurred in cases in which the cord has been permanently reduced in diameter by pressure, and impaired in its texture by interstitial growth, or the development of secondary ascending and descending lesions. It is obvious that those patients who have caries of the cervical vertebræ incur many more, and more serious, risks than those who suffer from dorsal caries; and it may be added that when the atlanto-axial articulation is the seat of disease sudden death from rupture of the ligament and consequent sudden compression of the upper extremity of the cord is to be dreaded. Apart from the causes of death which have already been enumerated, paraplegic patients are apt to sink from the effects of bed-sores, or from the consequences of vesical and renal inflammation.

5. The symtoms referrible to *cervical pachymeningitis* are not unlike those which sometimes attend cervical caries. The affection presents two stages. The first, or painful stage, which lasts two or three months, is characterised by extremely acute pains in the back of the neck, shooting thence to the head and along the upper extremities. These are for the most part constant, but liable to exacerbations. The pains are attended with rigidity of the muscles, most strikingly manifested in those of the neck, which is kept fixed in a position identical with that which is assumed in cervical caries. At the same time the patient complains of formication, a sense of weight in the limbs, and more or less loss of muscular power. Bullous eruptions, too, are not unfrequent. The above phenomena are due to compression and irritation of the nerves. In the second stage the nerves become more or less disorganised, and the spinal cord suffers. The pains in the arms now cease, but the muscles become paralysed and undergo atrophy. It is remarkable, however, that the muscles of the forearm supplied by the musculo-spiral and median nerves are mainly implicated, especially those supplied by the median. Consequently the extensors predominate over the flexors, and the hand assumes the form of a claw. This peculiarity, though not special to cervical pachymeningitis, is special to it among diseases of spinal origin causing muscular atrophy. Subsequently contractions of the affected limbs take place, and areæ of total anæsthesia appear in them and on the upper part of the trunk. Later the lower extremities become paralysed and contracted, but do not undergo atrophy.

Treatment.—The treatment of inflammatory affections of the dura mater and of the lesions so commonly associated with them is on the whole unsatisfactory. We have, as a rule, little or no direct influence over the progress of acute internal inflammation; and chronic inflammatory processes occurring in deep-seated parts are equally seldom amenable to direct treatment unless they be due to certain specific diseases.

1. If we have reason to suspect the presence of circumscribed suppuration between the dura mater and bone, or in the parts immediately

internal to the dura mater, the question of aiding its escape will naturally present itself. If, therefore, the patient have a scalp wound, or be suffering from fracture, necrosis, or syphilis of any part of the skull, it will probably be deemed advisable to apply the trephine. If the source of mischief be the ear, that organ must be carefully examined; if there be evidence of accumulation of matter in the tympanum, the membrane, assuming it to be whole, should be punctured or incised; if it be already perforated and the discharge offensive, the cavity should be washed out carefully with antiseptic solutions; if there be evidence of suppuration in the soft parts about the mastoid process, or behind the angle of the jaw, a free incision should be made; and, further, if we have reason to suspect the presence of pus in the mastoid cells, these should be laid open by the trephine. But the inflammation is not always suppurative; and the application of leeches over the mastoid process or in its vicinity often affords relief, especially in the early stage of the disease, and may possibly tend to arrest its progress. Hot fomentations and poultices to the part are not unfrequently grateful to the patient, and they may be rendered more so by the addition to them of opium, belladonna, aconite, or other preparations having sedative properties. Evaporating lotions or ice to the head also are generally serviceable. As regards internal treatment, it is well in the first place to maintain free action of the bowels, and to restrain as far as possible sickness or other distressing symptoms. Iodide of potassium may also be administered. There is a general feeling against the employment of narcotics in these cases; we must declare, however, that we have often seen much relief to agony and restlessness afforded by the exhibition of largish doses of laudanum or morphia, and never any injurious consequences. If the affection take a chronic or subacute course, it may be well to administer iron, preferably perhaps the syrup of the iodide, or quinine, or cod-liver oil, and to have recourse to counter-irritants. The patient should of course be kept extremely quiet, and be carefully watched, and his diet and secretions should be regulated.

2. The treatment of cerebral pachymeningitis is that of old paralysis and other chronic organic lesions of the brain.

3. For the local treatment of general acute inflammation of the theca vertebralis, leeching, fomentations, the application of ice and counter-irritation may be enumerated; but more important perhaps than any of these is the maintenance of the patient at perfect rest, either on his back, or in a position midway between the back and side. Iodide of potassium may be administered here as in the former case; but tonics, stimulants, and opium are more likely to be of service.

4. Paraplegia from vertebral caries must be treated by absolute rest in the supine position, on a bed specially adapted to the case, and with suitable arrangements for the discharge of the patient's evacuations without the need of movement. And this rest must be main-

tained for a considerable period; indeed, as has already been remarked, a period of one or two years or more may elapse before even a trace of returning muscular power can be observed. In addition, counter-irritation to the neighbourhood of the part affected appears often to be of essential service; the best forms are issues, which should be kept open, or the actual or galvanic cautery, applied on one or both sides of the spine. The general health of the patient should be maintained by good diet, stimulants, and tonics; and great care should be taken to prevent the supervention of bed-sores and of inflammation of the bladder. If the muscles show signs of wasting from disuse, the employment of faradism or of the direct current may be had recourse to—a treatment which may also be beneficial in promoting recovery when recovery is in progress.

5. Spinal pachymeningitis should be treated on the same principles as paraplegia from caries.

III. CEREBRAL AND SPINAL MENINGITIS. TUBERCULAR MENINGITIS. (*Acute Hydrocephalus.*)

Causation.—The causes of meningitis are various. In some cases it depends on the spread of inflammation from adjacent parts—from the brain or cord, from the dura mater or the bones, and more especially from the internal ear; indeed the latter affections rarely produce cerebral symptoms without involving the pia mater to some extent. In some cases it is secondary to the presence of adventitious products, such as miliary tubercles, tumours, and apoplectic clots. It may be the result of injury or of direct exposure to the rays of the sun. It is sometimes of idiopathic origin, sometimes due to pyæmia, and sometimes (as apparently in epidemic cerebro-spinal meningitis) produced by contagion. Tubercular meningitis, which is in fact by far the most common form of cerebral meningitis, may occur at any date between early infancy and old age. It probably, however, occurs most frequently before puberty; but is common up to thirty.

Morbid anatomy.—1. *Cerebral meningitis* is characterised essentially by dilatation and hyperæmia of the vessels of the pia mater, and the effusion of coagulable lymph and inflammatory corpuscles into the meshes of the subarachnoid tissue. The first naked-eye evidence of inflammation is the presence of congestion, which often assumes a patchy character; to this opaline effusion into the subarachnoid tissue succeeds; and presently the corpuscular and other solid products accumulate, at first more especially on either side of the larger superficial veins, whence they gradually creep over the surface of the convolutions and into the depths of the sulci. In some cases the yellowish

or greenish opaque exudation here referred to occupies mainly the prismatic intervals situated between contiguous convolutions and the visceral layer of the arachnoid; in some, where it has spread to a greater or less extent over the convexity of the convolutions, the surface of the brain becomes mapped out into a series of rounded or irregular congested areæ, separated from one another by an irregular network of inflammatory exudation; in some cases again, the accumulation is so considerable that the surface is uniformly covered with it, and the sulci are widely distended. This exudation, which is often solid like an ordinary false membrane, occasionally becomes distinctly purulent. The inflammatory process, which is limited mainly to the substance of the pia mater and to the subarachnoid tissue, nevertheless affects to a greater or less extent the neighbouring arachnoid on the one hand, and the cortex of the brain on the other. The accumulation in the subarachnoid tissue tends to expel the fluid from the arachnoid cavity; and in cases of extensive inflammation this fluid wholly disappears, and the surface of the brain becomes more or less sticky and almost dry. The actual appearance in such cases of inflammatory products on the free surface of the arachnoid or within its cavity is rare. The intimate connection subsisting between the vessels of the pia mater and those of the cerebral cortex renders it almost essential that these should share to a greater or less extent in any process which involves the former. And to a certain extent they do thus share: they become dilated and congested, and more or less inflammatory change takes place in the brain-tissue enclosed within their meshes.

Inflammation may involve any part of the pia mater, and may spread to any extent over it; indeed meningitis, like erysipelas and many other forms of inflammation, has a marked tendency to diffuse itself superficially. Sometimes, however, it occupies mainly the convexity of the hemispheres; sometimes mainly the base of the brain; sometimes mainly the surface of the cerebellum, pons Varolii and medulla oblongata. In the last case the inflammation usually spreads to a greater or lesser extent along the spinal cord. It frequently also involves the velum interpositum and choroid plexuses; and probably on this account, the lateral ventricles usually get distended with fluid. Further the ependyma of the ventricles is often rough from the presence of minute granulations, and the white matter around the ventricles is often found reduced to a pulp.

2. *Meningitis due to tuberculosis* nearly always begins at the base of the brain, is often limited to the base, and is generally most intense there. It differs anatomically from simple meningitis in the presence of grey miliary tubercles, varying from the size of a pin's head downwards. These may be so few in number or so minute as almost to defy detection; they may be so abundant as to form large granular clusters, or irregular, cheesy patches of considerable extent and thick-

ness. They commence in connection with the arterioles whose channels they soon obliterate, and hence congregate especially along the vessels. They are found mainly in the neighbourhood of the circle of Willis, extending thence along the fissures of Sylvius to the lateral aspects of the hemispheres, around the crura cerebri into the great transverse fissure of the brain, and thence to the velum interpositum and choroid plexuses, and also over the pons Varolii. But they are not limited to these situations. They seldom involve the visceral arachnoid, or appear on its free surface; they tend, however, to become developed in connection with the small vessels of the cortex, so that if the pia mater be torn away a greater or smaller number of these vessels with tubercles in their walls are often also torn away, together with portions of the cortical matter itself. Not unfrequently, indeed, masses of tubercles at the bottom of the sulci appear to be imbedded in the substance of the brain. Minute superficial hemorrhages are not uncommon in this condition. When tubercles are few and small, they may sometimes be recognised by the finger as minute hard granules, or be seen on holding up detached laminæ of pia mater to the light; or they may need the microscope for their discovery.

3. *Spinal meningitis* corresponds essentially in all its characters to the description which has been given above; moreover, the presence of tubercles here is not unfrequent. They occur especially, Dr. Greenfield tells us, over the cervical and lumbar enlargements and on the inner surface of the dura mater.

Symptoms and progress.—1. *Cerebral meningitis.* It is impossible to make any practical clinical distinction between simple and tubercular meningitis; we shall include them, therefore, in a common description. Meningitis, especially when it occurs in children, is said generally to be preceded by premonitory symptoms which may vary in their duration from a week or two to some months. They are probably only observed in cases of tubercular meningitis, and are referrible in some degree to the fact that tubercles are already in process of development in the meninges, and in some degree perhaps to the presence of these bodies in other organs, such as the lungs, bowels, and serous membranes. The premonitory symptoms are variously described, and the majority of them have no distinctive characters. The child perhaps becomes emaciated, weak, and pallid, loses his appetite, suffers from constipation, is irritable, fretful, sad, indisposed to play, drowsy in the daytime, and wakeful at night, his sleep being attended with startings and grinding of the teeth, and disturbed by dreams, from which he wakes up frightened and screaming. He may suffer also from febrile disturbance. Amongst other occasional premonitory symptoms may be mentioned some of those which belong to the earlier stages of the established disease, especially vertigo, headache, squinting, sickness, and slowness with irregularity of pulse.

The symptoms of invasion, whether preceded by prodromata or

coming on without them, are exceedingly various. The patient complains: in some cases of pain across the temples, through the eyes, or elsewhere in the head, which is more or less persistent, but liable to paroxysmal exacerbations; in some of vomiting, coming on frequently, without apparent cause and not necessarily attended with marked impairment of appetite; in some of fever of irregularly remittent type, attended, it may be, with more or less severe rigors; in some of double vision. Sometimes the first indications of disease are furnished by more or less dulness, strangeness, or wildness of manner, by impairment of memory or defect of speech, or by the collective symptoms which characterise the early stage of delirium tremens. Sometimes the attack is ushered in by an epileptic seizure.

The progress of the disease is usually divided into three stages, which in typical cases are often fairly well marked. The first stage, which includes the invasion, is generally characterised by fever, elevation of temperature, increased rate of pulse, and the phenomena of nervous irritation; the second stage is usually attended with diminution or cessation of fever, slowness of pulse, or the phenomena of commencing paralysis; the third stage, or that of collapse, is the stage usually of convulsions and coma, during which also febrile symptoms not unfrequently again manifest themselves and the pulse becomes extremely rapid.

The first stage is ushered in for the most part with various combinations of the symptoms which have been above enumerated, and is generally attended with elevation of temperature—febrile exacerbations, often associated with rigors, coming on irregularly and sometimes several times a day; acceleration alternating with slowness of pulse; headache, which is often so severe that the patient screams out with it or supports his head with his hands, and which is not unfrequently associated with tenderness of the scalp and neuralgic pains in the back of the neck, extending, it may be, to the limbs; nausea, and more or less uncontrollable vomiting; constipation; disturbed sleep or sleeplessness; sadness and taciturnity, or querulousness, or tendency to delirium. To these symptoms are not unfrequently added hyperæsthesia, tremulousness and muscular debility, intolerance of light and sound, hemiopia, illusive appearances, double vision or squinting from spasm of the muscles of the eyeballs, and contracted pupils. In this stage young children are generally fretful, peevish, agitated at the approach of strangers, and resentful at the attentions of the nurse or mother, and not unfrequently even now utter the characteristic hydrocephalic cry.

In the second stage the patient becomes comparatively quiet, and passes into a drowsy condition; his temperature for the most part falls somewhat, and though generally still a degree or two above the normal, may sink to the normal or even below it; and his pulse becomes slower than natural, and at the same time more or less irregular. The

transition from unrest to rest, and the subsidence of fever give a delusive aspect of convalescence. In this stage the cephalalgia, the exaltation of the senses of sight, hearing, and touch, the nausea and sickness, and the irritability of temper, or sadness, or moroseness, all subside, and the patient becomes apathetic. He perhaps sleeps continuously, and is roused with difficulty to put out his tongue or take nourishment; he probably does not refuse food, but he does not ask for it; nor does he trouble himself to restrain his evacuations. His breathing, like his pulse, is irregular, and characterised by a series of rapid respirations followed by long intervals of complete apnœa. It is during this period that certain other phenomena are peculiarly apt to be present—to come on for the first time, or to undergo aggravation. The hydrocephalic cry, in children especially, now becomes a marked feature of the case; it is uttered at frequent but irregular intervals. The following is Trousseau's description of it:—'He groans from time to time, opens his eyes wide, which shine as they do in persons who are drunk. His face, which is usually extremely pale, flushes for a second or two; then he closes his eyes again, and resumes his former aspect. Generally, as he thus opens his eyes, and as his face colours up, the child utters a sharp plaintive cry, which is perfectly characteristic.' The face, which is generally pallid, is liable to sudden temporary flushes, and the tache cérébrale is easily produced. Retraction of the abdomen is almost always present. During this stage the patient is apt to roll his head from side to side, to move his hands and arms restlessly, to wave them or throw them about, to pluck at the bed-clothes, or to pick his nose, lips, or ears, and to perform various other movements; he may suffer from quiet delirium, or present partial convulsive movements of his face or limbs; his pupils may get dilated or unequal, and irresponsive to light; sight may fail; and paralysis may come on, especially ptosis, paralytic strabismus, paralysis of the portio dura or hypoglossal, or hemiplegia.

The third stage is characterised by the supervention of convulsions and coma, or of coma alone. The patient, who could hitherto be roused with more or less ease, now scarcely responds to any external influence. He is anæsthetic, deaf, blind, the pupils are dilated, probably unequal, and react slowly or not at all to light. Sometimes inflammation or ulceration of the cornea takes place. Paralysis has become more pronounced either in certain muscles or groups of muscles, or on one side of the body. He still, however, rolls his head about; still has subsultus or tremors, or picks at the bed-clothes; still utters the distressing cry peculiar to the disease. The respirations get more frequent and irregular. The pulse may still remain below the normal rate, but more usually becomes exceedingly rapid and feeble. The temperature for the most part rises, especially in the internal organs; that is to say, the limbs get cold and dusky, while the trunk and viscera are burning hot. But the temperature during this stage is liable to

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great variety: in one case it rises rapidly as death approaches; in another it remains elevated two, three, or four degrees above the natural standard; while in yet another it falls 10 or 15 degrees or more below it. The cheeks are alternately pale and flushed, and the surface bathed in sweat. The tongue, which during the earlier stages may have been natural, or may have presented more or less whitish fur upon its surface, in the third stage generally becomes thickly coated, dry, and brown, and the teeth get covered with sordes. Convulsions, as a rule, are now frequent, sometimes incessant, sometimes slight, sometimes violent, sometimes limited to the face or to the hands, sometimes unilateral, sometimes general. They are apt to increase in severity as the fatal end approaches; and the patient, who always dies comatose and sometimes collapsed, is not unfrequently carried off in a convulsion.

In addition to the delusive appearance of amendment which characterises the beginning of the second stage, it is not uncommon, towards the close of the second or beginning of the third stage, for the patient to wake up as it were from his semi-coma or coma, to recognise his friends, and to take an interest in what is going on around him. The amendment may last a day or two, and may recur, and is apt not unnaturally to raise the hopes both of the friends and of the medical attendant. Unfortunately, however, these hopes are, almost without exception, doomed to speedy disappointment, and sooner or later all the symptoms return, become aggravated, and death ends the scene. It is nevertheless a fact not only that temporary recovery occasionally takes place, but that patients who present distinct symptoms of meningitis now and then recover permanently.

The duration of cerebral meningitis is very uncertain. It is generally from one to three weeks, and not unfrequently about a fortnight. It may be only three or four days. The different stages also which have been enumerated vary in duration, both actually and relatively, and in all cases the transition from one to another is quite gradual.

It must never be forgotten, however, that although there is a common tendency for the progress of meningitis to divide itself into successive stages, and although these successive stages have a tendency to assume such characters as have been above assigned to them, in a very large proportion of cases the symptoms and progress of the disease diverge widely from the type. No disease, indeed, is more protean in its features than meningitis, probably none simulates so many other disorders. In some cases, as for example when meningitis complicates acute pneumonia, erysipelas, or other inflammatory affections, the only indication of what proves to be extensive meningitis may be the supervention of drowsiness, coma, and collapse during the day or two preceding death. In some cases, indeed, even when the inflammation of the brain is uncomplicated, drowsiness and coma are the only symptoms ever recognised. In many cases the early stage of the disease is mistaken

for inebriation or delirium tremens; and indeed the symptoms of meningitis are not unfrequently, during the greater part of its duration, almost exact counterparts of those of delirium tremens. In other cases the symptoms have a close resemblance to those of enteric fever, and in children to that vague and uncertain malady which is commonly termed 'gastric fever'—especially to those cases of these affections in which the accustomed diarrhœa is replaced by constipation, and the abdomen fails to present its ordinary flatulent distension, while at the same time nervous phenomena, such as headache, sleeplessness, irritability, and delirium prevail. In other cases, again, meningitis presents many of the features of general tuberculosis; it must be recollected, however, that general tuberculosis is very apt to be attended with involvement of the surface of the brain, and that hence the supervention of meningitis in the course of the general disease should not be overlooked. Sometimes the patient, from the commencement of his malady up to the occurrence of coma, is in a condition of mild delirium; occasionally, but much more rarely, he is in a state of maniacal excitement; sometimes epileptic convulsions predominate; sometimes he is sensible from first to last. In some instances headache and vomiting never present themselves; in some distinct paralysis never occurs; in some paralytic phenomena form the most striking features of the malady. The hydrocephalic cry may never be uttered.

Still, however obscure the case may be, there is almost always something during its progress which reveals to the observant practitioner its fatal character. The cloven hoof shows itself. There is vomiting without obvious cause, or fever of a certain character, or retraction of the head, or hyperæsthesia; or there is some affection of the pupils, or some temporary or permanent paralysis, even though it be limited to the levator palpebræ or one of the ocular muscles; or there is some convulsive movement, or the characteristic distressful cry, or some peculiar change in the mental condition; or the respirations are characteristically irregular; or the muscular debility or tremulousness are out of all proportion to the other symptoms exhibited. Further, optic neuritis is frequently present, even from the earliest period of the patient's illness; and generally also Trousseau's tache cérébrale, a phenomenon, however, of little importance, can be readily developed. It consists in the speedy appearance and long duration of a comparatively wide blush of redness in the course of a line made by drawing the finger-nail or the point of a pencil along the skin, more especially on the face, abdomen, or inner aspect of the thigh. Since the larger number of cases of meningitis are of tubercular origin, accuracy of diagnosis may often be insured by careful attention to the history of the patient and by careful examination of his lungs and other organs in reference to the presence of tubercular disease in them. Occasionally tubercles may be recognised in the choroid.

The great variableness of the phenomena which attend meningitis

becomes easy of explanation when we look to the morbid anatomy of the disease and consider how many parts are liable to suffer, and how unequally they may be involved. We have seen that the morbid process, especially if it be of tubercular origin, is peculiarly apt to invade the cortical substance of the brain. What wonder that mental phenomena and convulsions should ensue, and that these should vary largely in their details in different cases? We have seen how almost invariably the base of the brain suffers more than other parts, and how the nerves are consequently liable to be involved in the inflammatory process. What wonder that hyperæsthesia and paralysis, variable as to their seat and degree, should be present? We have seen that the lateral ventricles are apt to get distended with fluid, their parietes softened and compressed. Is it remarkable that, when these changes take place, coma and paralysis are of common occurrence?

2. *Spinal meningitis.*—The symptoms of acute inflammation of the spinal pia mater differ but little from those which have been ascribed to inflammation of the theca vertebralis, and necessarily have some resemblance to those developed in the course of myelitis. Indeed, many of the symptoms which occur in inflammation of the dura mater are really due to the extension of disease to the pia mater; and many of those of inflammation of the pia mater are essentially referrible to involvement of the subjacent nervous matter.

When any considerable length of pia mater is affected, more or less fever of a remittent character, and possibly attended with rigors, will very likely be present. There will probably also be anorexia, thirst, and abnormal rapidity of pulse. Sometimes, however, the pulse is slow. The patient most likely complains of pain in the course of the spine, not much increased by simple pressure, but greatly aggravated and sometimes amounting to unendurable agony when either muscular movements are performed or the spinal column is bent or twisted. There is generally more or less rigidity of the voluntary muscles—the muscles of the back, and more especially those of the neck, being chiefly affected, and the head consequently retracted; the elbows are apt to stand out from the body, and the forearms and hands to be somewhat flexed; the lower extremities are probably similarly affected; the jaws are often firmly closed, and the muscles of the face contracted so as to give to the expression the well-known risus sardonicus. Further, sudden twitches and spasms of a tetanic character are apt to occur from time to time, not only in the muscles of the back, which becomes consequently a little more arched, but in those of the limbs and head and neck. At the same time the patient complains of pain, not merely in the back, but also in the head, and especially in the extremities, into which it shoots in sudden paroxysms, which are excited whenever he executes any movement, or his muscles are forcibly disturbed by the hands of the attendant. There is not unfrequently also some hyperæsthesia.

Partly mingled with these phenomena, but more especially supervening upon them, is impairment of voluntary movement and sensation; the patient loses more and more the power over those limbs which correspond to, or are below the seat of lesion, and he loses also, in a greater or less degree, control over the rectum and bladder. Indeed incontinence of urine and fæces is apt to take place even when paralysis of the voluntary muscles is very slight, and while at the same time the patient's mental faculties appear to be intact. Tingling, formication, or numbness is not unfrequently present. If the disease involve the higher part of the spinal pia mater and that of the medulla, difficulty of respiration, speech, deglutition, and mastication may be present. Priapism and increased irritability of the excito-motor functions are rarely observed. Vertigo, headache, slight delirium, and other cerebral phenomena generally arise in the course of the malady, especially if the meningitis extend from the spine to the base of the brain.

The main features of the disease appear to be: pain, more or less intense, along the spine and shooting into the extremities, especially aggravated by movement; hyperæsthesia; rigidity with occasional spasmodic contraction of the voluntary muscles; and more or less want of power over the rectum and bladder. Absolute paralysis is rare, and absolute anæsthesia still rarer. Indeed the patient, who has probably been for some days confined to his bed, apparently unable to move, and suffering agony when involuntary or other movements are effected, and exerting no control whatever over his evacuations, will sometimes suddenly, in his restlessness, agony, or delirium, sit up in bed, or even get out of bed and pace the room.

The commencement of the disease, especially if it be secondary to any other serious malady, is often insidious and obscure. And even when it is of purely idiopathic origin, the symptoms may be so slightly pronounced during the first three or four days that the patient refuses to go to bed, and in some cases goes on with his usual avocations. The initial symptoms are not unfrequently more or less pain, usually supposed to be rheumatic, in the course of the spine and of the nerves which are given off from it, increased by movement, and attended with febrile disturbance, restlessness, irritability of temper, and sleeplessness.

Spinal meningitis is a very fatal malady, and although doubtless some persons recover from it, the great majority die, succumbing for the most part between the third or fourth day and the third or fourth week. Most deaths, however, occur within the week, and are due either to asphyxia or to asthenia, the latter often being promoted by the rapid supervention of bed-sores and other complications.

Treatment.—Cerebral meningitis for the most part defies all medical treatment; still, as recovery certainly takes place occasionally, even when it is of a tubercular origin, it behoves us to give some care to

the management of all cases of the kind that come under our charge. The patient should be placed in a room sufficiently darkened to be grateful to his irritable eyesight, sufficiently quiet to prevent all auditory disturbance, and at the same time cool and well-ventilated. He should be carefully watched by a quiet and judicious attendant. Everything indeed should be done to avoid the infliction of discomfort on the hyperæsthetic senses, to calm irritability, and to promote rest. If the limbs be cold, they should be kept at an equable temperature by means of flannel or other warm clothing. The headache which is so often present may be relieved by the temporary application of evaporating lotions, or ice, to the forehead or shaven scalp; and it may for the same and other purposes even be advisable to apply leeches behind the ear, or blisters or other counter-irritants to the temples, scalp, or back of the neck. Leeches, however, should only be applied early in the disease. The number to be employed, and the amount of blood to be removed, must be determined partly by the age of the patient, partly by the other circumstances of the case. Thus, if the meningitis occur in a healthy-looking adult, free removal of blood even by venesection will be well borne, and may be highly beneficial. In a young child two or three leeches are generally ample. The relief of head symptoms is often attended with alleviation of sickness. But this may sometimes be treated directly with advantage by the administration of either ice, bismuth, hydrocyanic acid, oxalate of cerium, or other remedies which are ordinarily given to assuage sickness. It is generally highly important to keep the bowels freely open, and this (in consequence of the extreme obstinacy of the constipation) is sometimes exceedingly difficult to effect. Enemata may be resorted to; but it is generally better to give purgatives by the mouth, and especially to give those which are not likely to upset the stomach. Sir T. Watson recommends as the best purgatives for children calomel and jalap, or calomel and scammony. Castor oil also generally agrees well with children. In the case of adults purgation should be actively employed. Amongst special remedies, may be enumerated: first, simple saline or febrifuge medicines, which doubtless have little efficacy; and second, iodide of potassium, which is probably advocated rather on theoretical grounds than from actual experience of its value. Opium is generally considered to be contraindicated; we must confess, however, that we have not unfrequently administered it during the earlier stages of meningitis, not only without obvious injury to the patient, but with manifest relief to his irritability and insomnia. Fluid nutriment should be administered in small quantities, and at frequent intervals.

Prophylactic measures are of paramount importance in the case of either children or adults in whom there is reason to fear the supervention of meningitis. Their studies or other mental labours should be intermitted; they should be kept quiet, in mind and body, should keep

early hours, be removed (if need be) to some healthy locality, occupy well-ventilated rooms, have ample wholesome nourishment, and be placed under a course of cod-liver oil and tonics. Further, all their functional disturbances should have due attention paid to them, sickness should be obviated, constipation overcome.

Adults as a rule may be treated far more actively than children, and those who are non-tubercular far more actively than those who are tubercular. For such patients the main remedies during the attack consist in cold to the head, leeching behind the ears, and active purgation.

As regards the *treatment of spinal meningitis*, it is essential: that the patient be kept in the recumbent posture on a suitable bed; that he be kept scrupulously clean and dry; that all parts liable to bed-sores be defended from the effects of pressure; and that the condition of his bladder and bowels be carefully watched, and the bladder, if necessary, relieved periodically by the catheter. In other respects the remedial measures to be employed are the same as those which are supposed to be serviceable in cerebral meningitis.

IV. ENCEPHALITIS AND MYELITIS.

Inflammation and Suppuration of the Brain and Cord.

Causation.—The circumstances which determine acute inflammation in the brain and cord are, like those which cause inflammation elsewhere, very various. In most cases it is due to extension from without: spreading directly from the pia mater, or (in those cases in which pus is extravasated into its cavity) from the arachnoid; or following upon inflammation originating in the dura mater or the bones of the skull or spine; or referrible to the direct spread in depth of erysipelatous and other like affections of the skin and subjacent tissues. In a large number of cases encephalitis or myelitis arises from the influence of some irritating mass, such as a clot, tumour, parasite, or patch of softening situated in the nervous substance or implicating it from without. It is a common consequence of injuries, even if these be unattended with fracture. It is sometimes referrible to pyæmia. And occasionally it seems to be an idiopathic affection, due to exposure or to causes which we fail to recognise.

Morbid anatomy.—1. *Encephalitis.* There is no reason to doubt that inflammation occasionally involves the great bulk of the brain, as it certainly sometimes does the whole thickness of the cord in no inconsiderable proportion of its length. The cases in which this phenomenon is believed to have been present are those in which the

general substance of the brain has been found after death much congested and softened, and in which the inflammatory process if present at all must have been in quite its initial stage. The condition, in fact, has differed little from what is seen in general congestion of the organ, as it is sometimes observed in fatal cases of epilepsy, delirium tremens, or sunstroke. In most cases of encephalitis the inflammation is of limited extent, occupying sometimes tracts of grey matter, sometimes tracts of white, and often involving portions of both. In some instances a single patch is present, in some there are several; and their sizes and forms vary within wide limits. If, however, they be not determined as to shape and extent by the fact of their being secondary to some clot or other adventitious mass, or to meningeal inflammation, they tend to assume a roundish or ovoid form, rendered more or less irregular by the configuration of the surface of the brain and the arrangement of its parts. The changes which mark the early stage of acute inflammation are more or less circumscribed congestion, effusion of fluid and other inflammatory products, and softening. The tissues of the affected part are swollen and pulpy, and generally admit of being readily washed away under the impulse of a stream of water, leaving a shreddy excavation behind. The redness is more or less intense according to the quantitative relation the congested vessels have to the amount of exudation, and is generally patchy. Sometimes small extravasations of blood occur. Further, more or less congestion and œdema, together with some yellowness of tint, may generally be observed in the surrounding tissues.

With the progress of the inflammatory process, exudation-corpuscles or pus-cells accumulate in the affected district, which gradually loses its congested aspect and gets yellowish or greenish, and less and less consistent. Presently the central portion becomes diffluent, and an abscess forms; the pus of which is thick, glairy, yellowish or greenish, and occasionally offensive, and the parietes of which are constituted by the still solid but soft and breaking down tissue. Occasionally the abscess becomes encysted, the capsule sometimes attaining a thickness of a quarter of an inch. The microscopical characters of the inflamed patches vary somewhat according to the stage at which the process has arrived. In the earlier periods there may be observed: vascular changes with accumulation of leucocytes in the perivascular sheaths; more or less destruction of the nervous elements —the myeline of the nerve-tubules breaking up into globules of various sizes; and a greater or less development of granule-cells. Somewhat later, the degenerating tissue becomes loaded with pus-corpuscles. In the fully-formed abscesses, the puriform matter is not unfrequently found devoid of pus or other cells, and comprising only fatty matter and débris of tissues.

Abscesses of the brain are not of very frequent occurrence, and are mostly solitary. When multiple, they are almost invariably pyæmic.

The abscesses of most interest to the physician are those which are secondary to disease of the ear, nose, orbit, or other parts of the skull. When secondary to ear-disease, they occur either in the adjoining part of the middle or posterior cerebral lobe, or in the corresponding lateral lobe of the cerebellum, or somewhat more rarely in the pons Varolii. In most instances the surface of the bone is carious, the dura mater over it is softened and inflamed, and the abscess is situated in a diseased patch of brain, which has become adherent; but occasionally the dura mater remains apparently healthy, and the abscess moreover is separated from the surface by a layer of healthy-looking brain-substance. The explanation of this fact is not clear; but it is generally assumed to be due to the plugging up of some of the vessels passing from the sinuses into the substance of the brain, or to the extension of inflammation along them. According to Sir W. Gull's and Dr. Sutton's statistics, it appears that abscesses occur equally on both sides of the brain; and that, although any part may be their seat, they are most common in the middle cerebral lobes. The presence of an abscess of medium or large size in the substance of the brain causes more or less general enlargement of the lobe or part in which it occurs, with flattening of the surface of the brain over it and in its vicinity, obliteration of the sulci, and displacement of the subarachnoid fluid. An occasional result of abscess is its extension, by bursting or by gradual erosion, into the cavity of the arachnoid, or into one of the ventricles, or its discharge externally through the ear or nose.

2. *Myelitis.*—Inflammation of the substance of the cord is attended with similar changes to those above described. When secondary to disease of the membranes it first involves the white substance, and only subsequently, and at a comparatively late period, extends to the central grey columns. Idiopathic inflammation, however, mostly affects primarily and principally the grey matter. It results in softening of the nervous tissues, with a much more marked liability to the extravasation of blood than attends cerebral softening; indeed, in Charcot's opinion, hemorrhage (however copious) into the substance of the cord is mostly, if not always, the result of previous inflammatory softening. Inflammation generally tends to implicate the whole thickness of the cord, and though frequently limited to comparatively short lengths of it, not very uncommonly involves extensive tracts. Actual abscesses are of rare occurrence.

Symptoms and progress.—1. *Encephalitis.* The symptoms which may be referred to inflammation of the brain-substance extend no doubt over a wide range. Indeed, it is unquestionable, as has already been pointed out, that many of the symptoms of cerebral meningitis are really due to implication of the subjacent grey matter. Further, these conditions are so generally combined that it would be a mere waste of space and of ingenuity to endeavour to make any absolute distinction between them.

Of acute general inflammation of the encephalon, there is little to be said. The cases are rare, the symptoms which they present are vague, and the morbid changes which are observed after death are, to say the least, obscure. They are mostly of rapid progress, and attended, as Dr. Wilks observes, with more or less severe pyrexia, delirium, dulness of intellect, and final coma, but perhaps no other symptoms specially referrible to the brain. Occasionally there is a preliminary stage in which, as he says, the patient may suffer from headache, sickness, slow pulse, and constipation. It need scarcely be observed that these are symptoms which are not unfrequently met with in cases of sunstroke, and occasionally in persons who have been indulging continuously, for some days or weeks, in excessive drinking.

The symptoms which attend inflammation secondary to the presence of apoplectic clots, patches of softening, tumours, and the like, in the substance of the brain are also exceedingly vague. A little accession of fever, a little increase of headache or giddiness, a little failure of appetite, a little aggravation of the paralytic phenomena from which the patient has been suffering, a little impairment of intelligence, the occurrence of delirium, convulsions or coma, and perhaps the development of some rigidity in the affected limbs, some hyperæsthesia, some pain in muscles or joints, or some tendency to the formation of bed-sores on the affected side, separately or in combination, are the main indications of its supervention.

The symptoms due to circumscribed inflammatory softening, or suppuration, are not less vague and obscure than those which belong to the varieties of inflammation already considered. Among those which may be present are febrile disturbance with rigors, anorexia, vomiting, and constipation, vertigo, headache often occurring mainly at one spot, dulness, stupidity, delirium, coma, epileptiform convulsions, paralysis, affection of speech or of the special senses, and want of control over the bladder and rectum. In addition, few if any of the other symptoms which may be observed in cerebral meningitis may not at one time or another, or in certain cases, be present here. On the other hand, an abscess may be found encysted in some part of the brain-substance, which has existed there for weeks or months, possibly years, without giving any hint of its presence. It is obvious, indeed, when we consider the various sizes which inflamed regions or abscesses present, the various positions which they may occupy in the substance of the encephalon, and the various lesions (pyæmia, disease of the ear, and inflammation of the dura mater, pia mater, or both) with which they are apt to be associated, that the symptoms which accompany them must present the greatest possible variety. Thus, as regards febrile symptoms, there can be little doubt that the inflammatory process in the brain will usually be attended with more or less marked fever, and not improbably with rigors, but that these phenomena will certainly be aggravated if pyæmia be present, or if suppuration be taking place beneath

the dura mater, or pus have escaped thence into the cavity of the arachnoid; while, on the other hand, they may be entirely absent if the patch of inflammation be small, or if it pass into the chronic state, or form an encysted abscess. Again, pain is one of the most common symptoms of abscess of the brain: pain, various in character, often referred to a particular spot, sometimes affecting the eyes, or shooting through the temples, or occupying the back of the head; but pain may be absent from first to last, and generally, when it is present in any marked degree, is due rather to co-existent affection of the bones or dura mater than to the cerebral disease. Another frequent consequence of localised inflammation or abscess is paralysis or interference with the functions of one or other of the nerves of special or common sensation. But while it will be admitted that an extensive lesion will probably cause hemiplegia on the opposite side of the body, it is obvious that the presence of hemiplegia, and especially of affection of any one nerve, or group of nerves, will depend less on the size of the lesion than on its situation. If it be seated in the pons Varolii or medulla oblongata, a wide extent of paralytic affections of the spinal nerves will almost necessarily ensue, and respiration and deglutition be largely interfered with. The importance, however, of analysing the complex nervous symptoms which may be present, in reference to the facts of cerebral localisation, with the object of determining the locality of the lesion, is sufficiently obvious. The evidences of optic neuritis may often be observed in these cases.

There are, indeed, no special symptoms or groups of symptoms the presence or absence of which will enable us to diagnose the presence or absence of a patch of inflammatory softening or of an abscess in the brain. We have good reason, however, for suspecting the presence of such lesions when symptoms of the kind above enumerated supervene in the course of chronic otorrhœa, or of syphilitic or other forms of caries or necrosis of the bones of the skull, or upon injuries to the skull or brain. Yet, even here, the symptoms which we take to be indicative of cerebral abscess may be due to suppuration beneath the dura mater, with extension to the arachnoid or pia mater.

The beginning of circumscribed inflammation or suppuration of the brain is sometimes marked by a sudden attack of convulsions, sometimes by unilateral or more limited loss of power or sensation, sometimes by cephalalgia with vertigo and vomiting, sometimes by impairment of intelligence, sometimes by fever. The pulse, as in other cerebral affections, is not unfrequently slow and irregular. The progress of the disease, like that of meningitis, may often be divided into a stage of irritation and one marked by impairment or abeyance of the functions of the nervous centres, passing into collapse. In the former we may observe vertigo, headache, intolerance of light, hyperæsthesia, irritability, wakefulness, with perhaps delirium and vomiting; in the latter, disappearance of pain, paralysis, want of con-

trol over the action of the bladder and rectum, drowsiness, stupor, coma. Convulsions may occur at any time; but, we repeat, the symptoms are variable in the highest degree, both as to their nature and the order of their succession; many who have abscess of the brain die without the presence of abscess having ever been suspected; and many cases in which we venture to foretell that abscesses will be discovered, falsify our prediction upon the post-mortem table. Reckoning the duration of cases from the time when acute symptoms indicative of brain disease first manifest themselves, they may be said to prove fatal usually from the fifth or sixth day down to the end of the third or fourth week. Death is commonly preceded by coma, but may be due to asthenia or to asphyxia.

2. *Myelitis.*—The symptoms of acute myelitis are, in the main, those of suddenly occurring paraplegia, and are therefore in many respects like those of spinal meningitis. They have a closer resemblance, however, to those which follow upon fracture of the spine attended with injury to the cord. In considering the symptoms due to myelitis there are two or three considerations which it is important to bear in mind. In the idiopathic affection the inflammation affects primarily, and in the highest degree, the central grey matter of the cord; hence it follows that sensation will probably be at least as soon and as profoundly involved as motion. In meningitis, and other diseases affecting the cord from without, the white matter is mainly implicated, and motion is lost in far higher proportion than sensation. In myelitis there is a great tendency for the disease to diffuse itself throughout the length of the cord, and thus not merely to cause gradually ascending paralytic phenomena, but also to annul the reflex functions of the cord and the electrical contractility of a gradually increasing number of muscles. The result is different, as we know, when paraplegia follows any limited lesion of the thickness of the cord. In myelitis the profound involvement of the elements of the grey matter naturally tends rapidly to induce the peripheral nutritive consequences of spinal lesions, especially wasting of muscles, development of bed-sores on the sacrum and elsewhere, and inflammation of the bladder and kidneys. In this respect especially idiopathic myelitis far more resembles the effects of serious accidental injuries to the spine and cord than any other form of disease. Further, the grey matter of the cord is, so far as we know, insensible to pain, and lesions which directly involve it are also unattended with pain. Myelitis is not, therefore, usually a painful disorder; pain, indeed, in spinal affections is almost always the consequence of pressure upon, or involvement of, the sensory nerves within the spinal canal, or of the posterior roots in their passage through the white matter of the cord. Hence pain in the back, extending to the trunk or extremities, is much less likely to be due to myelitis than to meningitis, and, it may be added, less likely to be due to meningitis than to the growth of tumours or the

extension of aneurysms. Lastly, twitchings and tetanic spasms of the muscles are in no sense an indication of myelitis. They are common, however, in meningitis.

The symptoms of acute myelitis sometimes come on gradually in the course of a few days, sometimes manifest themselves with sudden intensity. A patient, perhaps after long-continued over-exertion, or exposure to the weather, or sleeping on the damp grass, or it may be during the progress of vertebral caries, is attacked in his toes and feet with numbness and tingling, which gradually extend upwards, and are succeeded after a varying interval by the progressive or almost sudden annihilation of sensation and voluntary motion in the lower extremities, and up to the level of a horizontal line which corresponds to the upper limit of the distribution of the involved spinal nerves. The relative dates at which sensation and voluntary motion are lost vary in different cases, as also does the degree in which the corresponding limbs are involved. Nor does it necessarily follow that either sensation or motion is wholly abolished. There is not usually absolute pain, still less pain of a neuralgic character; but there is often a more or less distressing sense of restlessness and tingling in the paralysed limbs, and of constriction round the abdomen and chest. There may, however, be cutaneous hyperæsthesia at the upper limit of the affected region. Twitchings of the paralysed muscles may attend the earlier stages of paralysis; but generally these soon cease, and the directly implicated muscles, as a rule, speedily lose their electric contractility, and become flaccid and lifeless. The muscles, however, thus affected are only those supplied by the diseased length of cord; they are hence few or many, according to circumstances; while all those which are supplied by nerves given off lower down retain this and their reflex excitability and their bulk, as in ordinary cases of paraplegia. Priapism is occasionally present; the bowels are constipated, and the motions discharged unconsciously; and there is either retention or incontinence of urine. Bed-sores, especially over the sacrum, are apt to arise, in spite of every precaution, sometimes within four or five days of the commencement of paraplegia; and at the same time, even where the greatest care has been taken, the urine probably becomes ammoniacal and the mucous membrane of the urinary tract inflamed.

It need scarcely be added that the distribution and extent of the paralytic phenomena will be determined by the position and extent of the disease; that there will be involvement of the lower limbs only when the disease occupies the mid-dorsal region; of the upper and lower extremities when the cervical enlargement is included; hemiparaplegia when one side of the cord mainly suffers; difficulty of respiration when the intercostal nerves are implicated; apnœa when the origins of the phrenic nerves are also involved. In the last two cases difficulty of speech and inability to discharge accumulating mucus from the bronchial tubes add seriously to the patient's sufferings.

It must not be forgotten that, although cerebral symptoms, neuralgic pains, and spasmodic action of the muscles are no necessary parts of myelitis, they are not uncommonly superadded in consequence of the co-existence of some degree of meningeal inflammation and brain implication. Neither must it be forgotten that more or less marked fever is often present, which may be attended with remissions, rigors, and sweats.

Acute myelitis is a very grave disorder, and generally terminates fatally in the course of a few days or at the outside a few weeks. In the less severe or less extensive cases, however, life may be prolonged for an indefinite period with persistence of paraplegic symptoms; or the patient may recover in some degree, or may even undergo perfect restoration to health. The cause of death varies. The patient frequently dies, however, of asthenia, which may be largely determined by secondary affections of the skin and urinary passages; or of apnœa referrible to implication of the respiratory nerves.

Treatment.—For the treatment of inflammation of the substance of the brain little or nothing can be done directly. We may, if the symptoms be severe and their onset sudden, have recourse to the classical measures:—namely, cold to the head in the form of evaporating lotions, affusion, or the ice-bag; purgation by means of active drugs, and especially of such as cause watery evacuations; and the abstraction of blood, preferably by leeches, from the temples or behind the ears, or by cupping at the nape of the neck. It must be admitted, however, that these measures are not often of any obvious utility. For the most part, however, the same general treatment may be employed here as has already been suggested for meningitis. If, however, the affection be traceable to inflammation of the ear, or disease of the bones of other parts of the skull, the question of surgical interference will naturally arise.

For the treatment of myelitis we must also refer to remarks which have been previously made under the head of spinal meningitis.

V. SCLEROSIS. (*Chronic Inflammation.*)

The affections which we are about to consider under the above heading form a very interesting clinical group, which has been mainly investigated and unravelled by the labours of Duchenne, Vulpian, and Charcot in France, by Todd, Gull, and Lockhart Clarke in our own country, by Brown-Séquard, and in a greater or less degree by various other physicians both here and abroad. They are probably all of inflammatory origin; but the inflammation to the effects of which they are due is, like cirrhotic inflammation of the liver, marked, for the most part, by the slow development of adventitious fibroid tissue attended

with the gradual wasting and degeneration of the essential elements. In some instances, according to Charcot, the inflammatory process begins in the nerve-cells, in which case it may either continue strictly limited to them, or gradually involve the surrounding connective tissue to a greater or less extent.

In the majority of cases the first indication of disease would seem to consist in an overgrowth or hyperplasia of the neuroglia, indicated by the appearance in it, and in the perivascular spaces, of a greater or less abundance of the cells characteristic of embryonic tissue, together with an increase of the amorphous intercellular substance which takes part in the constitution of the neuroglia, and enlargement of the vessels. At this stage the affected tracts of tissue are somewhat swollen, but their nervous elements present little, if any, evidence of disease. As the morbid process advances, however, the hypertrophied neuroglia contracts and hardens, its newly-formed cells get small and indistinct, the originally amorphous matrix assumes a delicate fibrillated character, and the blood-vessels become thick-walled and their channels narrowed. Moreover, the nerve-tubules and nerve-cells of the diseased regions now undergo important changes. When the sclerosis is situated in the white substance of the cord or brain, the nerve-tubules are seen to be more widely separated from one another than natural, the width of the intervals between them depending of course on the quantity of adventitious matter which has accumulated there. The tubules for the most part diminish in thickness, mainly in consequence of the partial disappearance of the white substance of Schwann, and sometimes become moniliform; but intermingled with such tubules others may generally be observed which are either of normal thickness or are actually increased in diameter from swelling of the axis cylinder. In the most advanced conditions of disease the nerve-tubules are greatly atrophied, and in many instances wholly deprived of their myeline sheaths; but they are rarely, if ever, absolutely destroyed. When the sclerosis occupies the grey matter, as, for example, the anterior cornua of the cord, we find, in addition to atrophy of the nerve-tubules, corresponding changes in the nerve-cells. In some instances, as Charcot points out, they become swollen, and, it may be, enormously enlarged, faintly granular and opalescent; and at the same time their processes appear more or less thickened and twisted. These changes, which he ascribes to irritation, belong to the early period of the disease. Much more commonly the changes observed are of an atrophic character. In some cases the cells undergo pigmentation, diminish in size, and assume a more or less globular form, their processes at the same time becoming attenuated and shortened; and after a while they get reduced to simple roundish accumulations of pigment, and finally perhaps wholly dissipated. In other cases no pigmental deposit takes place, but the cells shrink in all their dimensions, each one drying up, as it were, into a kind of mummy. The processes

partake in this change, and disappear in a greater or less degree. Corpora amylacea are usually more or less abundant in sclerotic tracts; and Lockhart Clarke has described as sometimes present in them irregular patches of disintegration, from which all traces of the normal elements of the part, whether neuroglia, blood-vessels, nerve-tubules, or nerve-cells, have disappeared.

Sclerotic change is indicated, as to its coarser features, by a more or less greyish translucent aspect of the affected part, with induration, and, according to the length of time it has been in progress, either slight tumefaction, or a greater or less degree of contraction. Further, there is usually close adhesion of the affected part to the pia mater over it, and more or less equivalent change in the pia mater itself. In most cases death occurs at an advanced period of the disease, and hence induration and diminution of bulk are generally observed.

Sclerosis has a remarkable tendency to be limited in certain cases to definite tracts or regions of the nervous centres, and then comparatively rarely transgresses these limits. Thus certain cases (infantile and adult spinal paralysis, general spinal paralysis, and progressive muscular atrophy) are characterised by the limitation of the sclerosis to the anterior cornua of the grey matter of the cord, or more exactly (in many cases) to the groups of large nerve-cells therein situated—involving these parts, it may be, in their entire longitudinal extent, and rarely extending horizontally beyond them; other cases (lateral sclerosis) are distinguished by the fact that the sclerotic change has its special seat in the lateral white columns, which are then usually symmetrically affected in a considerable part of their length; other cases, again (locomotor ataxy) are peculiar in the fact that the sclerosis involves mainly and often exclusively the posterior white columns, or rather perhaps the outer bands of these columns, the fasciculi of Goll in many cases escaping; while sometimes (glosso-labio-laryngeal palsy) the motor nuclei of the medulla oblongata are the special seat of disease; and at other times (disseminated sclerosis) the sclerotic change is scattered irregularly in patches throughout the nervous centres.

A. *Infantile Spinal Paralysis.* (*Infantile Paralysis.*)

Definition.—By these terms, as also by that of 'essential paralysis,' is known a peculiar paralytic affection coming on in young children, with acute symptoms, and for the most part with fever, and ending speedily in rapid atrophy of certain muscles or groups of muscles, and permanent paralysis.

Causation.—Infantile paralysis may, according to the statistics of M. Duchenne (fils), come on at any time from birth up to ten years of age. But the great majority of cases occur during the second year of life. Its causes are obscure; it seems, however, neither to be hereditary nor to be dependent in any degree on privation or other conditions

associated with poverty. Dentition would appear to be largely concerned in its causation, and it has often been observed to follow on measles, 'gastric fever,' and other febrile maladies. Exposure to cold and damp is undoubtedly a common cause of the disease.

Morbid anatomy.—The morbid processes of infantile paralysis concern the spinal cord, the motor nerves, and the organs of locomotion. In the spinal cord the only lesions which are usually observable occupy the anterior cornua. They consist mainly in pigmental degeneration and atrophy of the groups of large cells, which tend ultimately to disappear completely. Herewith, however, is usually associated more or less sclerotic change in the tissues in which these cells are imbedded. The affection is obviously inflammatory, and, although commonly involving the neuroglia as well as the nerve-cells, is sometimes limited to the cells, or to these and the portions of neuroglia immediately surrounding each. Whence Charcot regards the inflammatory process as commencing in the nerve-cells, and as implicating the neuroglia secondarily only. The morbid process affects the various regions of the anterior cornua indifferently, and by no means necessarily symmetrically; it may involve them in patches, or uniformly throughout a considerable vertical extent. The diseased cornua ultimately shrink in proportion to the degree and duration of the morbid process.

The motor nerves are involved secondarily to the spinal cord, and only at a comparatively late period of the disease. They undergo atrophy, the ultimate tubules diminishing in size and tending to lose their myeline sheaths. The muscles which are implicated in the disease shrink rapidly. In the first instance the only obvious and constant change is a diminution in the diameter of the fibres, with some hyperplasia of the cells of the sarcolemma, and, according to some observers, more or less overgrowth of the intervening connective tissue. But even in the early stage a few muscular fibres will often be found to have lost their transverse striation and to have become granular. At a late period of the disease the atrophy of the fibres has become more complete, and they then not unfrequently, but by no means necessarily, present well-marked fatty degeneration. At this time, also, there is often more or less increase of intervening connective tissue, and sometimes a large accumulation of fat. The consequences, as regards the general form and bulk of the muscles, are various: in some cases they appear simply shrunk to an extreme degree; in other cases they are found to retain more or less of their normal size and shape; and occasionally they present a positive increase of bulk, owing to the accumulation between their fibres of adipose or fibrous tissue.

Symptoms and progress.—The onset of infantile paralysis is usually sudden, and marked by more or less intense fever, occasionally attended with convulsions, coma, or other cerebral symptoms. The duration of this febrile attack, which, however, is not always present, varies from a few hours to a couple of weeks. The paralytic condition of the

muscles for the most part comes on quickly and unexpectedly. Sometimes the child is found paralysed on waking up from sleep; sometimes on the subsidence of coma or convulsions, or on the disappearance of some specific fever or of the special fever of the disease, or in the course of that fever. The paralysis increases rapidly from the moment of its first appearance, so that at the end of a day or two, sometimes however after a longer interval, it has attained its maximum degree and extent. Its extent varies of course in different cases: sometimes both arms and legs are uniformly and completely paralysed; sometimes the legs only; sometimes the arms only; sometimes a single extremity; and sometimes, and on the whole more commonly, groups of muscles belonging to one limb or to several limbs. The paralysis is marked from the first by flaccidity of the muscles, and abolition or impairment of reflex excitability. Moreover (and this is a point of capital importance), great diminution or absolute abolition of electrical contractility rapidly supervenes in the affected muscles, so that at the end of five days, or it may be a week, many of the muscles may have entirely ceased to contract under the influence of faradism. Occasionally pain in the back and pain on movement of the limbs would appear to attend the commencement of the disease, but these phenomena form no essential feature of its clinical history, and certainly in a large number of cases are wholly wanting. According to Dr. West, more or less cutaneous hyperæsthesia is present at this time, and may continue for several weeks; but this is far from being a constant phenomenon. Indeed, it may be regarded as characteristic of the disease that absolute paralysis of certain muscles, attended with flaccidity and loss of reflex and faradic contractility, is linked with an almost total absence of pain, retention of cutaneous sensibility, perfect control over the rectum and the bladder, and an entire lack of all tendency to inflammation of the urinary organs, formation of bed-sores, or appearance of cutaneous eruptions. The first stage of the disease characterised by the phenomena which have just been enumerated lasts from two to six months—sometimes, however, a longer, sometimes a shorter time—and is then followed by the second stage, which continues probably for another six months. During this stage more or less amendment generally takes place; certain of the paralysed muscles, and more especially those in which faradic contractility had not been wholly abolished, slowly regain their normal reflex and electrical properties and their power of spontaneous movement; a greater or lesser number, however, of the muscles which had lost their faradic contractility probably remain (singly or in groups, or occupying the whole of one or more limbs) permanently paralysed; and not only remain paralysed, but undergo more or less rapid atrophy. Any improvement, excepting in those muscles which are already in progress of amendment, can scarcely be hoped for after the lapse of eight or ten months from the commencement of the disease.

The changes which now slowly ensue are interesting. In the first place the permanently paralysed muscles, which had already perhaps given indications of shrinking at the end of a month from the beginning of the disease, become rapidly atrophied—generally undergoing much reduction in size, but sometimes retaining more or less of their natural bulk in consequence of overgrowth of interstitial connective tissue and fat. They continue perfectly limp, and wholly impassive under the influence of every kind of stimulus. In the second place, it often happens that the bones of the affected members become arrested in their development, and are consequently at the time of full growth thinner and shorter, sometimes considerably shorter, than they should be. This result has been observed several times by Volkmann in cases in which the primary disease was of short duration, and paralysis had wholly disappeared. In the third place, the paralysed limbs show a striking and permanent diminution of temperature, a diminution which is more marked than in any other form of paralysis. It appears to be connected with a general diminution in the bore of the blood-vessels. In the fourth place, various deformities, mainly of the hand and foot, are apt to ensue. These generally begin to show themselves about the end of the first year, and depend apparently on the unequal degrees in which opposing muscles are affected, and on the predominant action therefore of the healthy or less completely paralysed muscles. Their production is largely aided by the remarkable laxity of the ligaments, which is also usually present in these cases. The most common deformity of the foot is talipes equinus.

Cases of infantile paralysis occasionally depart from the type above sketched. Sometimes the disease lasts for a few days or a few weeks only, and complete restoration to health ensues; sometimes the paralysis, instead of being developed with sudden intensity, creeps on gradually and attains its full development only after some length of time; sometimes, again, the patient suffers from occasional exacerbations or relapses.

Treatment.—In the early stage of infantile paralysis, treatment must be mainly expectant. Salines, laxatives, and other febrifuge medicines may be used with some advantage, and in some cases perhaps counter-irritation, or the abstraction of blood by leeches or cupping-glasses applied in the course of the spine. The patient should, of course, be kept absolutely at rest, and careful attention should be bestowed in order to relieve symptoms and obviate the occurrence of complications. But after all febrile symptoms have passed away, and all acute inflammatory mischief has subsided, which will probably be at the end of three or four weeks, it will be necessary to adopt measures to promote the restoration of those muscles which are capable of restoration. The affected muscles may be divided into three categories: first, those which, though paralysed, have their faradic contractility but slightly affected; second, those in which the

faradic contractility is much enfeebled or abolished, but which respond to galvanism; and, third, those which fail to re-act to any form of electricity. Muscles belonging to the first category tend to recover completely, independently of all treatment; but nevertheless the periodical application of faradism or galvanism to them tends to hasten their recovery. Muscles belonging to the second category for the most part undergo more or less considerable atrophy, which may continue to progress for many months, and may result in their permanent emaciation and weakness, even though complete restoration of the nerves and nerve-centres in relation with them ultimately takes place. In these cases the long-continued and systematic use of galvanism, especially if it be commenced early, will often serve to arrest the wasting of the muscles, and ultimately to bring them back to the condition of health. The prospects as regards the muscles in the third class are much more gloomy. They invariably waste rapidly, and in a very large proportion of cases fail absolutely to undergo any kind of improvement. Still, even here, the solicitous and long-continued use of galvanism sometimes succeeds in effecting a more or less important improvement. It need scarcely be said that the longer recourse to electric treatment is delayed, the less is the chance which it affords of benefit. Nevertheless, a case recorded by Duchenne, in which the complete restoration of many muscles of the arm (which had been atrophied and had lost all electric contractility for a period of four years) was effected by means of faradism applied periodically during the space of two years, proves that we need not despair even when circumstances seem most adverse. An important result to be derived from electricity, even when it fails to cure absolutely, is the prevention of the deformities due to the unequal action of antagonistic muscles. In conjunction with electricity, other measures may be used to improve the condition of the muscles; among which may be enumerated exercise, rubbing, shampooing, baths, and mechanical measures to obviate the tendency of certain muscles to contract and cause deformity. Iron and other tonics are, in a certain sense, useful. Strychnia has been largely recommended.

The treatment of the results of infantile paralysis belongs to the surgeon.

B. *Adult Spinal Paralysis.*

Both Duchenne and Charcot have published cases (and, indeed, it seems probable that such cases are not uncommon) in which adults have been attacked with disease resembling in all essential particulars infantile paralysis. The recorded cases show: that it comes on in adults, as in children, with more or less severe febrile symptoms, lasting, it may be, for a few days, and attended with or followed by motor paralysis of a greater or lesser number of voluntary muscles, but

without implication of cutaneous sensibility, loss of control over the rectum or bladder, or tendency to the formation of bed-sores; that the muscles are flaccid, incapable of excito-motor action, and tend rapidly to lose their faradic contractility, and to waste; and that, after the paralysis has reached its highest degree, more or less amendment takes place in some of the muscles. It not uncommonly happens in the case of the adult, as probably happens also in the child, that pain in the spine, with forward curvature, and some degree of pain in the limbs, attend the onset of the disease. The chief point in which the history of the disease in adults differs from that of the disease in children is the necessary absence from it of all mention of the various deformities resulting from defective development; the bones do not become relatively short; and deformities connected with the joints are less extreme.

The pathology and treatment are the same as in the infantile disorder.

C. *General Spinal Paralysis.*

Definition.—The malady referred to under the above name is in the third edition of Duchenne's treatise 'De l'Electrisation Localisée,' entitled 'paralysie générale spinale antérieure subaigue.' It is characterised by more or less general paralysis with wasting and flaccidity of the muscles, and marked loss of electrical contractility, unattended with implication of the rectum or bladder, or with brain-symptoms, and tending in many cases to end in recovery.

Causation.—The causes of this affection are not known. It has been referred to exposure to cold and wet. There is no reason to regard it as hereditary. It comes on mainly between the ages of thirty-five and forty.

Morbid anatomy.—The anatomical lesion which underlies the phenomena by which the presence of general spinal paralysis is revealed has not yet been ascertained; but there is reason to believe that the disease resembles acute spinal paralysis of children and of the adult in the facts, that the parts which are specially implicated are the anterior cornua of the grey matter of the cord, and that the lesion is inflammatory.

Symptoms and progress.—The paralytic phenomena may begin in the upper extremities, and, thence travelling downwards, gradually become general; or they may take their origin in the legs, and thence extend upwards to the rest of the body. The latter course is by far the most common. In that case the patient first experiences weakness in one or both lower extremities—if in both, with predominance in one of them. Should a careful examination be made at this time, it will probably be found that the flexors of the foot on the leg suffer first and most severely, then those of the thigh on the trunk, and

subsequently the extensors of the leg upon the thigh. The paralysis increases progressively until the patient can neither stand nor walk, and ultimately his limbs become entirely motionless. No trembling, no convulsive movements, no inco-ordination, no rigidity or contraction attends the progress of the disorder. The affected muscles are, however, flaccid from the beginning, and very rapidly, though somewhat irregularly, lose their faradic contractility, until at length, as sometimes happens, it ceases totally. Very soon, also, the paralysed muscles waste—the atrophy not affecting single muscles, as in progressive muscular atrophy, but involving all the paralysed muscles coetaneously, so that the limbs shrink in their whole extent, and acquire a resemblance to those of a mummy. The surface at the same time is apt to get cold and livid. The paralysis remains limited, for a longer or shorter time, to the lower extremities. In some cases the hands begin to lose power ere the affection of the lower limbs is five or six weeks old. In other cases the upper extremities are not involved until after the lapse of several months or years. But when once the paralysis has reached them, it pervades them progressively and rapidly, first attacking the extensors of the fingers, and then taking much the same course that it previously took in the lower extremities. Subsequently the muscles of the trunk and of the head and neck become implicated. Generally the paralysis is more marked on one side of the body than the other. If the paralysis be not arrested in its course, or do not undergo amendment, it extends at length to the muscles supplied by the nerves of the medulla oblongata; whence result difficulty of articulation, difficulty of deglutition, and respiratory trouble.

The progress of the disease may be uniform and continuous, or it may be interrupted from time to time by long intervals, during which the symptoms remain in abeyance or undergo more or less obvious amendment. Sometimes the patient recovers completely, only to have a relapse at some subsequent period. Not unfrequently patients succumb, especially under the effects of implication of the medulla oblongata, or from syncope. On the other hand, many persons recover absolutely, even after they have suffered from the disease for some months or years, and even after the wasting of the limbs and the loss of faradic contractility have persisted for a considerable period. The duration of the disease varies from a few months to many years.

Among the more striking characteristics of general spinal paralysis are: its insidious origin, unattended with febrile or other symptoms; its progressive invasion of all the voluntary muscles, and the rapid loss of their faradic contractility and bulk; the wasting of the limbs in mass, and not muscle by muscle; the absence of all paralytic tremblings and convulsive movements; the retention of cutaneous sensibility, of control over the emunctories, and of the mental faculties; and the tendency which is manifested to ultimate recovery.

Treatment.—In treating cases of general spinal paralysis, it must

always be remembered that many cases ultimately do well quite irrespective of medical treatment. There is, indeed, but little to be done medicinally. The usual round of drugs which are employed in nervous disorders may be tried. With more hope of benefit faradism or galvanism, periodically and persistently administered, may be had recourse to for the treatment of the paralysed muscles.

D. *Progressive Muscular Atrophy.* (*Wasting Palsy.*)

Definition.—This disease is characterised by progressive atrophy commencing in certain muscles, usually those of one hand, next involving, as a rule, corresponding muscles on the opposite side of the body, and gradually spreading to other muscles of the limbs and trunk.

Causation.—Progressive muscular atrophy occurs both in children and in adults, and principally in males. Its causes are obscure. It has been traced apparently to exposure, to excessive bodily or mental exertion, and to violent emotions; but mainly it appears to be an hereditary affection. In almost all cases of its occurrence in children which have come under Duchenne's observation, it appears to have been hereditary.

Morbid anatomy.—The pathology of this affection differs little from that of infantile spinal paralysis. The parts affected are in this case, as in that, the anterior cornua, the motor nerves which emerge from them, and the muscles which these nerves supply. The affection is limited in many cases to certain groups of large cells—the cells undergoing the various forms of degeneration which have already been described, and finally, it may be, disappearing altogether; or it may include with these changes a greater or less amount of sclerosis of the contiguous portions of the anterior cornua, and corresponding atrophy of the nerve-tubules. In the early stage the blood-vessels of the affected parts are enlarged and their walls thickened. The anterior nerve-roots, which are connected on the one hand with the diseased portions of cord, on the other with the affected muscles, undergo similar changes to those which have been described in connection with infantile paralysis; but the degeneration is never so extreme or general, nor does it distinctly manifest its presence until the muscular atrophy has made considerable progress. The muscular change consists essentially in a mere attenuation of the muscular fibres, with more or less proliferation, of an abortive character, of the cells of the sarcolemma. Granular and fatty degeneration, with disappearance of the transverse striæ, may supervene at a late period of the disease; but it is merely a secondary phenomenon, and has no special significance. Hypertrophy of the connective tissue investing the muscular fibres, and accumulation of fatty matter in the same situation are not uncommon. The affection of the nerve-centres is distinctly inflammatory, and precedes and causes the lesions of the motor nerves and muscles.

Symptoms and progress.—Progressive muscular atrophy, unlike acute spinal paralysis, comes on insidiously. Its advent is unattended with fever or other obvious constitutional disturbance. Indeed, the first indication of its presence is, almost without exception, wasting and loss of power of some muscle or group of muscles. The muscular atrophy may commence at any part, but most commonly commences in the hand, especially the right hand, whence it spreads first to the corresponding muscles of the opposite side of the body, then to those of both forearms, and presently becomes distributed with more or less irregularity, but symmetrically, throughout the trunk and lower extremities.

When it takes the orthodox course, it is the muscles of the ball of the right thumb which are usually first affected. Then the muscles of the hypothenar eminence and the interossei suffer; and the hand acquires an almost pathognomonic claw-like form. Next the muscles of the forearm waste, with some irregularity as to the order of their wasting. Subsequently the muscles of the upper arm and shoulder suffer, the triceps for the most part retaining its normal condition longer than the others. The muscles of the trunk usually suffer coetaneously with those of the upper arm, but are involved irregularly. The usual order, however, of their involvement is, according to Duchenne: first, the trapezius (of which the lower portion suffers earliest); then successively the pectorals, the latissimus dorsi, the rhomboidei, the levator anguli scapulæ, the extensors and flexors of the head, the sacro-lumbalis, and the abdominal muscles. When the atrophy has extended thus far, the muscles of respiration and deglutition usually become affected. The clavicular portion of the trapezius is the last of the muscles of the trunk to succumb. The lower extremities are involved subsequently to the arms and trunk, but their muscles are apt to suffer equally in degree with those of other parts. The muscles which are earliest affected are the flexors of the foot on the leg, and those of the thigh upon the pelvis.

Not unfrequently progressive muscular atrophy first shows itself among the muscles of the trunk, attacking sometimes the pectorals, sometimes the serratus magnus, sometimes the spinal muscles, sometimes those of the abdomen, and thence extending to the other trunk-muscles and to those of the extremities. Its commencement in the lower extremities is exceedingly rare.

A curious circumstance is pointed out by Duchenne, namely, that when progressive muscular atrophy attacks young children, its invasion is marked by the effacement of some of the muscles of expression; that their aspect consequently becomes more or less idiotic; that their cheeks become flaccid and their lips large and pendulous; and further, that it is only subsequently, and it may be after the lapse of years, that the muscles of the trunk and those of the limbs partake in the atrophic change.

In addition to the fact that progressive muscular atrophy is a disease of an essentially chronic character, and tends from insignificant beginnings slowly to involve a large number of the voluntary muscles of the limbs and trunk, it presents several other remarkable features which help to distinguish it from acute spinal paralysis and from other spinal disorders to which in many respects it has a more or less close resemblance. First, the enfeeblement of the affected muscles does not precede the atrophy; it follows upon the atrophy, is due to it, and is proportional to it; and indeed the motor power is not wholly lost until a very late stage of the disease, or until the muscles have undergone complete atrophy. Second, the faradic contractility of the affected muscles remains unimpaired, or rather diminishes only in proportion to the effacement of the constituent muscular fibres, and never disappears absolutely until voluntary power is wholly lost, namely, when atrophy has reached its extreme degree. Third, a very common feature of the malady is the presence in the affected muscles of irregular vibratile movements of the individual fibres, which may be seen and felt, and which, when superficial muscles are under observation, give to the eye an appearance as though innumerable slender worms were in active parallel progression under the skin. These fibrillar oscillations are occasionally so violent as to cause movements of flexion and extension in the fingers or other parts. They are not constant, and are usually evoked either by the patient's effort to bring the muscles into action, or by tapping, pinching, or otherwise exciting them. They are not peculiar to progressive atrophy, although they commonly attend it.

Characters which are common to this affection and to spinal paralysis are a lowering of the temperature of the affected limbs, absence of spinal or neuralgic pain and of tendency to the formation of bedsores or to the appearance of cutaneous eruptions, and retention of control over the emunctories. Cutaneous sensibility is generally retained. It is well to bear in mind that the wasting of muscles is often concealed by the presence of an excess of subcutaneous fat, and that hence the true condition of things can often be determined only after careful investigation.

The course of progressive muscular atrophy is always slow and irregular. It may appear in some of the muscles of the face or hand, and years may elapse before it extends either to neighbouring muscles or to the corresponding muscles of the opposite side of the body. Or it may invade the muscles of the arms or legs with comparatively great rapidity, and then a long interval of quiescence may ensue. It may even become arrested in its onward progress never to be reawakened. Most commonly, however, it advances either uniformly or by fits and starts, until the patient becomes utterly helpless. The duration of the affection is very various. In many cases the patient survives for eight or ten years, or even for twice that period. And, indeed, there is nothing to interfere with the duration of life unless the muscles of

respiration or those of deglutition get involved. If this happen, the patient is liable to be cut off either by inability to swallow, by choking, or by difficulty of breathing, and the pulmonary complications which are then so apt to ensue. If these important parts are early implicated, the patient may succumb within two or three years from the commencement of his malady.

Treatment.—The treatment of progressive muscular atrophy calls for no special observation. It may be the same that has been recommended for the chronic stages of acute spinal paralysis, and especially the systematic use of electricity to the affected muscles. M. Duchenne, who strongly urges the beneficial influence of this treatment, says that the progress of the disorder may sometimes be arrested, and muscles not too far advanced in atrophy may occasionally be restored by it. He prefers faradism applied in turn to each affected muscle, but recommends also the concurrent use of the direct current—constant or interrupted. He specially advocates the solicitous treatment of those muscles which are the most important either for the maintenance of life or the usefulness of the limbs. The following are some of the rules which he lays down. The more a muscle is atrophied, its contractility diminished, and its sensibility benumbed, the longer it should be subjected to stimulation, the more intense should be the current and the more rapid its intermissions. But when sensibility returns it is prudent to diminish the intermissions, and abate the intensity of the current, and even to abridge the number of sittings, lest unmanageable neuralgia be provoked. No sitting should be prolonged beyond ten or fifteen minutes, and rarely more than one minute should be given to each muscle.

E. *Lateral Sclerosis.*

Definition.—The affections which it is intended to comprise under the above designation are those to which M. Charcot applies the names of 'amyotrophies spinales deutéropathiques,' and 'sclérose latérale amyotrophique.' They are essentially characterised: first, as regards their morbid anatomy, by a sclerotic change affecting the lateral white columns of the cord symmetrically and in their whole length, and in the majority of cases extending thence so as to involve more or less of the anterior cornua, and occasionally of the sensory elements; second, as regards their clinical phenomena, by paralysis of the limbs with rigidity and tendency to contract, associated with more or less wasting of muscles and sometimes with neuralgic pains.

Causation.—The causes of the inflammatory process which induces sclerosis in the lateral columns are various. This lesion is a common consequence of effusion of blood into the substance of the cerebrum, or of the presence of a patch of softening, and equally follows any destructive lesion of the crura, pons, medulla oblongata, or spinal cord.

It is occasionally of idiopathic origin, under which circumstances, according to Charcot, it does not appear to be hereditary, seems to occur more frequently in women than men, and mainly between the ages of twenty-six and fifty. Some patients refer the disease to the influence of cold and wet, and some to injury.

Morbid anatomy.—The simplest form of lateral sclerosis, both pathologically and clinically, is that which occurs secondarily to some cerebral or other lesion. We have already drawn attention to the fact of its occurrence under the conditions here specified. In old cases of cerebral hemorrhage or softening, a tract of sclerosis, the upward limit of which has not yet been determined, may often be traced along the crusta of the corresponding crus cerebri, through the pons, into the anterior pyramid, which may be involved in pretty nearly its whole horizontal and vertical extent, and along the decussation to the opposite side of the cord, in which it occupies almost exclusively the lateral white column, being separated, however, from the surface by a persistent lamina of still healthy white matter. In the neck the sclerotic change may extend from the outer angle of the anterior cornu in front to the posterior nerve-root behind. But in its passage down the cord it occupies a smaller and smaller space, both relatively and actually, and at the same time limits itself more and more to the neighbourhood of the posterior cornu and the nerve-fibres springing from it. Thus in the middle of the dorsal region its anterior limit corresponds to a transverse line drawn through the commissure, while in the lumbar enlargement it occupies only the posterior fourth of the lateral column. Occasionally the fasciculi of Türck (narrow tracts of white matter situated on either side of the anterior median fissure, and belonging apparently to the same system as the lateral columns) share in the morbid change. It is very rare, however, for any other part to be implicated.

Lateral sclerosis of idiopathic origin affects identically the same tracts, but in this case both sides of the cord are as a rule simultaneously implicated, and the morbid process presents a symmetrical character. Another important distinction between the secondary and idiopathic forms of the lesion is afforded by the fact that in the former the morbid process rarely extends horizontally beyond the lateral white columns, while in the latter such extension constitutes the rule. When lateral extension takes place, it is seldom if ever general; it comparatively rarely involves the sensory tracts of the grey matter of the cord, the posterior columns, or the posterior roots; but almost always it is the anterior cornua with their groups of large cells which suffer. The anatomical lesion, in fact, which constitutes the basis of the group of affections last considered, becomes superadded to the primary lesion of the lateral columns. This lateral extension, however, does not occur in all situations equally, nor does it take place here and there indifferently. It is almost invariably most advanced in the cervical portion

of the cord, and diminishes gradually thence downwards. It comes on also, though at a comparatively late period of the disease, in the medulla oblongata, leading then to special implication of the nuclei of the spinal accessory, hypoglossal, vagus, and facial nerves. Both the naked-eye and the microscopic characters of sclerosis have already been sufficiently considered. It need only be added that the sclerotic process in the anterior cornua, which is associated with lateral sclerosis, is undistinguishable from that which occurs primarily in them; and that it is followed sooner or later by the same secondary changes in the motor nerves and voluntary muscles which have already been described in connection with infantile paralysis.

Symptoms and progress.—The symptoms specially referrible to disease of the lateral columns are gradually developing paralysis of the muscles with which the affected tracts are in relation, attended with more or less violent tremors during the attempt to perform voluntary movements, and gradually increasing rigidity, often coming on in paroxysms, and easily induced by any kind of irritation. The rigidity is at first general, and induces extension of the limbs; but subsequently the flexors tend to overpower the extensors, and the limbs become more or less strongly flexed. Besides the sudden spasmodic contractions just referred to, the paralysed muscles are liable to more or less prolonged paroxysms of convulsive trembling. The affected muscles do not waste, and they retain their faradic contractility and reflex excitability unimpaired. Nor is there loss of sensation or pain.

These phenomena are often observed in the lower extremities of persons who are paraplegic from pressure, or disease involving a limited length of the cord, and are then referrible to the descending lesion of the lateral columns which is so apt to ensue under these circumstances. Rigidity and contraction, due to the same cause, are also of common occurrence in the paralysed limbs in cases of old cerebral mischief. In idiopathic sclerosis, however, of the lateral columns, the extension of disease horizontally into other regions of the cord necessitates the superaddition of other symptoms to those which have just been enumerated; these are, occasionally more or less pain, numbness or tingling from implication of the posterior roots or posterior horns, but especially more or less rapid wasting of some of the paralysed muscles from involvement of the anterior cornua. The phenomena of the idiopathic affection are, of course, largely dependent, as regards their gravity and distribution, on the longitudinal extent to which the lateral columns are implicated, and on the destination of the motor nerves whose nuclei suffer from its horizontal extension.

In most cases the idiopathic disease comes on insidiously, without fever or premonitory symptoms other than perhaps some numbness or tingling in the limb over which paralysis impends. The arms are usually first implicated, first one, probably, and the other after a short interval. They become enfeebled and more or less emaciated; the

enfeeblement, however, precedes the emaciation, or goes along with it, and does not, as in wasting palsy, follow it. Moreover, in lateral sclerosis, the paralysis and wasting affect all the muscles of the affected limbs simultaneously instead of, as in the other case, creeping from muscle to muscle. Further, the shrinking muscles are liable, as are those of wasting palsy, to fibrillar vibrations, and retain like them their faradic contractility; and, moreover, so long as any voluntary motor power remains in them, their movements are generally attended with more or less violent trembling. But, in addition to the phenomena above enumerated, the emaciated and paralysed limbs soon become rigid and contracted, and, as a consequence of this contraction, deformed. Each arm is kept closely applied along the side of the body, and the shoulder resists when we attempt to abduct it; the forearm is semiflexed and pronated, and cannot be extended or supinated without the use of considerable force, and exciting pain; the hand is flexed on the forearm; and the fingers also are flexed. In the further progress of the case emaciation tends to become extreme; and at length with the atrophy of the muscles their capability of contracting under the stimulus of electricity may fail, and their rigidity and contraction may to some extent disappear. In some cases the muscles of the neck get rigid, like those of the arms, and the patient has pain and difficulty in moving his head.

The atrophy of the arms in this affection is far more rapid than in progressive muscular atrophy, mainly, no doubt, because all the muscles are involved at one and the same time; and at the end of four, five, or six months, or at the outside a year, the emaciation is as extreme as we observe it to be in cases of progressive muscular atrophy which have been in progress for several years. From six months to a year or more after the commencement of the disease, the lower extremities generally first give signs of involvement. The patient complains of numbness, tingling, and loss of power in them; but in their case there is very seldom any atrophy, owing evidently to the fact that the motor nuclei of the dorsal and lumbar regions are scarcely ever involved. The paralysis of the legs, however, makes rapid progress; the patient soon cannot walk without being supported on both sides; and before long all voluntary movement ceases in them. Long ere the paralysis is complete the legs are liable to spasmodic contractions, which come on without obvious cause, and are readily excited by any form of stimulus; they become suddenly rigid, sometimes flexed, sometimes extended, and are apt to remain for some time in that condition. These spasms are chiefly liable to occur when he attempts to walk, and then especially cause powerful adduction of the thighs, with more or less marked extension at the different joints. The extension at the ankle-joints imparts to the feet the attitude of talipes equino-varus. Associated with the spasms are more or less violent tremblings, which add to the patient's difficulty of walking. When the paralysis

is complete, the rigidity becomes permanent, the legs being in some cases extended, in some flexed, and exceedingly difficult to bend or straighten. At this time prolonged paroxysms of spasmodic trembling can often be readily induced. After a while, but this is a remote and comparatively rare event, the muscles of the legs may undergo atrophy like those of the arms; and with the advance of this atrophy the rigidity gradually relaxes. There is no paralysis of bladder or rectum, or tendency to the formation of bed-sores.

While the legs are becoming paralytic, or it may be after arms and legs have lost all power of voluntary movement, another series of phenomena gradually supervenes, referrible to implication of the motor nuclei of the medulla oblongata. These are: paralysis of the lips and face; paralysis of the tongue; paralysis of the soft palate; and paralysis of the parts to which the vagus is distributed—a group of phenomena which will hereafter be considered as a distinct affection, under the name of 'glosso-labio-laryngeal palsy.'

The progress of idiopathic lateral sclerosis is not always in accordance with the above sketch. Sometimes it commences in the lower extremities, sometimes it is limited to one arm or leg, or assumes a hemiplegic form, and rarely it first reveals itself by implication of the nerves of the medulla oblongata.

The prognosis of the disease, at all events when it presents distinctive characters, is exceedingly gloomy. Its various stages follow one another surely and rapidly, and death usually takes place within one, two, or three years from the commencement of paralytic symptoms. The contrast in this respect between lateral sclerosis and wasting palsy is very striking. Death is generally due to accidents connected with involvement of the nerves of the medulla oblongata.

Treatment.—It is needless to endeavour to lay down rules for the treatment of this disease. All that can be done is to attend to the general health, to resort to electricity, friction, and such-like measures, and to obviate as far as possible the various discomforts and dangers to which the patient is exposed.

F. *Tabes Dorsalis* (*Locomotor Ataxy.*)

Definition.—By the above terms is understood a peculiar affection characterised: anatomically by sclerosis of the posterior columns of the cord; clinically by loss of co-ordinating power in the lower extremities, gradually increasing in degree and extent, and generally sooner or later involving the upper extremities and other parts. Various nervous lesions, which need not now be specified, are frequently associated with the muscular inco-ordination.

Causation.—The causes of locomotor ataxy, like those of most other affections of which a sclerotic condition of the nervous centres forms the anatomical basis, are exceedingly obscure. The disease has

been referred by some to sexual excesses, by some to exposure to cold and wet, by some to over-exertion, injury or shock. But little more can be said positively than that in many cases some one of these various conditions has preceded the occurrence of the nervous phenomena. In the greater number of cases, however, no cause whatever can be assigned. The disease is occasionally hereditary, or runs in families, and, further, seems not unfrequently to be associated in families with insanity, epilepsy, and other affections of the nervous system. It seldom occurs in women; and generally first makes its appearance between the ages of twenty and forty-five. It sometimes, however, comes on at an advanced period of life, and sometimes about the age of puberty.

Morbid anatomy.—The specific lesion of locomotor ataxy is sclerosis of the posterior columns of the cord: both columns, as a rule, being equally affected, and the morbid change being most advanced in the lower part and diminishing from below upwards. In many cases the posterior columns are involved in their whole horizontal extent. But it is shown by Charcot that such an amount of disorganisation is unnecessary for the production of the characteristic symptoms of the disease; that in some cases the posterior pyramids remain perfectly healthy; and that, in fact, the tracts whose lesions induce ataxic symptoms are two narrow bands of white matter, lying, one on each side, between the inner and posterior aspect of the posterior cornu and nerve-roots on the one hand, and the posterior pyramid on the other. The sclerotic change occurring in these parts calls for no specific naked-eye or microscopic description; the affected columns become indurated, grey, and translucent, in the early stage a little swollen, but at a later period notably diminished in bulk. The disease, however, rarely remains strictly limited to the tracts which are its primary seat; in a large number of cases (as has been already indicated) the posterior pyramids become involved; and generally the internal radicular fibres of the posterior roots of the nerves, and more or less of the adjoining parts of the posterior cornua get implicated to some extent. Occasionally, also, the disease invades the lateral columns, and occasionally even reaches the anterior cornua: not, however, Charcot thinks, by gradual involvement of all the intermediate tissue, but by extension along the internal radicular fasciculi.

Symptoms and progress.—The invasion of locomotor ataxy is sometimes quite sudden; in other words, impairment of co-ordinating power is the first symptom to declare itself. In the great majority of cases, however, the specific characters of the disease are only revealed after the patient has suffered for an indefinite time, sometimes many years, from premonitory symptoms. These are very various, but many of them are full of significance, and most belong equally to the fully declared disease. The more important of them are as follows:—First, *Pains.* These are of various kinds and are referrible to different parts. The

most common are momentary sharp shooting pains, following the course of certain nerves, for the most part connected with the trunk or lower extremities. An erythematous or vesicular eruption sometimes appears in the area of distribution of the affected nerves. Another variety of pain is of a boring or stabbing character, and is generally limited to certain definite regions in the neighbourhood of the joints or along the back; and its occurrence is usually associated with hyperæsthesia of the same parts. A further variety is of a constrictive character; it mostly affects the trunk, but may involve the limbs or any part of them. These various forms of pain are often associated. The last of them is more or less persistent; but the others occur in momentary twinges, and their continuance is usually effected by a more or less rapid succession of such twinges. Sometimes they come on at irregular and long intervals, and then not unfrequently continue by successive paroxysms for several hours or several days; sometimes they recur many times daily; sometimes they are constant, and wear the patient out by their unceasing severity. They are generally worst at night-time. *Pains referrible to the viscera* are also not unfrequent. Among them may be included pain in the bladder attended with frequent desire to make water, pain in the urethra excited by the act of micturition, and pain in the rectum as if the bowel were being distended, associated with violent tenesmus. The most important and characteristic of them, however, are attacks of gastralgia of extreme intensity, attended with vomiting, faintness, deranged action of the heart, and an extreme sense of illness. The pains in the stomach shoot to the back, about the abdomen, and in various other directions. Second, *Paralyses of motor and sensory nerves.* These are sometimes temporary, and apt to recur at intervals, sometimes permanent. Among the least common of them are hemiplegia, and anæsthesia in the area of distribution of the fifth pair; among the most common, paralysis of the external rectus or of the internal rectus or other muscles supplied by the third pair. Third, *Affections of the eye and ear.* We have already referred to the fact that the patient may have an internal or external squint, or ptosis. It may be added that extreme contraction of the pupil is a marked feature of the disease; that the pupils are sometimes unequal; and that the contracted pupil is apt to dilate under the influence of the attacks of pain to which the patient is subject. But besides these conditions, which are obvious to casual observers, there are others of yet greater significance and importance. The patient's eyesight in many cases becomes defective: he sees double; or his vision gets dim or indistinct, and he cannot distinguish small objects or the contours of objects so clearly as he formerly did; or his field of vision becomes contracted, limited perhaps to one side; or there is some failure in the power of distinguishing colours—he recognises yellow and blue, but fails to distinguish red and green, and the various secondary tints in the production of which these colours are concerned;

or these various conditions are associated in a greater or less degree. These affections of the eyesight tend to increase slowly, and at length culminate in absolute blindness. They are due to progressive grey atrophy of the optic disc, revealed ophthalmoscopically by chalkiness and opacity, with absence of the marginal rosiness of tint, and inability to trace the trunk-vessels of the retina as they sink into the substance of the optic nerve—they seem to terminate abruptly. The atrophy, according to Charcot, is due to a change occurring in the optic disc (identical with that which goes on in the posterior columns of the cord) and gradually extending backwards along the optic tract as far at least as the corpora geniculata. Headache referred to the back of the head and forehead, and neuralgic pains in the course of the branches of the fifth pair, and in the eyeball, frequently attend the above visual legions. Deafness in one or both ears is not uncommon. Fourth, *Affections of the joints.* These are of occasional occurrence; they are observed mainly in the knees and hips, sometimes in the shoulders. They consist in rapid effusion into the joints and tissues which surround them, taking place with little or no pain or fever, and usually followed at the end of some weeks or months by restoration to health. Occasionally they end in erosion of the ends of the bones, or disorganisation of the joints, followed after a time by dislocation. Fifth. A curious early symptom has been pointed out by Westphal. When healthy persons sit with the legs pendulous, a sharp tap on the ligamentum patellæ causes sudden contraction of the quadriceps extensor, attended with a corresponding forward movement of the foot. This is due to reflex action. In cases of tabes dorsalis this phenomenon is absent almost without exception during the whole course of the disease, even in its earliest stages and before symptoms of inco-ordination have manifested themselves. Lastly, among other occasional precursory symptoms may be enumerated: nocturnal incontinence of urine; spermatorrhœa, sometimes attended with erection and voluptuous sensations, sometimes occurring independently of erection or of orgasm; a peculiar aptitude for repeating the sexual act many times within a short period; and, lastly, a permanent acceleration of pulse, attended, according to M. Eulenburg, with habitual dicrotism.

The explanation of the phenomena which have just been enumerated is for the most part obvious. They are dependent on the progress and distribution of the morbid process which is going on in the nervous centres, but which has not yet destroyed, sufficiently to cause obvious inco-ordination, those portions of the cord which minister to the co-ordinate actions of the lower extremities. Thus the various forms of neuralgic pain and cutaneous eruption are due to implication of the intra-rhachidian portions of the sensory nerve-roots; the affections of the eyes and ears are referrible to involvement of the ophthalmic and auditory nerves, or their nuclei; and there are good grounds for believing that the lesions of the joints are the conse-

quence of implication of the anterior cornua; and that various phenomena, such as those presented by the pupils and those connected with the action of heart and character of the pulse, are of sympathetic origin.

The so-called 'premonitory' symptoms are in truth an integral part of the disease, and if recognised may be taken as sure evidence of the insidious progress of those central organic lesions which ultimately induce the proper ataxic phenomena. Some one or more of these premonitory symptoms may continue for years before the occurrence of obvious ataxia; the disease may even stop short with them; but in many cases those which first made their appearance undergo gradual aggravation, others become superadded to them, and presently the ataxic phenomena supervene and become mingled, as it were, with them. In other cases, again, want of co-ordination in the movements of the lower extremities is the very first indication of nervous disease, and various of the phenomena hitherto spoken of as prodromal appear as complications only during its later progress.

The earliest of the special phenomena of locomotor ataxy is the gradual supervention of a certain difficulty in walking, frequently associated with more or less numbness and tingling of the toes and feet. The difficulty is peculiar in its character; it does not consist in any loss of muscular power or any inability to take long walks without discomfort or fatigue, but in a certain clumsiness or uncertainty which manifests itself especially when the patient first rises from his seat, or when he is endeavouring to avoid obstacles, or when he attempts to turn suddenly on his heels, or to go upstairs. It becomes especially obvious in the dusk; and, indeed, the very first indication of disease is sometimes the difficulty which the patient experiences in walking in the dark. Under all these circumstances, his movements become more or less tumultuous, and there is an obvious difficulty in the maintenance of his equilibrium. This difficulty becomes evident in the most marked manner, even in the very earliest stage of the disease, when the patient is made to stand blindfold with his feet together. At once he begins to totter and to sway, and, unless he opens his eyes or is supported by others, soon falls to the ground. With the progress of the disease the movements all become more tumultuous, and the difficulty of progression increases proportionately; the patient now perhaps experiences considerable difficulty in assuming the erect posture; in endeavouring to attain it his legs jerk here and there, apparently urged by an uncontrollable impulse, and he has to resort to a stick or the arm of his chair, or to a friendly hand to aid him in his efforts. When once he is on his legs, he pauses for a while to balance himself, and then starts off with his body bent forwards and his legs apart. Every movement of his legs is now tumultuous; the leg with which he steps out is lifted from the ground and thrown forwards and upwards with needless suddenness and violence, and is then brought down with equally unnecessary force, and even when on the ground still presents a tendency to

jerk, which may be continued even while the other leg is in its turn executing its series of awkward progressional movements. The patient continues to walk in this manner either without assistance, or with the aid of a stick or chair, or between a couple of friends, according to the stage which his loss of co-ordinating power has reached. But if he be able to walk alone or with a stick, his movements usually become a little less wild after he has taken a few steps; and he may continue to walk with excessive violence of movement, no doubt, and with short hurried steps and the body thrown forwards, but nevertheless with considerable power and efficiency. Patients in this state will sometimes walk ten, a dozen, or twenty miles at a stretch, with comparatively little fatigue; but in some cases the mere violence of his muscular movements involves such rapid exhaustion of power that the sufferer can scarcely do more than walk across the room. A time, however, comes sooner or later in which his want of control over the movements of his lower extremities becomes so extreme that it is absolutely impossible for him to make a step or two consecutively or even to stand. His legs, when he attempts to use them, move, as Trousseau observes, like those of a puppet or a marionette. Thenceforward he is confined to his chair or bed. It is a remarkable fact, for the due appreciation of which we are indebted to Duchenne, that the muscles of the affected limbs retain, as a rule, their bulk, their tonicity, their electrical contractility, and their strength, little if at all impaired, not only so long as the patient can walk or stand, but long after his limbs have become absolutely helpless. And often, at a time when the patient cannot rise from his chair or stand, he can freely execute movements of extension and flexion as he sits or lies, and successfully resist all manual efforts on the part of his physician to extend or flex his legs. The numbness and tingling to which reference has already been made generally persist, and for the most part increase in degree and extent, and always from below upwards. There is sometimes total abolition of cutaneous sensibility in the feet, and there may be some impairment of it extending even to the abdomen. Occasionally it is absent. This impairment of sensibility gives to the patient the impression, that his feet are swollen and soft, or that they are enveloped in some thick soft covering, and when he stands or walks that he treads on sponge or wool, or some other yielding and elastic material, or even that he treads on air. However great the loss of tactile sensibility, that which takes cognisance of differences of temperature usually survives to the last.

The symptoms of ataxy do not generally remain limited to the lower extremities. In most cases, sooner or later, numbness, at first perhaps occasional but after a while permanent, is complained of in the tips of one or two of the fingers—generally the little and ring fingers; and the numbness may remain thus limited, or may gradually involve more and more of the hand and arm, always, however, continuing most highly developed in the parts which were first attacked. In associa-

tion with this, more or less clumsiness in the movements of the fingers, and probably of the hands and arms, may be observed. The patient experiences considerable difficulty in performing all delicate manipulations; he cannot pick up a pin lying upon a hard smooth surface; he cannot button or unbutton his clothes or tie a bow, especially if he be unable to direct the operation with his eyes; in grasping a pen or any other similar object which is handed to him he first opens his hand wide and then closes it with more or less violence upon it, entirely failing to execute those delicate combined movements which are necessary to the precision of his performance, and which impart such grace to the natural movements of the hand. The same clumsiness is observable in his efforts to transfer the object from one hand to the other, and if it be a pen, in acquiring that hold of it which is proper for writing. Further (and this is a defect belonging equally to the lower extremities, but less readily recognised in their case), the patient is unable without the assistance of his eyes to judge of the position of his hands, or so to adjust the action of his muscles as to determine accurately the direction or extent of the movements of his arm. Hence, if his eyes be closed, he cannot if he wishes to clasp his hands bring them together with any certainty; they are brought towards one another at different elevations, or one in front of the other, and it is only after several failures have been made that they finally meet. Similarly, if he tries to touch his nose with his finger, he probably strikes his eye or his forehead or his mouth. The voluntary movements of the arms are occasionally effected by a series of jerky movements. But notwithstanding the widespread affection of his voluntary muscles, he probably during the whole duration of his illness retains perfect control over the rectum and bladder, and has no tendency to bed-sores.

Various other phenomena, due mostly to extension or multiplication of the nervous lesion, are apt to supervene in the course of the disease. In some cases the muscles of the trunk and of the head and neck become implicated in the same way as the muscles of the extremities, and the patient executes slight oscillatory movements when he sits up unsupported. In some cases difficulty of articulation comes on; the patient is slow, yet somewhat jerky and indistinct in utterance; he can pronounce every letter perfectly, but fails to pronounce them accurately in combination, and slurs over his syllables. There is often, too, a manifest over-exertion of the muscles of the mouth and tongue in the effort to speak, and fatigue is soon experienced. In some cases (if such phenomena have not appeared earlier in the disease) paralysis of the third, fourth, sixth, portio dura, hypoglossal, or vagus of one or other side, comes on; or double vision, impairment of vision, or amaurosis supervenes; or the patient grows absolutely deaf; or he becomes subject to the various forms of pain which have already been described; while, on the other hand, if these have previously existed, they may disappear. Further, he may now be liable to severe and

continuous aching pains in the forehead and back of the head, along the spine, and in the trunk and extremities, in connection with which, as with the earlier neuralgic pains, cutaneous eruptions may appear temporarily; or he may, late in the disease, suffer from retention or incontinence of urine, and equivalent conditions affecting the rectum; and generally he loses sooner or later all sexual desire and power. Occasionally, in the far advanced stages of the disease, rigidity, contraction, and wasting of muscles come on; complications which are obviously due to the extension of disease from the posterior columns to the lateral columns and anterior cornua.

Locomotor ataxy does not always involve the opposite limbs symmetrically; it often commences earlier in one leg than the other, and invades one arm in advance of its fellow; and in the subsequent progress of the disease the legs or arms may continue to be unequally affected. The course, too, of the disease is very various. Sometimes the symptoms arise and succeed one another so rapidly that the patient becomes bed-ridden at the end of a few months. But much more commonly the successive phenomena are slowly and irregularly evolved; periods of apparent amendment from time to time intervene; and ten, twenty, or thirty years may elapse before the disease attains its full development. It is more than doubtful if absolute restoration to health ever takes place when the clinical phenomena are so fully declared as to render diagnosis clear. It is not, however, doubtful that many persons do experience great amelioration of their symptoms, and that such amelioration is sometimes of long duration. Occasionally, indeed, the course of the disease appears to be permanently arrested. In the vast majority of cases, however, the progress of the patient, excepting for occasional interruptions, is uniformly from bad to worse, until death ends the scene. The causes of death are various. Generally it is due to some intercurrent malady; but it may be referrible to implication of the muscles of deglutition and respiration, to secondary bladder and renal mischief, or the formation of bed-sores.

Treatment.—When temporary improvement has occurred under our own observation, it has always seemed due simply to avoidance of over-exertion, rest, protection from cold and wet and other such adverse influences, judicious dieting and good hours; in fact to careful attention to the general well-being of the bodily health. It is not clear that any remedy exerts any, even the slightest, direct influence over the course of the disease. Nitrate of silver has been strongly recommended, iodide of potassium has been employed, iron and other so-called nervine tonics are often called into requisition. For the relief of pain, sedatives, such as opium or belladonna, or local applications, such as counter-irritants, frictions, and galvanism, may prove serviceable; and indeed it may be said generally that all complications and all discomforts arising in the course of the disease should if possible be relieved. As regards electricity, Duchenne observes that faradism

and galvanism with intermittent current, are either of them often serviceable both in relieving pain and in restoring voluntary power to the affected muscles in the earliest stage of the disease. But, while not forbidding their employment at a later period, he is evidently not sanguine as to the results which are then likely to be obtained. This is in accordance with general experience.

G. *Glosso-labio-laryngeal Palsy.*

Definition.—This name has been given by Duchenne to a paralytic disorder due to an affection of the medulla oblongata (whence also it has been termed *paralysie bulbaire*) involving mainly the seventh, ninth, and spinal accessory nerves, and revealing itself during life by paralysis of the lips, tongue, soft palate, and larynx.

Causation.—Its causes are as obscure as those of other affections of the same class. It has been referred to the effects of cold and moisture, and it has appeared to follow upon strong moral emotions. It seems to be a disease of adult life, and to affect women more largely than men.

Morbid anatomy.—The essential lesions of this disease are identical, so far as regards their nature, with those of locomotor ataxy, lateral sclerosis, and the like. They affect, however, a different region. Post-mortem examinations conducted on patients dead of this affection have revealed sclerosis, with more or less atrophy, of the roots of the spinal accessory, hypoglossal, and facial nerves, and sometimes similar changes in the roots of the vagi, in the motor roots of the fifth pair, and in the anterior roots of several of the upper cervical nerves; but they have also revealed (which is of still greater importance) that these changes in the nerves are secondary to pigmental atrophy of the large cells contained in the nerve-nuclei situated in the medulla oblongata, associated with more or less circumambient sclerosis. Certain phenomena in the clinical history of these cases, and the fact of the frequent supervention of the symptoms of glosso-labio-laryngeal palsy in the course of lateral sclerosis, render it probable that the disease, when occurring in the uncomplicated form, is often due less to a primary lesion of the nerve-nuclei than to their secondary implication in the course of some sclerotic change occupying the anterior pyramids.

Symptoms and progress.—In most uncomplicated cases of glosso-labio-laryngeal palsy the symptoms of the disease come on gradually. The tongue usually suffers first. The patient experiences some difficulty in the articulation of words, especially of those which need the special employment of the tip of the tongue, and presently also more or less difficulty in mastication and deglutition; and he suffers from the accumulation of saliva in his mouth. The paralytic condition of the tongue gradually increases; he has difficulty in protruding it and in drawing it in again, and ere long it lies motionless or nearly so on

the floor of the mouth, with its tip behind the anterior incisors and its edge pressed and indented against the arch of the lower teeth. It is sometimes reduced in size and wrinkled; sometimes it feels large to the patient, and either retains its normal dimensions or exceeds them. Whilst the lingual paralysis is in progress, the muscles of the soft palate and arch of the fauces become implicated, the patient's voice acquires a nasal quality, the difficulty of swallowing becomes aggravated, and his food is apt to pass into the posterior nares. The arches of the palate may occasionally be seen to be unequal, with the uvula pointing to one side; but it is remarkable that even when the paralysis, so far as deglutition and enunciation are concerned, is complete, the velum often can still be excited by local irritation to violent action. The lips also are early involved: the orbicularis becomes enfeebled, the lips get large, the lower one pendulous, and it is soon difficult or impossible for the patient to close his mouth, to prevent the flow of saliva from it, to utter the labial consonants, to whistle or blow out a candle, or to perform any function requiring the use of the lips. According to Duchenne, it often happens that the quadratus menti and triangularis oris of each side become implicated, so that the angles of the mouth cannot be drawn down and extended; but he says that the buccinators rarely suffer, and that the muscles of expression of the upper part of the face remain unaffected, and by their tonic contraction so act on the angles of the mouth as to cause the transverse elongation of the orifice, and at the same time so deepen and modify the direction of the naso-labial sulci as to impart to the patient's physiognomy the appearance of crying. Not unfrequently when a patient in this condition is made to laugh or cry, his mouth becomes widely opened, and remains open until the upper lip is restored to its original position by hand. The muscles by which the upward and downward movements of the lower jaw are effected for the most part retain their normal force, so that the patient can bite powerfully up to the last. Nevertheless, difficulty of mastication, already extreme in consequence of the paralytic condition of the tongue, is enhanced by paralysis of the pterygoid muscles, which renders the movements necessary for trituration impossible. Sooner or later the muscles of the pharynx and even those of the larynx share in the general paralytic affection, and hence the difficulty of deglutition becomes further aggravated.

In the latter stages of the disease the patient ceases to utter any articulate sound, although a laryngeal grunt, indicative of the due action of the vocal cords, may attend each effort to speak. The saliva which is constantly dripping from his lips accumulates in his mouth, becomes sticky from long retention, and on opening his jaws, hangs in ropes and festoons between the opposite surfaces. His food collects in the buccal pouches, or falls out through the open lips, and can only be made to reach the fauces either by throwing the head backwards or by pushing the food onwards with the fingers. The pharyngeal

stage of deglutition is equally difficult. Pultaceous matters are swallowed best; but these have to be passed to the back of the mouth in small quantities and with great care; and even then constantly cause choking—either finding their way into the windpipe, or into the nose, or being ejected by the spasmodic action of the pharyngeal muscles. The entrance of food into the larynx is mainly due to the failure of the tongue and epiglottis to descend over the superior laryngeal orifice during the act of swallowing; for it is only in somewhat rare cases that suppression of the laryngeal voice, indicative of paralysis of the laryngeal muscles, is observed.

Sooner or later, however, the pneumogastric nerves become implicated, and then symptoms referrible to the respiratory and circulatory organs are superadded. Attacks of difficulty of breathing, not due to the entrance of food or saliva into the windpipe, are now of frequent occurrence. They come on by day or night, and are often provoked by exertion. They do not appear to be connected necessarily either with pulmonary disease or with any paralytic condition of the ordinary respiratory muscles. Duchenne refers them to paralysis of the bronchial muscles. There is no doubt, however, that catarrhal affections of the bronchial tubes are now exceedingly apt to arise, and that these, however slight they may be, greatly aggravate, if they do not induce, dyspnœal attacks. Remarkable feebleness of circulation also supervenes at this period; and especially the patient is liable to syncopic attacks, which sometimes accompany the fits of dyspnœa, and are attended with præcordial anxiety, fear of death, and extreme feebleness, irregularity, and generally quickening of the pulse.

The phenomena above described are all unattended with febrile disturbance, loss of sensation, pain, giddiness, or any form of mental defect; the appetite continues good, the corporeal functions generally are well performed, and the system at large for the most part retains its powers, excepting in so far as they may become impaired by the starvation which the difficulty of swallowing gradually induces. Hence some patients who are far advanced in the disease will continue to go about the house and even to take long walks. In many cases, however, towards the close of life, they are confined to the chair or to bed.

The course of glosso-labio-laryngeal palsy is generally rapid, and its end is invariably death, which may come on within six months of its onset, and is very seldom delayed beyond three years. The causes of death are: starvation from inability to take nourishment; asphyxia, from the impaction of a lump of solid food at the back of the throat, or from the repeated entrance of portions of food or saliva into the larynx; an attack of dyspnœa or syncope; and lastly, pulmonary complications—bronchitis and the like—which are especially dangerous when involvement of the respiratory muscles renders the discharge of bronchial accumulation difficult or impossible.

Although glosso-labio-laryngeal palsy conforms in a large number of cases to the description which has just been given, it is not unfrequently a fragment, as it were, of some more widely diffused nervous disease. Thus, as is subsequently pointed out, it often forms one of the complications of disseminated sclerosis; its supervention constitutes, almost without exception, the last stage of lateral sclerosis; and, further, it is not uncommonly associated with progressive muscular atrophy, generally coming on late, but sometimes manifesting itself at an early period. The most important cases of the last group are those (and they are not rare) in which the respiratory muscles also waste. Again, it is important to recollect that groups of symptoms closely resembling those of glosso-labio-laryngeal palsy may be caused by effusions of blood into the pons or medulla oblongata, or by syphilitic or other disease of the same parts; and may even arise in connection with the descending lesions which follow chronic forms of cerebral disease.

Treatment.—Nothing that we are acquainted with is capable of arresting the course of this formidable malady. In the early stages electricity may be applied to the enfeebled muscles, and possibly with slight temporary apparent benefit. In the later stages we must endeavour to relieve symptoms; and it may then be of service to feed the patient either by the aid of the stomach-pump, or per anum.

H. *Disseminated Sclerosis. (Multiple Sclerosis.)*

Definition.—'Sclérose en plaques disseminées' is the name which Charcot (to whom we are mainly indebted for its recognition and description) has given to the affection which we here term disseminated sclerosis. Dr. Moxon calls it insular sclerosis. It is characterised, post mortem, by the presence of a number of small roundish patches of sclerosis, scattered irregularly throughout the nervous centres; clinically, by a variety of symptoms, among the most characteristic of which are tremblings of the head, neck, trunk, and limbs, coming on only when the muscles are being exerted, difficulty of speech, oscillation of the eyeballs, gradually supervening paralysis, with contraction, mainly of the lower extremities, and some impairment of the mental functions.

Causation.—Disseminated sclerosis is mainly a disease of adult life, usually coming on between the ages of twenty and twenty-five, rarely after thirty, but sometimes at the period of puberty, and even in childhood. It is more common in women than in men. It has been attributed to the same causes as those to which other forms of sclerosis have been attributed, namely moral influences and exposure to wet and cold. Its advent has sometimes been heralded by hysteria, neuralgia, or other nervous symptoms.

Morbid anatomy.—Sclerotic patches may appear in the cerebrum,

cerebellum, pons, medulla and spinal cord, either collectively or separately; but generally are distributed in several of these organs at the same time. In the cerebrum they occupy mainly the neighbourhood of the ventricles, and are found, therefore, in the corpus callosum, septum lucidum, corpora striata, and optic thalami; they occur also in the centrum ovale, but seldom in the grey matter of the convolutions. In the cerebellum, their almost exclusive seat is the corpus dentatum. As regards the pons and medulla oblongata, they may be either superficial or deep-seated. In the former they affect mainly the anterior and inferior aspect, extending thence to the cerebral peduncles and corpora albicantia; in the medulla, they occupy all parts indifferently, inclusive of the region forming the floor of the fourth ventricle. In the cord, as in the medulla, all parts are liable to be implicated. The cerebral and spinal nerves sometimes emerge, unaffected, from diseased tracts; in other cases they are studded with similar morbid patches, or are involved generally. The cerebral nerves which chiefly thus suffer are the first, second, and fifth pairs. The patches of sclerosis vary in size, but are for the most part well-defined, and of roundish form. They are dense, hard, slightly translucent, and of a greyish colour, closely resembling that of the healthy grey matter of the brain. They sometimes project a little above the general level, sometimes are more or less obviously depressed below it. Microscopically they present all the ordinary characters of sclerosis; and usually, according to Charcot, may be divided into three zones, of which the outermost represents the disease in its earliest phase, the innermost represents it in its most advanced condition. In the outermost zone, the neuroglia is increased in amount and its nuclei in number, and the nerve-tubules are diminished in diameter at the expense of the white substance of Schwann; in the next zone, the neuroglia has still further increased and has become distinctly fibrillated, the nerves, more widely separated than they were, have become yet more reduced in size, and the white substance has almost wholly disappeared while the axis cylinder has in many cases undergone enlargement; in the central area, the overgrown neuroglia reigns paramount, the nerve-cells and nerve-tubules have for the most part vanished, and those which still survive are far advanced in atrophy. It may be added: that the gradual disappearance of the white matter of Schwann involves the production of a large number of free oil-globules and granule-cells, which stud the outer two zones, and tend to accumulate within the lymphatic sheaths of the vessels; that the blood-vessels become enlarged, and the nuclei in their walls increased in number; that the nerve-cells undergo pigmental atrophy, shrink, and finally disappear; and that corpora amylacea tend to become developed in the course of the vessels.

Symptoms and progress.—The symptoms to which patches of sclerosis may give rise must depend partly upon their size, partly on their situation, partly on their number. Thus it is obvious: that if a patch

of sclerosis should interrupt the continuity of the posterior columns of the cord, symptoms more or less identical with those of locomotor ataxy would be developed; that if it should involve one of the lateral columns, the symptoms referrible to it would have more or less resemblance to those characteristic of lateral sclerosis; that if the anterior cornua should be implicated, more or less rapid wasting of certain muscles might be expected to follow; that if the medulla oblongata should be its seat, some of the symptoms of bulbar paralysis or of glosso-labio-laryngeal palsy would necessarily arise; and that if seated in the cerebrum, hemiplegia, convulsions, impairment of intelligence, or other of the various consequences of brain-lesion, would almost certainly follow. It is, further, obvious that if many sclerotic patches should be distributed throughout the nervous centres, the consequences due severally to them would blend, as it were, into a common whole producing collective symptoms of more or less complexity.

It is, nevertheless, a fact that a large number of cases of disseminated sclerosis, in which the nervous centres are generally implicated, are attended with groups of symptoms which collectively afford almost positive proof of the nature of the malady which is in progress. We will consider the more important of these symptoms successively.

1. *Rythmical tremors.*—These constitute one of the most distinctive features of the disease. They are absent when the patient is asleep, they are absent also when he lies at rest, with his limbs and head supported; but they come on whenever he makes any muscular effort, and become more and more pronounced the greater and more sustained that effort is. When he raises his hand from the bed-clothes more or less violent convulsive movements seize his fingers, his hand, and his arm; if he attempt to raise his hand to his lips, these tremulous movements increase; and if, further, the muscular effort be rendered greater by his having to lift some heavy body, or something which requires care and precision in handling, as, for instance, a glass of water, they are apt to become exceedingly tumultuous, and to increase in tumultuousness as the task set him approaches completion. Occasionally they are induced when the arm is apparently at rest, by the nervous efforts of the patient to keep it still. If he sit up unsupported, similar movements affect his trunk and his head and neck. If he endeavour to stand or to walk, they become universal, and the legs, arms, trunk, and head are all violently agitated. It is not pretended that these tremulous movements are present in all cases of disseminated sclerosis; but they are present in the great majority of cases. Nor must it be assumed that, when present, they are always of general distribution; they may (for a time at least) be limited to one arm, or to both arms, or when present in both may affect them unequally; or the legs may chiefly suffer. Neither must it be supposed that they are present during the whole course of the malady. Rhythmical trembling is rarely one of

the earliest symptoms of the disease; but it usually comes on before long, and then invades the various parts slowly and irregularly; and it disappears as the patient becomes more and more enfeebled, and especially when paralysis supervenes. The movements are peculiar; they are rhythmical, and yet there is some degree of irregularity both in the extent of the successive vibrations and in the intervals which separate them. They have some resemblance to those of paralysis agitans; but in the latter the vibrations are more rapid, and more regular, moreover, they occur when the patient is at rest, and seldom, if ever, directly implicate the head and neck. They have a greater resemblance still to those of chorea, but they are less wanton, less violent, and altogether more rhythmical than these; and, further, the vibrations in sclerosis for the most part take the direction of the general movement of the limb or part which is engaged in movement. It must not, however, be forgotten that tremulous movements, undistinguishable from those of sclerosis, may attend various other affections, and especially chronic mercurial poisoning, chronic cervical meningitis, and sclerosis of the lateral columns.

2. *Affections of the eyes.*—Double vision is a not uncommon symptom of the earlier stages of sclerosis, as it also is of locomotor ataxy, but is for the most part transitory and unimportant. Indistinctness of vision is a much more frequent phenomenon, and is generally permanent, but rarely ends in blindness. It is often present when no signs whatever of disease can be detected with the ophthalmoscope. But sometimes more or less advanced atrophy of the optic disc is present, which becomes complete in cases of total blindness. Nystagmus is a symptom of considerable importance, and is present in about half the total number of cases; it consists in consensual small oscillations of the eyeballs, which in slightly advanced cases may be apparent only at the moment when the patient endeavours to fix his glance upon some fresh object, or looks out of the corners of his eyes; but they are generally constant, although aggravated by voluntary movements of the eyeballs. They cease when the patient is asleep, or when his eyes are shut in repose. Nystagmus is rarely present in locomotor ataxy.

3. *Defect of speech.*—This is nearly constant. In well-marked cases the utterance is slow and drawling. The words (to use Charcot's expression) are 'scanned,' as it were, there is a pause after each syllable, and the syllables themselves are slowly evolved. Moreover, they are imperfectly pronounced, certain letters or difficult combinations of letters being slurred, and sometimes to such a degree that speech becomes unintelligible. Further, the lips and tongue are often tremulous: the lips tremble previous to the utterance of articulate sounds, and during the course of utterance; and the tongue when it is protruded is in constant fibrillar movement. This tremulousness of the organs of speech adds to the difficulty of articulation, and imparts to it a peculiar tremulousness or uncertainty. A very similar defect of

speech is apt to accompany locomotor ataxy, but in that case the tremulousness of the lips and tongue is absent, and (at least according to our observation) the muscular efforts to utter articulate sounds are unnecessarily violent.

4. *Vertigo* is an early symptom in about three-fourths of the total number of cases. It is mostly gyratory, and generally comes on in paroxysms of short duration, but is sometimes almost continuous. It often interferes seriously with locomotion. The presence of nystagmus is also a cause of vertiginous sensation, the oscillations which take place in the eyeballs being referred by the patient to the objects which are figured on his retinæ. Vertigo is not common either in tabes or in paralysis agitans.

5. *Paresis* of the limbs, and more especially of the lower extremities, comes on at an early stage of the disease. It generally begins in one leg. This feels weak and heavy, and drags in walking, but there are no movements indicative of inco-ordination. Soon the other leg becomes affected; but even then (so different from what occurs in tabetic patients) so long as he has sufficient strength to stand, he is capable of maintaining his equilibrium even when his eyes are shut. The weakness subsequently extends to the arms. This enfeeblement of the limbs gradually increases until it culminates in absolute motor paralysis: the convulsive oscillations of the earlier period undergoing proportionate diminution until they finally cease. The paralysis which commences in the lower limbs becomes as a rule complete in them while the arms are yet comparatively little involved. The patient not unfrequently complains of some degree of tingling and numbness, but there is rarely if ever any obvious impairment of cutaneous sensibility. Moreover, the muscular sense remains unaffected, so that he recognises exactly the position of objects and the amount of force necessary to accomplish various voluntary movements. There is no paralysis of the bladder or rectum; the affected muscles retain their form, bulk, and tonicity; and reflex and electrical contractility are unimpaired. The paretic condition of the limbs is liable to remissions.

6. *Contraction of limbs.*—At some period or other in the course of the paretic symptoms, the lower extremities, either spontaneously or under excitement, become suddenly stiffened in extension, and pressed one against the other. These attacks may last some hours, or even some days, and are at first separated from one another by comparatively long intervals. But by degrees the intervals shorten, and at length ceasing, the rigidity of the muscles becomes permanent. At this period the thighs are extended on the trunk, the legs on the thighs, the feet on the legs, and the members lie in close apposition and cannot be separated. Sometimes the flexors overcome the extensors, and the limbs are flexed at all the joints. Occasionally, but at a later period, the arms become rigid and pressed against the sides of the body. At this time violent tremblings, lasting for a few minutes or even longer,

are apt to arise in the stiffened limbs. These seem sometimes to come on spontaneously; but they may be excited by exposure to cold, or by pricking, tickling, electricity, or other forms of irritation; and they may extend from the limb in which they were first induced to the opposite limb, and even cause general trembling of the body. They may be at once stopped, according to Brown-Séquard, by forcibly flexing the great toe. This stiffening of the limbs may be developed while their movements are still in some degree under the control of the patient, and does not therefore necessarily incapacitate him from walking with assistance.

7. *Expression and mental condition.*—During the course of the disease a marked change in the expression is apt to come on. The patient's glance is vague and uncertain, his lips pendulous and apart, his general aspect sad, weak, or fatuous. At the same time there is some change in his mental condition; the memory fails, the conceptions are slow, and the intellectual and affective faculties generally impaired. He is stupidly indifferent to all that goes on about him, and is apt to laugh or cry without occasion. Sometimes he becomes maniacal or demented.

One or more of the symptoms which have just been enumerated may fail in a greater or less degree in certain cases. But, on the other hand, additional phenomena are not unfrequently superadded. We have already suggested as possible complications certain phenomena which actually do not unfrequently present themselves in the course of the disease:—namely, inco-ordination of the movements of the lower extremities, and even of the hands and arms; wasting of certain of the voluntary muscles; and difficulties of deglutition, respiration, and circulation, indicative of involvement of the medulla oblongata. But, further, apoplectiform attacks are not unfrequent. These may come on without warning, or may be preceded by rapid failure of the mental faculties. They recur as a rule several times at irregular and long intervals. They are often attended with convulsions, which are usually unilateral, or with hemiplegia, associated sometimes with flaccidity, sometimes with rigidity of the paralysed muscles. In these attacks the pulse becomes greatly accelerated, and the temperature of the internal parts rises rapidly, so that probably in the course of the first few hours it mounts to 102°, and within twenty-four hours to as much as 104°. If the case is about to prove fatal, the temperature may reach 108° or 109°. In these cases bedsores also are apt to form with great rapidity upon the sacral region. These apoplectiform attacks (which are not peculiar to disseminated sclerosis, but occur equally in cases of general paralysis and tumours of the brain, and in cases in which embolic softenings or apoplectic effusions have left chronic lesions behind them) are distinguishable from those due to hemorrhage by the fact of this sudden and rapid rise of temperature.

Charcot divides the clinical history of cases of disseminated sclerosis into three periods. The first extends from the first appearance of symptoms down to the supervention of rigidity of the limbs. The second includes all that time subsequent to the first appearance of rigidity during which the patient's symptoms undergo gradual aggravation, but during which the organic functions as yet maintain their integrity. The third commences with the failure of the nutritive functions.

First period.—The mode of invasion is various. Sometimes the disease commences with symptoms referrible to the brain, such as vertigo, or diplopia, soon followed by embarrassment of speech and nystagmus. More commonly the first symptoms are spinal, especially weakness of the lower extremities, which may continue for months or even for years before it becomes complicated with other phenomena. This weakness is liable to remissions, and is usually unattended with pain, loss or impairment of sensation, or difficulty of micturition or defecation. It presents nothing distinctive. Rarely the disease commences with symptoms like those which usher in locomotor ataxy. The early progress of the disease is usually slow, but now and then the symptoms appear and follow one another with great rapidity. The contraction of the limbs, the supervention of which terminates this stage, does not usually show itself till after the lapse of two, four, or even six years.

Second period.—This is usually of long duration. During it all the characteristic symptoms of the disease are present and undergo gradual aggravation, until the patient becomes utterly helpless and confined to his chamber or his bed.

The *third period* comes on with progressive weakening of the organic functions. At the same time some of the symptoms proper to the disease come into special relief. Intelligence fails; the patient becomes, perhaps, fatuous; the sphincters cease to act, and the evacuations are all passed unconsciously; the bladder inflames; bed-sores form; and appetite for food declines. At this time, also, various intercurrent maladies are apt to come on, such as pneumonia, dysentery, or diarrhœa, or difficulty of deglutition with other signs of involvement of the medulla oblongata.

The duration of the cerebro-spinal form of the disease usually varies between six and ten years; but if the cord only be affected, life may be prolonged for twenty years or more. The causes of death are numerous. Among the more important may be enumerated: apoplectic attacks, the consequences of affection of the medulla, pneumonia and other intercurrent disorders, inflammation of the bladder, bed-sores, and debility from failure of the nutritive powers.

Treatment.—This appears always to have failed. Charcot observes that both strychnine and nitrate of silver have served for a time to

check the trembling of the muscles, but have had no permanent good effect. Arsenic, belladonna, ergot of rye, and bromide of potassium have all been used at various times, but without obvious beneficial results. Little that is favourable can be said even of hydropathic treatment, or faradism, or the continuous current.

VI. PARALYSIS AGITANS. (*Shaking Palsy.*)

Definition.—This is a disorder mainly of advanced life and of chronic progress, characterised especially by trembling of the limbs arising independently of voluntary movements, and for the most part sparing the head and neck. The patient in an advanced stage, without vertigo, is unable to maintain his equilibrium when walking.

Causation.—The causes of paralysis agitans are various.. It would seem to be not unfrequently brought on more or less suddenly by violent emotion, such as terror, grief, rage, and the like. It is often referred, and probably with truth, to long-continued exposure to cold and wet. And it is asserted that it is occasionally traceable to wounds or bruises involving peripheral nerves; in favour of which statement is the fact that severe neuralgic pains referrible to such injuries have been succeeded by trembling of the parts involved, and subsequently by the general phenomena of paralysis agitans. There is little proof that the disease is hereditary. Neither does it belong to one sex more than the other. It is for the most part a malady of advanced life, usually first making its appearance after the age of forty. It may, however, occur at an earlier period, and cases are on record in which it commenced at twenty or even sixteen.

Morbid anatomy.—Of the condition of the nervous system in this affection nothing definite is known. Previously to Charcot's investigations, paralysis agitans and disseminated sclerosis were usually confounded with one another, and the lesions of the latter disease were consequently regarded as having an important connection with the clinical phenomena of the malady now under consideration. In cases, however, of true paralysis agitans no constant lesions, sufficient at all events to explain the peculiarities of its symptoms, have yet been discovered. In some recent examinations of Charcot's, there were found obliteration of the central canal of the cord by increase of its epithelial lining, overgrowth of the nuclei which surround the ependyma, and marked pigmentation of the nerve-cells, chiefly those of Clarke's posterior vesicular columns. It must be observed, however, that post mortem examinations in cases of this disease are almost necessarily made on persons far advanced in life, in whom, therefore, on other

grounds such changes as are here referred to are likely to be met with.

Symptoms and progress.—The symptoms of paralysis agitans may come on gradually or suddenly. In the great majority of cases the onset of the disease is insidious. The part attacked is the hand or foot, or the thumb. If the hand be affected, its different segments oscillate on one another in a manner which is almost distinctive. The thumb moves on the other fingers as in the act of twisting wool, rolling a pencil, or crumbling bread. If the affection involve also the rest of the upper extremity, these movements of the fingers are associated with similar rapid backward and forward movements of the hand as a whole on the forearm, and of the forearm on the upper arm. At this period of the disease the trembling is often transitory. It comes on occasionally only, and maybe at long intervals. It comes on, moreover, when it is least expected—when the patient is at complete rest, mentally and bodily; and it may be arrested by an effort of the will, and often ceases when he walks, or when he uses the affected limb for writing, lifting a weight, or other purposes. The trembling may be confined for an indefinite time to the part first attacked. But it generally spreads sooner or later: first, if a part only of a limb have been involved, to the rest of the limb, and subsequently, and often after longish intervals, to other limbs. It usually assumes in the first instance the hemiplegic form, affecting first the arm and then the leg of the same side, and extending later to the arm and leg of the opposite side. Sometimes it puts on the paraplegic character, spreading from one leg to the other, before the upper extremities get involved. It seldom extends from one arm to the other, leaving the legs unaffected, or from the arm of one side to the leg of the opposite side. In some cases tremulousness is not the first symptom of which the patient complains. But its occurrence is preceded for a longer or shorter time either by a sense of profound fatigue, or by rheumatic or neuralgic pains referrible to the limb or part of a limb in which convulsive movements subsequently manifest themselves. In rare instances the affection comes on suddenly, with tremulousness either of a single limb or of all the limbs. Under these circumstances it may subside at the end of a few days. But other similar attacks are liable to follow at decreasing intervals, until ultimately the disease becomes established. The duration of the initial stage to which the above account refers varies usually from one to two or three years.

When paralysis agitans has attained its complete development the trembling not only involves several limbs, and probably all of them, but is also (at all events in severe cases) almost incessant. It is liable, however, to remissions and exacerbations, the latter of which seem to be often induced by emotional disturbance or muscular exertion, yet not unfrequently come on without obvious cause. Natural sleep, or

that induced by chloroform, is always attended with entire cessation from convulsive movements. It is at this period that the tremors put on their most distinctive characters. They consist of involuntary rhythmical oscillations, which have little amplitude, follow one another rapidly, and present considerable uniformity; and which, when the hand is involved, give to its different segments the aspect of being collectively engaged in the performance of some delicate process or operation. The head and neck remain as a rule free from convulsion. So far indeed from being agitated, the muscles of the face are immoveable, the look is fixed, and the features present a permanent aspect of sadness or hebetude. Nystagmus, so common in disseminated sclerosis, is absent here. The muscles of the jaw also are free from movement, but the tongue, when protruded, not unfrequently presents well-marked tremulousness. There is no real failure of language, but speech is slow, hesitating, laborious, as though the enunciation of each syllable were attended with considerable effort. It may, however, become tremulous in consequence of the transmission of the tremulousness of the limbs to the head and neck. Deglutition is performed without difficulty, but is perhaps somewhat slow; and often in old cases saliva tends to accumulate in the mouth. Respiration does not suffer.

A striking phenomenon of the disease, to which Charcot has especially called attention, is rigidity of the muscles, which comes on for the most part late in the malady, though occasionally at its commencement. It affects the muscles of the extremities, trunk, and neck. The supervention of this rigidity is attended with cramps, followed by contraction, which is at first transitory, but after a time becomes persistent, though even then liable to exacerbations. The flexor muscles suffer in the chief degree. The rigidity and contraction becoming permanent give a peculiar aspect to the patient. The head is thrown strongly forwards and fixed in that position; and the trunk, when the patient stands, is bent in the same direction. The elbows are separated a little from the trunk; the forearms are slightly flexed on the upper arms; and the hands, similarly flexed on the forearms, rest upon the waist. The hands, moreover, are more or less deformed; usually the fingers are slightly flexed in mass at the metacarpo-phalangeal joints, with an inclination to the ulnar side of the arm, and with the thumb resting against the forefinger as in the ordinary position for writing; but in some cases the fingers, though substantially occupying the same position, are flexed at the proximal and distal joints, but extended at the middle joints. The rigidity of the lower limbs is such as sometimes to give the appearance of paraplegia with contraction; the feet are occasionally in the position of talipes equino-varus, and the toes bent into the form of a claw. The patient, however, retains the power of voluntary movement, and the muscles are never thrown into the tetanic spasms which are so common in many spinal diseases.

The difficulty of movement which characterises patients suffering from shaking palsy is due no doubt in some degree to the muscular rigidity and contraction which have just been described. But it often manifests itself long before the rigidity has become particularly obvious.

The same peculiarity attends speech. It would seem that unwonted efforts are needed for the transmission of the motor impulses; and indeed the slightest movements are followed by extreme fatigue. This group of phenomena has been taken to imply the existence of true paralytic weakness. But it is not so, for on testing the strength of different limbs by the dynamometer, it has been often found, that (excepting in the case of patients in the last stage of the malady) muscular force is remarkably preserved. Sometimes, indeed, the muscles of the most tremulous and apparently weakest limb are really more powerful than those of its seemingly healthier fellow.

The mode of walking in paralysis agitans is usually highly characteristic of the disease. The patient rises perhaps with difficulty from his seat, then steadies himself for a few seconds, and at length, with his head and trunk in advance, runs straight forwards in spite of himself with rapid steps. He appears to be losing his equilibrium, and running forward to regain it; and not unfrequently falls down. This difficulty of maintaining his balance in walking is not wholly due to the position which his body generally assumes, for it may occur while he is yet capable of retaining the erect posture; and, further, in some cases the patient has a tendency to fall or run backwards even when his body is bent forwards. Neither is it connected with the presence of vertigo, for the patient does not as a rule suffer from this sensation.

Various other symptoms besides those which have been enumerated complicate the course of shaking palsy. Patients usually complain of a sense of persistent tension or traction in the affected muscles, or of cramps; they experience a feeling of prostration or utter fatigue which especially comes on after fits of trembling; or they are the victims of an undefinable malaise or fidgetiness. They want incessantly to shift their position; and if they be not assisted in their desire their sufferings become unendurable. They suffer most in this respect at night and when in bed. Another cause of suffering is an habitual sensation of excessive heat, referred mainly to the epigastrium and back, but not limited to these situations. It varies in intensity, and is usually most severe after the occurrence of a paroxysm of trembling. It is not attended with any actual elevation of temperature. Cutaneous sensibility is in no degree affected. The patient retains his mental faculties and the power over his rectum and bladder.

The final stage of the disease supervenes for the most part at the end of some years. It is indicated: by aggravation of the difficulty of

movement, the patient being consequently compelled to keep his room or his bed; by failure of nutrition in which the muscles chiefly suffer, occasionally becoming fatty; by impairment of intelligence and of memory; by general prostration; and by the formation of bed-sores. At this time the convulsive movements not unfrequently cease. Death results sometimes from gradual asthenia, more commonly from the supervention of some other disorder, especially pneumonia. The duration of the disease may extend to twenty or thirty years.

Treatment.—All kinds of treatment have been employed, but for the most part with little success. Among the medicines which have been recommended may be named iron, nitrate of silver, chloride of barium, arsenic, zinc, strychnia, ergot of rye, belladonna, opium, hyoscyamus, and Calabar bean. Of these iron is advocated by Elliotson, and has perhaps been instrumental in improving the general health of patients; and strychnia has been lauded by Trousseau, but seems to have been found injurious by Charcot. The only one of the sedative drugs which the latter authority thinks serviceable is hyoscyamus, and this effects no permanent improvement. Warm baths, cold baths, and shower baths are also sometimes of temporary service. Electricity has been largely employed, but the only form which seems to have been of real efficacy is the constant current. A few cases of recovery under its use have been recorded. It must not be forgotten, however, that cases which have not advanced beyond the early stage occasionally get well spontaneously. On the other hand, this fact justifies the hope of benefit from judicious treatment. Hygienic measures should never be neglected.

VII. PSEUDO-HYPERTROPHIC PARALYSIS.

Definition.—This is a form of paralysis, first recognised and described by M. Duchenne, occurring in children, and attended with remarkable enlargement of some of the paralysed muscles.

Causation.—It has hitherto been observed in childhood only, and almost exclusively in boys. It has been met with also in several children of the same family. But beyond these facts nothing whatever is known in reference to its causation.

Morbid anatomy.—The morbid process, so far as the muscles are concerned, appears to consist mainly in the gradual growth of connective tissue in the interstices between the ultimate fibres; this becomes abundant and dense, and in some cases the seat also of the formation of fat. It is to this overgrowth that the apparent hypertrophy of the muscles is due. The muscular fibres appear to dwindle

away under the influence of the pressure to which they are subjected; and, although retaining their transverse striation for a long period, at length undergo degenerative changes—the transverse striæ becoming indistinct, or effaced, longitudinal markings perhaps unusually apparent, and more or less abundant deposit of granular or fatty matter taking place. The condition of the muscles in the earliest stage of the disease has been less thoroughly investigated than their condition in the later periods. M. Duchenne believes that at that time there is an œdematous state of the tissue which itself causes a certain amount of increase of volume.

Symptoms and progress.—The course of pseudo-hypertrophic paralysis has been divided by M. Duchenne into three periods. Of the first but little is certainly known, for its symptoms are slight, and children are rarely at that time brought under medical treatment; moreover the symptoms are in no degree distinctive of the disease, and are apt therefore to be misunderstood. The first symptoms appear to be due to gradual enfeeblement of the muscles of the lower extremities, and perhaps of those of the back. The child presents certain peculiarities of gait. He stands with his legs widely separated, and his shoulders thrown back, probably beyond the buttocks, the concavity of the small of the back being correspondingly deepened; he also walks with his legs apart, lifting the knee of his advancing leg needlessly high, while the foot is more or less extended, and the toes point downwards, and swaying his body from side to side in association with the peculiar position and movement of his lower extremities. This stage, according to M. Duchenne, usually varies in duration from a few months to a year. It may, however, be delayed, or it may so speedily merge in the second stage as to be unappreciable.

Fig. 11. Portrait of a boy suffering from pseudo-hypertrophic paralysis.

The second stage is marked by the gradual extension of the disease and the enlargement of certain muscles. The paresis, which probably always commences symmetrically in the lower extremities, gradually mounts, involving successively the muscles of the back and of the trunk generally, the muscles of the arms, and in some cases those of the face—more especially the temporals and masseters. Possibly some fulness of the calves may already have been apparent in the first stage; but now they augment rapidly and considerably in volume; and by degrees various other groups of muscles become similarly affected.

The degree of enlargement and its distribution differ in different cases. In some, only the calves become hypertrophied, while the muscles of the rest of the body either retain their normal bulk, or shrink; in some the calves and buttocks are the chief seats of overgrowth; in some the increase of bulk involves all the muscles of the lower extremities together with the posterior muscles of the spine; in some, again, the deltoids share in the widely-diffused hypertrophy; and occasionally all the muscles of the limbs and trunk become enormously increased in volume, and the child (though so feeble, perhaps, that he can scarcely move) acquires the appearance of an infant Hercules. This enlargement of the muscles, even if it be limited to the calves, is a very striking phenomenon; especially when, as in these cases, it goes along with progressive loss of muscular power. During the progress of this stage the phenomena which have already been referred to as attending the acts of standing and walking, become more pronounced; the legs are kept widely apart; the shoulders are thrown far backwards; and the peculiar swaying of the body from side to side, which attends the efforts to raise and project the legs successively forwards, becomes considerably exaggerated. Moreover, the child has the greatest difficulty in rising from the ground on which he is sitting into the upright posture; he gets on all-fours, then protrudes his buttocks like a dog in the act of stretching, and probably finds all his efforts fruitless unless he can manage to raise his head and shoulders hand over hand by means of a chair or bedstead. This stage attains its full development in a year or a year and a half, and may then continue with little change for two or three years more.

The third stage is characterised by extension of paralysis to the upper extremities, supposing these to have escaped hitherto; by arrest of the progressive enlargement of the muscles; and possibly even by their diminution. The child grows more and more helpless; the voluntary elevation of the arms becomes difficult and at length impossible; and he gradually loses all power in his lower extremities, and is hence condemned to pass the rest of his existence on his chair or in bed. Since respiration, circulation, and digestion remain unaffected, life may be sustained in this condition for a considerable period; but sooner or later the vital powers of the patient become prostrated, and pneumonia or some other intercurrent affection carries him off. Death usually occurs during the period of adolescence.

In order to complete the picture of the disease, two or three other facts in relation to these patients must be mentioned. No febrile symptoms manifest themselves at any period of the disease. The muscles in the early stage retain their electro-contractility almost unimpaired; but later on, as their fibres undergo degeneration, electro-contractility necessarily dies out. It has been shown by Dr. Ord that the tem-

perature of the legs, in cases in which the calves are hypertrophied, is three or four degrees higher than that of the thighs, a fact which he connects with the active growth of fibroid material taking place between the muscular fasciculi. It has often been observed that children afflicted with this disease are or become defective in their intelligence; and that if they be attacked before they have learned to speak, they are slow in learning to speak, and imperfect in their articulation. Lastly, there is no impairment of sensation, and no loss of control over the bladder or rectum.

Pathology.—Notwithstanding the symmetrical character of pseudo-hypertrophic palsy, its tendency to become generalised, and its association with impaired intelligence, there is no sufficient reason to regard it as of nervous origin. For no lesions whatever have been detected in the nervous centres or in the nerves; while the absence of rigidity, inco-ordination, and rapid wasting, and the retention of sensation, control over the sphincters and muscular contractility, equally point to integrity of the spinal cord. On the other hand, in the muscles themselves progressive changes have been discovered which are ample to explain the main phenomena of the disease.

Treatment.—According to M. Duchenne, pseudo-hypertrophic paralysis may sometimes be cured or arrested in its first stage by muscular faradism, aided by baths and kneading, or shampooing. When once, however, distinct enlargement of muscles has taken place, no treatment that has yet been adopted avails to delay the fatal progress of the disease.

VIII. MORBID GROWTHS. ANEURYSMS. ENTOZOA.

Various forms of adventitious growths affect the nervous centres or structures in relation with them. It is quite impossible, however, to distinguish them from one another during life by reference simply to the nervous symptoms which they induce. It is needless, therefore, for clinical purposes, to discuss each variety separately; and we shall content ourselves with first giving a brief sketch of some of the most striking pathological phenomena which the more important forms of growths present, and then discussing the clinical history of such tumours as a whole.

Morbid anatomy.—1. *Tubercle.* In a strictly scientific arrangement of disease, we ought of course to include under this head miliary tubercles of the pia mater. We have considered these, however, elsewhere in association with meningitis, which they generally induce, and independently of which they rarely if ever cause symptoms. The

variety of tubercle which we have now specially to consider is that which originates within the nervous substance and forms tumours there varying from the size of (say) a pin's head to that of a fowl's egg. They are well-defined, rounded, or lobulated masses, opaque, of a yellowish or greenish tinge, with much of the consistence and aspect of cheese. They correspond pretty exactly to the description usually given of typical yellow or crude tubercle; but although they may become disintegrated at points, they seldom, if ever, break down into cavities. They are made up of an aggregation of smaller masses, and differ in no important respect from the tubercular aggregates which in cases of tubercular meningitis are found along the vessels or in the depths of the sulci, and are the result of the coalescence of miliary tubercles. They may be solitary, or may exist in large numbers; and they may occur in any part of the nervous centres, involving, however, by preference the grey matter both of the brain and of the cord. No doubt, from their large size, the cerebral lobes are pre-eminently liable to suffer. But tubercles have also a remarkable aptitude to form in the substance of the cerebellum; and then (according to Andral) in the ascending order of frequency in the pons, medulla oblongata, spinal cord, peduncles of cerebrum and cerebellum, optic thalami, and corpora striata. They are much more common in the upper part of the cord than in the lower part. Tubercular tumours of the brain appear to occur more frequently in boys than girls, and are rarely met with either in adults or in children under two years of age. They occur most commonly between the ages of three and seven. Tubercle of the nervous centres is probably always associated with tubercle in other parts; and although ordinarily there is no connection between them, tubercular meningitis sometimes supervenes on the presence of tubercular masses of old date in the substance of the brain.

2. *Syphilis.*—The ordinary seat of intracranial syphilis is the dura mater. The disease may involve the outer aspect of that membrane, in which case it is usually associated with disease of the bones of the skull, and affects the brain mainly by pressure. Or it may involve the substance of the membrane, or its inner aspect, leading to the development of hard dense gummata which may be solitary or multiple, localised or scattered over a considerable extent of surface, and may vary individually from the size of a hazel-nut downwards. These tend gradually to involve the contiguous structures. The visceral arachnoid becomes adherent to them, and not unfrequently similar growths then develop in the subarachnoid tissue and pia mater. Subsequently the subjacent brain suffers, becoming first indented, and then either softened or the seat of gummatous growths. The parts of the dura mater which are most commonly affected are those corresponding to the convexity of the hemispheres, and those in relation with the anterior

and under surface of the brain, more especially in the neighbourhood of the sella turcica, whence the disease may spread to the surface of the petrous portions of the temporal bones and to the tentorium cerebelli.

Gummatous tumours originating in the pia mater or in the substance of the brain are much less common than the last, and as a rule are softer and more transparent and jelly-like; they are usually of small size, but may attain the bulk of a hen's egg. Those which are developed primarily from the pia mater affect mainly the under aspect of the brain, more especially between the optic commissure in front and the pons behind, and in the course of the cerebellar peduncles. These, too, are the situations in which they attain their greatest bulk. Tumours of the substance of the brain arise chiefly in the hemispheres, and the larger ganglionic masses, especially the optic thalami. After these parts they affect mainly the pons Varolii and the cerebral and cerebellar peduncles. It is an important fact that syphilis, whether of the dura mater, pia mater, or nervous tissue, has a marked tendency to affect the parts at the base of the brain, and consequently to implicate the nerves there situated. Although there are good clinical reasons for believing that the cord and its membranes are not unfrequently the seat of this disease, there are but few published cases in which the diagnosis has been verified by post-mortem examination.

It is not rare for the cerebral arteries in connection with syphilitic growths to become obstructed with thrombi; but it also not unfrequently happens, in cases of secondary or tertiary syphilis, that, independently of the formation of gummata, the walls of certain of the arteries at the base of the brain become thickened, indurated, and translucent, and the channels subsequently obstructed, partly from this thickening of the walls, partly from thrombosis.

3. With respect to other neoplastic formations, notwithstanding the importance and frequency of some of them, we need not, for many reasons, go into much detail. They are mainly the following:—fibroma, psammoma, melanoma, and cholesteatoma (to which, on account of their rarity or of their insignificance in a clinical sense, we shall make no further reference), and myxoma, glioma, sarcoma, and cancer.

a. Myxomatous tumours are not altogether unfrequent. They sometimes originate in the membranes of the brain or cord, sometimes in the cerebral substance. Their most common seat, however, is the cerebral hemispheres, where they form transparent gelatinous growths, which often become cystic, and tend to acquire large dimensions. They may attain the size of a man's fist.

b. Gliomatous tumours, also, are not unfrequent, and, indeed, are almost special to the nervous centres. They are greyish or pinkish in tint, translucent and highly vascular, infiltrate, as it were, the tissues in which they are found, and blend insensibly with them at their edges.

Moreover, though varying somewhat in colour, transparency, and density, they have a considerable resemblance to the grey matter of the nervous centres. There are two forms of glioma, the one hard, the other soft. The former has a considerable anatomical resemblance to simply sclerosed tissue; the latter, which is the more common, blends on the one hand with myxoma, on the other with small round-celled sarcoma. Gliomatous tumours of minute size sometimes stud the ependyma of the ventricles. They are usually found, however, in the substance of the hemispheres, more especially in their posterior lobes and in their upper and lateral parts. But they may be met with elsewhere in the nervous centres, and even in the spinal cord. They are for the most part solitary, of slow growth, and apt to attain a large size, as that of the fist, or even of the fœtal head. Owing to the great vascularity of the softer forms of tumour, they are liable to attacks of congestion, and to more or less abundant internal hemorrhage.

c. Sarcomatous tumours occur both in the dura mater of the brain and cord, and in the substance of these centres. They vary widely in their microscopical structure, and in their aspect and rapidity of growth. But they may be divided roughly into two forms—hard and soft. The former has some resemblance to fibroma, the latter is usually more or less translucent, white or grey, vascular, and, from its general resemblance to brain-substance, has been termed in other organs than the brain, cerebriform. Sarcoma of the cerebral dura mater generally occurs at the base, in the neighbourhood of the sella turcica or petrous bones; that of the theca vertebralis affects no special seat. Sarcoma originating in the nervous tissue is usually of the soft form and solitary, and often grows to a large size. In the brain its usual seats are, not the hemispheres, but the optic thalami, corpora striata, corpora quadrigemina, pons, cerebral peduncles, and cerebellum. It is only occasionally met with in the substance of the cord. Sarcoma originating in the nervous centres is seldom, if ever, malignant; the solitary tumours, therefore, which have just been considered, are not associated with the presence of similar tumours in other parts. On the other hand, it must not be forgotten that malignant sarcomas (melanotic and other) of other organs are apt to be attended with multiple secondary tumours in the substance of the brain. Primary sarcoma of the brain is mostly a disease of early childhood.

d. Carcinoma of the nervous centres, and of the parts about them, was formerly believed to be of common occurrence; but by all authors up to a recent date, sarcoma, glioma, and probably other forms of tumours, were all regarded as varieties of it. Carcinoma, in the restricted sense of the term, originates rarely if ever in the brain or cord, and not often in the bones and soft parts immediately surrounding them. Frequently, however, during the period of generalisation, it involves all these parts, and hence scirrhous, encephaloid, and mela-

notic tumours are not uncommon as secondary occurrences in the brain or cord, and in the membranous and bony parietes of these organs. Cancerous tumours therefore are generally multiple, and seldom reach a large size. Carcinoma of the skull, vertebræ, or periosteum of these parts, is apt in its progress to reach the surface of the brain or cord, and to involve these organs either by pressure or by direct extension; it is especially apt, moreover, to constrict the bony channels by which the nerves escape, to implicate the nerves, and finally to destroy them.

4. *Entozoa.*—The only entozoa which infest the brain of man are the cysticercus cellulosæ and the hydatid.

a. Although a considerable number of cases of cysticerci of the nervous centres are on record, they are met with very rarely. The cysts, which are of the size of a pea or horse-bean, vary in number from one or two to a hundred or more, and they occupy either the subarachnoid tissue, the choroid plexuses, or the nervous parenchyma. In the last case they are most common in the cerebral hemispheres; but they have been met with in the cerebellum, medulla oblongata, and other parts.

b. Hydatids of the brain are rare. They are generally solitary, but a couple or more have been found in the same case. They are almost always barren. Their size varies; but they not unfrequently attain a couple of inches in diameter before they cause death, and may be much larger. They generally occur in the substance of the cerebral hemispheres, but have been found in the cerebellum, and elsewhere in the nervous parenchyma. They also affect the meninges, and have been discovered in the lateral ventricles, and in the subarachnoid tissue of the cord. They seldom cause inflammatory changes in the surrounding parts, or other mischief than that arising from simple pressure. Neither do they appear ever to become the seat of suppuration. Hydatids of the brain are not unfrequently associated with hydatids of the liver or other organs. They are said to occur chiefly in persons between ten and twenty years of age.

5. *Aneurysms of the arteries at the base of the brain.*—We speak of these now only as tumours, and because from their bulk and situation they are exceedingly liable to interfere with the functions of important parts. They arise chiefly in the internal carotid arteries and their middle meningeal branches, and in the basilar; but they may also be found in other vessels, such as the anterior and posterior cerebrals, the anterior and posterior communicating branches, and at the bifurcation of the basilar. An occasional seat is that portion of the internal carotid which lies within the cavernous sinus. They usually vary in size from that of a pea to that of a marble, but have been met with as large as a hen's egg. From their position they are liable to compress some of the nerves at the base of the brain, and to indent the surface of the brain itself. They usually occur in persons over forty, but they

have been met with even at the age of puberty. Males are more liable to them than females.

Symptoms and progress.—1. *Brain.* The symptoms referrible to tumours involving the brain present the greatest variety:—a statement which is not likely to be disputed when one takes into consideration the various circumstances under which tumours arise, the different proclivities of different tumours, and the wide range of functionally distinct parts of the surface or substance of the brain which they may implicate. It is impossible, indeed, to draw up any scheme of symptoms generally applicable to cases of the kind; and we propose, therefore, to consider *seriatim* the more important symptoms which the presence of cerebral tumours may induce.

Vertigo is generally present at some period or other. Sometimes it is the first symptom of which the patient complains, and often it is the most constant.

Headache is for the most part a prominent symptom. In some instances it is one of the earliest, in association with vertigo and occasional vomiting. It is often persistent, but liable to exacerbations, and sometimes only comes on at irregular intervals. It may be little complained of, or even be wholly absent. It varies in character: is sometimes a sense of constriction or pressure, sometimes a feeling as though the head would burst, sometimes shooting, aching, or boring. It is referred to different parts in different cases: sometimes affects the vertex, forehead, or occiput mainly; sometimes shoots through the ears or temples—in th latter case probably involving the eyeballs, and associated with more or less intolerance of light. The situation of the pain is no sure guide to the seat of disease; nevertheless pain referred to the occiput and back of the neck is not unfrequently connected with disease in the posterior fossa of the skull.

Vomiting is a common symptom of many cerebral diseases, and is often an early indication of the presence of cerebral tumours. Indeed, it is well known, especially in reference to the tubercular tumours of children, that unaccountable vomiting is often the first warning of the affection which is in progress. The sickness often comes on at irregular intervals without obvious cause, is not unfrequently attended with nausea or loss of appetite, and is generally associated with constipation. It may continue on and off during the whole of the patient's illness, but is mainly a symptom of the earlier stages.

Slowness of pulse, with more or less irregularity, is of frequent occurrence, more especially during the period of invasion; subsequently also the same condition of pulse may prevail. But on the other hand it is then often of normal rate, or increased in frequency.

Hemiplegia and Hemianæsthesia.—Hemiplegia is no doubt entirely absent in a large number of cases, and when present usually comes on insidiously during the latter stages. There is, however, great variety as regards this symptom. In some instances almost the first indication

of disease is an apoplectic or epileptiform fit followed by hemiplegia. In some the attack of hemiplegia comes on suddenly in the course of other symptoms. And in either of these cases more or less complete recovery from the paralytic phenomena may ensue, to be followed by a relapse or by a series of recoveries and relapses. The hemiplegia generally follows the rule of ordinary hemiplegia in the fact that the arm is more affected than the leg, and the lower distribution of the seventh nerve than the other motor nerves of the face. But occasionally the paralysis is slight or limited, and reveals itself only in the face or arm. It may or may not be associated with numbness, tingling, or anæsthesia of the paralysed parts, or with hyperæsthesia, tenderness, or pain. Rigidity and contraction of the affected limbs may supervene.

Local paralyses are very common, sometimes in association with hemiplegia, sometimes independently of it. They are generally due, not as hemiplegia or hemianæsthesia is to disease involving the opposite corpus striatum, optic thalamus, or cerebral hemisphere, but to direct implication by pressure or by involvement in the morbid process of the nuclei of origin of the affected nerves, or of the nerves themselves. If, therefore, they be due to the same mass that causes hemiplegia, they occur on the opposite side of the body to the hemiplegia. But more tumours than one are not unfrequently present, and tumours of the crura cerebri, pons, or medulla, or growths in the neighbourhood of the circle of Willis, may readily involve directly several nerves of either side, even when causing at the same time distinct hemiplegic phenomena. In some cases there is paralysis of one or both external recti, leading to single or double internal squint; in some, paralysis of the whole or a part of one of the third nerves, involving ptosis, with paralysis perhaps of the internal rectus and an outward squint; in some the portio dura suffers, and Bell's paralysis is the consequence, probably associated with paralysis of the corresponding arch of the fauces; in some the hypoglossal becomes implicated. It is important in reference to these local paralyses to bear in mind that, contrary to what occurs in ordinary hemiplegia, the faradic contractility of the affected muscles rapidly disappears, and acute wasting is apt to ensue.

Implication of sensory nerves.—The fifth nerve occasionally suffers, either generally or in some of its branches; in some instances intense burning or neuralgic pains arise, in some tingling, numbness, or absolute anæsthesia. In the last case the surface of the eye, among other parts, becomes insensible, and consequently unconscious of irritation and liable to inflame. Sometimes from implication of one or both olfactory nerves, or one or both gustatory nerves, the sense of smell or taste is lost on one or both sides. As regards the ears, there is not unfrequently more or less deafness, with buzzing, rushing, or singing noises; and absolute deafness on one side may ensue. The most interesting and important complications, however, are those which involve the visual properties of the eye. We have alluded to the fact

of the occasional occurrence of double vision and of intolerance of light. But, besides these phenomena, we often meet with more or less obscurity of vision, which may go on to complete blindness, in one or both eyes; hemiopia, the field of vision being eclipsed in the identical halves of both retinæ; the appearance of muscæ; and other visual derangements. The presence of cerebral tumours is, moreover, almost always associated with optic neuritis, or that form of it to which the name of 'choked disc' has been given, and which may after a time result in more or less atrophy of the optic disc. The same rule applies to paralyses of individual sensory nerves as to paralyses of individual motor nerves: they are usually due to direct implication of the nerves or of their nuclei, and are observed therefore on the same side of the face as the cerebral tumour which causes them. The question whether they be dependent on local causes or on disease of the nervous centres above their nuclei can, in doubtful cases, generally be determined by the fact that in the former case reflex phenomena cease, in the latter they may be readily excited (M. Jaccoud). If, for example, the disease causing blindness be in the optic nerve, the pupil will be dilated, and will remain dilated when exposed to light, while, if it be situated above the corpora quadrigemina, the patient, though equally blind, will have free action of the pupil under the influence of the ordinary stimuli.

These various local sensory and motor affections may come on at any period of the disease; they are liable to appear and disappear before they become permanent; and they tend to increase in degree and in number with the advance of the disease.

Convulsions and spasms.—These constitute some of the most striking phenomena of cerebral tumours. They may be tonic or clonic, limited to the distribution of a single motor nerve, or implicating a group of muscles, a limb, the head and neck, or one side of the body. They may come on rarely and at distant intervals, or in frequent daily paroxysms, or may be almost continuous. And in either case they are apt to disappear wholly for a time, or to cease altogether. They are often distinctly epileptiform in character; but, unlike true epilepsy, are often unattended with loss of consciousness; or loss of consciousness comes on in the course of the attack instead of at the beginning. It is in cases of this kind, rather than in cases of simple paralysis, that, as Dr. Hughlings Jackson has shown, the seat and distribution of the peripheral phenomena point to the implication of definite cerebral areæ.

Intellectual and emotional disorders present great variety. In some cases one of the earliest indications of cerebral tumour is the occurrence of attacks, sometimes momentary, of incoherence, delirium, failure of speech, or loss of consciousness, associated or not with some partial convulsive movement or paralysis, or of attacks which may exactly simulate hysterical fits, or apoplectic seizures. On the other hand, these may be delayed until a late period of the disease, and may occur only as the

immediate precursors of death. Sometimes they come on at long and irregular intervals; sometimes they are very frequent, occurring many times a day, and even in long-continued sequences. In a large proportion of cases the patient suffers from gradually increasing failure of memory and hebetude: he becomes aphasic, or incoherent, or fatuous, and under such circumstances possibly loses, or fails to exert, control over his evacuations; or he gets delirious or maniacal; and associated with some of these mental derangements, we not unfrequently find him either given to boisterous laughter, or low-spirited and apt to cry.

Obstruction of venous sinuses.—Cerebral tumours occasionally cause obstruction either of the cavernous sinus or of the sinuses between this and the internal jugular vein; and, as a consequence, the veins of the eyelids and of the corresponding side of the forehead become more or less obviously distended. Similar dilatation of veins sometimes occurs in these cases, even when no obvious obstruction is present.

Lastly, it may be pointed out that bed-sores are often developed sooner or later—occasionally early in connection with the occurrence of irritative or inflammatory processes; more frequently late, when the patient is bed-ridden, paralysed, and fatuous.

We repeat that the symptoms due to cerebral tumours display remarkable diversity; nevertheless, careful attention to all the phenomena of the case will generally allow of a fairly accurate diagnosis being made. The onset of the disease may be gradual or sudden; and the symptoms which attend it may be of the most varied kind. The subsequent progress of the case is equally uncertain: sometimes the symptoms increase progressively and rapidly until death takes place; sometimes, and indeed in the great majority of cases, the patient is liable to remissions, or intervals of apparent restoration to health. But always such remissions become less and less marked with the advance of the disease, and at length continuous illness is established. The duration of life from the first development of symptoms varies largely; sometimes the patient sinks at the end of a few weeks; sometimes death is delayed for several years. But the commencement of symptoms cannot always be determined—especially when the cerebral tumours complicate other diseases. The causes of death are various. In some cases the patient sinks from innutrition and the formation of bed-sores; in some he is carried off in an attack of convulsions; in most, death is ushered in by coma.

It is not always possible to distinguish the symptoms of cerebral tumours from those caused by other affections of the same parts. Nor is this surprising, when we bear in mind: that many other diseases attack districts of the brain which tumours also affect, and that these as well as tumours are liable to be attended with swelling, inflammation, and softening of surrounding parts, and to produce both general and local symptoms. Among the affections here referred to are apo-

plectic effusions, embolic softenings, abscesses of the brain, and chronic diseases of the dura mater.

The determination of the site of a tumour must rest upon a consideration of the various details of the paralytic and other phenomena which the patient presents; and especially we may here be guided by our anatomical and physiological knowledge, and the ascertained facts of cerebral localisation. In many cases we may come to a fairly accurate conclusion on these points. But it must not be forgotten that insuperable difficulties are often presented by the fact, either that tumours are multiple, or that they occupy some tract within the hemispheres, or at their surface, lesions of which are not necessarily attended with hemiplegia or any specific nerve-phenomena.

Our recognition of the nature of a tumour must depend partly on our knowledge of the circumstances under which different growths are apt to arise, partly on our knowledge of the parts of the brain which they are severally most prone to affect, and partly on the duration of the disease. *Tubercle* occurs chiefly in children; the cerebral phenomena due to its presence are often remarkably slow in their evolution; and the disease is generally associated with tubercular disease elsewhere. Moreover, symptoms of tubercular meningitis are apt to supervene. *Syphilitic* tumours occur in adults who have usually either a distinct history of having contracted a chancre, or obvious traces of constitutional syphilis. They have, moreover, a remarkable tendency to affect the under part of the brain, and to involve the nerves there situated; to cause cephalalgia, defect or loss of smell, hemiopia, paralysis of oculo-motor nerves, deafness, paralysis of the portio dura, bulbar palsy, and above all, perhaps, trigeminal neuralgia or paralysis, and, in connection with these, the nutritive lesions, which have already been described. Further, it must not be forgotten that syphilitic patients are (even in the secondary period) liable to have sudden thrombotic occlusion of cerebral arteries, and symptoms identical with those attending embolism. Secondary *malignant* growths would be suspected if the patient were suffering also from a mediastinal tumour, or some form of malignant disease involving the skin, bones, mamma, uterus, or other organs. The presence of *hydatids* might be surmised if there were a total absence of all constitutional symptoms or taint and of all indications of local inflammation or softening, if moreover the patient were young, and especially if an hydatid tumour were detected in the liver or some other accessible organ. The symptoms due to *aneurysms* are generally much more obscure than from the position of the tumours might be supposed. Indeed, their presence is often not suspected until their rupture causes apoplectic phenomena and death.

2. *Spinal cord.*—Tumours involving the spinal cord, its membranes, or the nerves which spring from the cord, cause symptoms due partly to the compression or destruction which they effect upon the substance

of the cord, partly to involvement of the nerves, partly to local conditions of inflammation and the like.

a. Those which originate in the substance of the cord are attended with much the same symptoms as compression of the cord connected with vertebral caries. They cause more or less complete paraplegia in the parts which derive their innervation from the portion of cord below; and the distribution and character of the paralysis will necessarily vary according as the tumour is situated higher or lower in the cord, and according to the tract which it primarily involves, and its horizontal extension. There are some points, however, in regard to these tumours, which it is well to remember: their presence is rarely, if ever, attended with either central or peripheral pain; they originate mainly in the grey matter, and hence both sensation and motion are as a rule early affected; they commonly involve one side of the cord or some other limited portion of the cord, in the first instance, and hence induce irregular or cross paralysis, so that during the earlier period of their development there is very likely to be motor paralysis on the side of the lesion, and anæsthesia on the opposite side; and the progress of the paraplegic symptoms is liable to remarkable remissions. It may be added: that, owing to certain peculiarities as to their primary site, it is possible that their first symptoms may simulate those of locomotor ataxy or those due to lateral sclerosis; that they tend ultimately to produce absolute paraplegia; and that, wheresoever originating, they are liable to be followed by ascending and descending degenerative changes, and by spasms and contractions of the affected muscles, with more or less rapid wasting of some of them.

b. Tumours taking their origin in the meninges of the cord are apt at a very early period to implicate the sensory or motor roots of the nerves which are in relation with them. Hence arise (and sometimes before any paraplegic symptoms are developed) twitchings of certain muscles, followed by paresis, paralysis, and rapid wasting, and burning or quasi-neuralgic pains referred to the peripheral distribution of certain nerves (it may be in the first instance to a single spot)—pains which are subject to great variations, are often exceedingly intense, and are occasionally attended by cutaneous eruptions. The paraplegic symptoms of such cases are usually undistinguishable from those accompanying vertebral caries, and are (at all events in the first instance) due to compression of the cord alone. It is obvious that the distribution of the paralytic phenomena, and the order of their sequence, must depend largely on the position of the tumour and the direction in which pressure on the cord is applied.

c. Tumours which are primarily developed in the tissues external to the membranes, more especially therefore aneurysms and malignant growths, usually involve the sensory and motor nerves in the neighbourhood of their origin long before they involve the cord itself. These, far more even than tumours originating in the meninges, are thus apt

to induce severe sensory and motor troubles of limited distribution. The pain which they provoke is burning, wrenching, or crushing, constant, but liable to frequent exacerbations, which are often quite beyond endurance, and during which the patient grinds his teeth, groans, or actually shrieks. It is often attended with hyperæsthesia of the affected surfaces, and probably followed after a while by circumscribed anæsthesia, and by bullous or erythematous eruptions. The motor troubles are mainly paresis, and rapid wasting and contraction, of certain groups of muscles. The above phenomena occur with special intensity in cases of carcinoma involving the bodies of the vertebræ, owing partly to the tendency of the affected bodies to collapse, partly to the tendency to direct implication of the nerves. They are apt, moreover, not only to be exceedingly acute, but to have a comparatively wide distribution. Symptoms due to compression of the cord come on (if they come on at all) at a later period.

Assuming the presence of a tumour, its nature can only be determined in certain rare cases. If tubercle be ascertained to exist in other organs, we have some reason to suspect that associated paraplegic symptoms (if not due to vertebral caries) are due to a tubercular mass in the substance of the cord. If paraplegic symptoms come on during the reign of constitutional syphilis, we may have in that association a clue to the nature of their cause. If they be preceded by agonising pain, such as has been above described, we have grounds to suspect the presence of some tumour involving the vertebræ; and if they come on in the course of mammary or abdominal cancer, and especially if we find the spine presenting some localised obtuse bend in the neighbourhood of the point from which pain radiates and paraplegic symptoms begin, we have confirmatory evidence of the strongest kind.

Treatment.—The treatment of tumours, whether of the brain or cord, must be for the most part simply palliative. We must endeavour to relieve sickness by some of the various methods which are usually had recourse to for that purpose; to alleviate pain, either by the application of cooling lotions, ice, aconite, belladonna, or other sedatives, to the seat of pain, or by the internal exhibition of sedatives or narcotics, especially Indian hemp or opium; to calm convulsions or mental excitement by suitable remedies; to promote appetite; to keep the bowels free, and the patient clean; to prevent the formation of bed-sores; and generally to relieve symptoms as they arise. There are certain cases, however, in which treatment is of real value, either in arresting the progress of a tumour or in causing its removal. Tubercular masses are often of exceedingly slow growth, and may, in fact, remain quiescent for months or years. If we have reason to suspect the existence of such tumours, it is of course important to have recourse to iron, cod-liver oil, and other drugs and modes of treatment serviceable in tuberculosis. Tumours of syphilitic origin may often, if attacked early, be so far influenced by treatment that the patient becomes practically restored

to health; and, even if complete restoration be not effected, great and permanent amendment may ensue. Iodide of potassium and mercury are the drugs specially indicated in these cases.

IX. CEREBRAL AND SPINAL HEMORRHAGE. (*Apoplexy.*)

Causation.—Excepting those forms of hemorrhage (which have little clinical interest) occurring in the course of purpura, small-pox, and other specific disorders, and due to an abnormal condition of the blood, all hemorrhages within the skull or spinal canal are consequent on the rupture of blood-vessels. Rupture due to violence, as for example to blows on the head or spine, or to fracture of these parts, may of course occur at any age. Idiopathic hemorrhage, however, although it occasionally arises below the age of twenty, becomes common only after forty; from which time onwards its frequency in relation to the number of persons living at each successive lustrum rapidly increases. Old age, therefore, has great influence in its causation. But there are certain other conditions which are of more direct importance than even old age: these are the presence of chronic Bright's disease, and that of degenerative affections of the arterial system. It is more common in men than in women.

Morbid anatomy.—Hemorrhage may occur either between the dura mater and the bone, within the cavity of the arachnoid, in the subarachnoid space, in the nervous substance, or, lastly, in the ventricles.

1. *Meninges.*—Effusion of blood between the cranium and dura mater is not uncommon in adults, especially as the consequence of blows on the head or fractures of the skull, and is usually immediately referrible to laceration of the middle meningeal artery. The extravasated blood separates the dura mater from the bone in some limited area, and forms a convex protuberance, which displaces the cerebral surface in relation with it. If the patient survive, the blood undergoes those changes which are common to all such extravasations, and, after a while, becomes absorbed. Hemorrhage external to the theca vertebralis is also mostly due to mechanical violence. It may, however, result from the rupture of an aortic aneurysm.

Hemorrhagic accumulation in the cavity of the arachnoid is always referrible to escape of blood either from the dura mater or from the subarachnoid tissue. If the dura mater be its source, it may be either a direct consequence of mechanical violence, or derived from a patch of pachymeningitis, with hemorrhage between its laminæ. If the subarachnoid tissue be its source, it may be due to any one of the causes, to be presently discussed, of effusion of blood

into that part. The arachnoidean cavity appears to be a frequent seat of effusion of blood in new-born children, probably from violence in the process of being born. Blood escaping into this cavity readily diffuses itself throughout its whole extent. Here, as in other cases, if the patient live, the blood for the most part undergoes gradual absorption; occasionally, however, it gets converted after a time into a thin-walled cyst, full of limpid serous fluid, with little or no tendency to undergo further change.

Hemorrhage into the subarachnoid tissue is frequently due to the rupture of an aneurysm of one of the arteries at the base of the brain. It is generally then very abundant, and distends primarily all the lax tissue which abounds in this locality: encircling the vessels and nerves and concealing them from view, together with the surface of the crura cerebri, pons, and adjoining part of the medulla oblongata; and extending thence into the laminæ of the velum interpositum and the corresponding duplicatures connected with the fourth ventricle, along the fissures of Sylvius, and, according to circumstances, over more or less of the surface of the cerebral hemispheres and lobes of the cerebellum. Sometimes the blood escapes by laceration from a hemorrhagic cavity in the substance of the brain either into the ventricles or on to the surface. This accident is not uncommon in the neighbourhood of the island of Reil, in which case the centre of the meningeal extravasation will be the recess at the bottom of which the island is situated; it is apt also to occur when blood is effused into the pons or crus cerebri. Another cause of subarachnoid hemorrhage is punctiform extravasation, or extravasation from injury to the surface of the brain, as is caused by contre-coup. Subarachnoid hemorrhage is occasionally also observed in connection with the cord.

2. *Brain.*—Hemorrhage into the substance of the brain, especially if it be into certain parts of the cortex, may be due to laceration from violence; but it is a far more common consequence of the rupture of diseased vessels or of the miliary aneurysms which Charcot and Bouchard have shown to be frequently present mainly in the optic thalami, corpora striata, cerebral convolutions and pia mater, in cases of cerebral hemorrhage and in old people. The vessels in which rupture takes place are usually the seat of either fatty degeneration, calcareous deposit, or chronic arteritis, with hyperplasia of the corpuscles of the outer wall and perivascular sheath. The minute aneurysms which usually stud them vary, perhaps, from the size of a small pin's head downwards, but occasionally they are as large as a grain of wheat, or larger. The escape of blood in some instances, doubtless, is from a single aneurysm or vessel; but much more frequently it takes place simultaneously from many lacerations occurring within a circumscribed area. In some cases the hemorrhage is mainly from capillaries; it is then apt to be spotty, and a careful examination will probably reveal in the centre of each spot a capillary vessel, with

its lymphatic sheath distended with blood—a capillary dissecting aneurysm, in fact. In other cases, and more especially in those in which the effusion is considerable and in mass, the presence of miliary aneurysms, and even the ruptured aneurysms, can generally be easily recognised. But here also the rupture is first into the perivascular sheath, so that a dissecting aneurysm precedes the actual escape of blood into the surrounding nervous tissue. The quantity of blood which may be poured out into the brain-substance varies, roughly speaking, from a few minims to several ounces. Groups of minute or capillary extravasations are occasionally alone present; and generally, when a large hemorrhagic cavity exists, the tissues around are studded more or less abundantly with similar small hemorrhagic spots. The escaping blood necessarily tears up the brain-substance; and thus, when its amount is large, a very irregular cavity is produced, the interior of which is occupied by blood mingled with the débris of the broken-down nervous tissue, while the margins are formed by the irregular interdigitation of the lacerated brain-substance and of the peripheral portions of the clot.

The extravasated blood speedily coagulates, and if the post-mortem examination be performed shortly after its effusion, will be found to present the ordinary characters of recent clot. If, however, the patient survive, changes gradually ensue in it and in the brain-substance around. The irregularities of the cavity get smoothed away, its form becomes more rounded, and its margins denser and better defined. The clot contracts, grows drier and more friable, assumes a brownish or rusty tint, and gradually undergoes more or less complete absorption: the final result being the formation either of a cicatrix (which can only happen if the effusion were very small), or, as far more commonly occurs, of a cyst traversed by delicate processes of connective tissue, occupied by a thin serous or milky fluid, and studded as to its parietes with pigmentary particles and crystals of hæmatoidine. The time required for the total disappearance of a clot depends upon its size: a small one may be absorbed within a week or two, a large one within six weeks.

The effects of clots on the surrounding brain-tissue must not be omitted. In the first place, they always cause more or less displacement and pressure, and, if large, flattening of the convolutions, obliteration of the sulci, and displacement of subarachnoid fluid from a greater or less extent of the surface which overlies them. In the second place, the surrounding tissue, for some little distance, always becomes yellow from imbibition of the colouring matter of the clot, œdematous, and more or less softened. In the third place, they are very apt to set up inflammatory mischief in the parts which are in their immediate vicinity. And, lastly, at a later period, descending atrophic changes, which have already been described, are liable to supervene.

Hemorrhage may occur in any part of the brain; but it takes

place mainly in the corpus striatum, and is then generally due to laceration of some of the twigs of a particular branch of the internal carotid, to which Charcot calls special attention, and which we have already referred to. After the corpus striatum, the parts most likely to suffer are the optic thalamus and the white substance of the brain immediately external to these bodies. Hemorrhage occasionally also takes place in the crus cerebri, pons, or cerebellum, and, though much more rarely, in the medulla oblongata. Large effusions may implicate the optic thalamus and corpus striatum at the same time, and even destroy these bodies completely. More frequently, perhaps, they involve the corpus striatum and external capsule. They are very apt to rupture into the lateral ventricle, or, if they extend outwards, into the subarachnoid tissue in the neighbourhood of the island of Reil. In the latter case, more or less abundant effusion of blood takes place on to the surface of the brain; in the former case, one or both lateral ventricles, or the whole system of ventricles, including the fourth, becomes inundated with blood and sometimes enormously distended. Hemorrhage into the pons is not unfrequently continued thence by rupture either into the fourth ventricle or into the subarachnoid tissue below. It is not common for more than one extensive hemorrhage to take place in the brain at one time. But it is by no means uncommon to discover, after death, the remains of one or two or even more extravasations in addition to the recent one which has caused death.

Hemorrhage into the ventricles is almost always secondary to hemorrhage into the brain-substance or to rupture of aneurysms at the base.

3. *Cord.*—Effusion of blood into the substance of the cord is very rare. It depends no doubt in some cases on the laceration of diseased vessels; but in the great majority of cases is probably (as Charcot suggests) secondary to inflammatory softening.

Symptoms and progress.—1. *Brain.* The term 'apoplexy' is so commonly used to imply cerebral hemorrhage, and is on the whole so misleading when thus used, even if its scope be limited by the prefix 'sanguineous,' that it may be well to observe here that typical apoplexy—that condition in which the patient suddenly falls down in complete coma, with total abolition of motion and sensation, and of sense, with full pulse, and slow, stertorous breathing—is very seldom observed in cases of effusion of blood within the cavity of the cranium. Further, in most works, and especially in those of the older school, much stress is laid upon the type of body which is most liable to apoplexy, on the habits of life which predispose to it, and on the various symptoms which were supposed for weeks, months, or even years, to herald the approach of the actual seizure. It is certain, however, that although there was some amount of truth in the observations which led to these generalisations—a sort of rough connection between the collective antecedents above hinted at and the supervention at

some period or other of death, ushered in with an apoplectic seizure, or due to hemorrhagic effusion—there is little or no direct connection between them and the rupture of a blood-vessel in or on the brain.

In a large number of cases of cerebral hemorrhage the attack comes on suddenly and unexpectedly, although it may be freely admitted that in no inconsiderable proportion of them there has pre-existed, for a longer or shorter time, either chronic Bright's disease, or some distinct evidence that degenerative changes have been going on in the arterial system. In other cases there have been more or less distinct precursory symptoms, referrible to local disturbance of the cerebral circulation, caused either by partial obstruction of some artery or by the occurrence of capillary bleeding, or it may be by the actual formation of a hemorrhagic cavity which, either from its smallness or from its situation, is unattended with striking symptoms or permanent injury. Among the symptoms here adverted to may be enumerated headache, vertigo, confusion of thought, failure of memory, drowsiness, want of sleep, irritability of temper, and the like. Others are bleeding from the nose and retinal hemorrhage. But the most important are temporary paralytic phenomena, such as numbness or tingling on one side of the body or in the arm or leg, loss of power in the same parts, or in one half of the tongue or face, difficulty of articulation or deglutition, and double vision. It must not, however, be assumed that any of these symptoms necessarily points to the occurrence of hemorrhage. They may equally indicate the presence of a tumour or other circumscribed lesion, or be connected with epilepsy or other purely functional affections of the brain.

The symptoms which attend effusion of blood into the brain are very various both in kind and in severity. Sometimes the patient, while engaged in his ordinary avocations, suddenly finds that he has lost the use of his arm, and presently becomes hemiplegic; sometimes while engaged in conversation his articulation becomes thick, and he presently discovers that his mouth is drawn to one side, and that an arm and leg are limp and weak; sometimes the first intimation that there is anything amiss is the accidental discovery by the patient that one side of his body is totally useless when he attempts to rise from his bed in the morning, or from a chair in which he has been sitting quietly or dozing. In other cases the appearance of paralysis is attended or preceded by sudden giddiness or confusion of thought, or by a pain or sensation in the head which makes the patient cry out. In some instances he talks and acts for a few seconds like a drunken man. In some he suddenly becomes faint and collapsed, with pallid face, cold damp skin, feeble irregular pulse, and vomiting—the affection is ushered in indeed with an attack of syncope, during which he may become more or less completely insensible, but from which he often recovers. In exceptional cases only does the patient become at once insensible; and then the attack is apt to commence with a con-

vulsion. The last, it may be added, are for the most part cases in which blood is effused into the pons Varolii, or on to the surface of the brain from rupture of a large vessel or aneurysm.

The further progress of the disease presents the greatest variety. In some instances the patient's symptoms stop short at that indistinctness of speech or that unilateral paresis with which he was probably first seized; and he remains in this condition for a few hours, a few days, or a few weeks. In some instances these primary symptoms become aggravated up to the supervention of absolute hemiplegia, with or without anæsthesia; in which condition again the patient may remain for a variable time, sometimes recovering completely sooner or later, sometimes undergoing imperfect recovery, and remaining more or less feeble on the affected side, or in some degree inarticulate, for the remainder of his life. Not unfrequently headache, vertigo, impairment of intellect, or alteration of temper, not only is present during the continuance of the paralysis, but persists even after its amelioration or disappearance. Occasionally there is temporary deviation of the eyes, and even of the head and neck, towards the paralysed side. In many instances more or less profound coma presently succeeds the symptoms of invasion. This may come on in the course of a few minutes, or a few hours, or a few days, even in those cases in which the initial symptoms are of the mildest charactor. It generally supervenes before long in those whose first symptoms are those of shock: the patient recovers from his faintness, perhaps to find himself hemiplegic, not improbably to feel fairly well; but by degrees drowsiness and stupor creep on and gradually deepen into profound coma.

But, however the coma may come on, whether it be gradual in its invasion, whether it supervene in the course of symptoms already pointing to cerebral hemorrhage, or whether it become developed in all its intensity within a few minutes or a quarter of an hour of the first signs of illness, its symptoms do not on that account present any differences. The patient lies on his back insensible, with face more or less flushed, skin moist, pulse slow, perhaps irregular, but full and more or less hard, respirations slow and attended with stertor or snoring as he draws his breath in, and puffing of the cheeks as he exhales, and more or less depression of temperature which may continue for some hours. In the early condition of this, which is sometimes termed the apoplectic state, the patient is still perhaps capable of being roused; when spoken to loudly he makes some incoherent sound, when pinched or pushed he indicates by some movement or gesture that he is not altogether without feeling. But soon he becomes utterly unconscious. In this condition many various symptoms cluster, as it were, around his unconsciousness. In some cases he lies on his back quietly as if asleep, his expression placid, his limbs apparently unaffected assuming a natural attitude. But frequently there is some obvious muscular affection: the limbs of one or both sides are flaccid—when raised falling back

helplessly on the bed; or they are rigid, and offer more or less resistance to the attempts to move them; or convulsive twitches or more powerful spasmodic movements occur from time to time either generally or on one side of the body. There may, in fact, be simply that failure of the muscles to move which stupor alone involves, or there may be general or unilateral paralysis with or without flaccidity, rigidity, or convulsive movements. In the face the same conditions may be observed; sometimes the muscles are in repose; sometimes more or less obvious facial palsy is observed upon one side, sometimes twitching of the muscles. The eyelids are generally shut. The condition of the eyes varies: frequently the pupils are dilated, especially towards the fatal close; sometimes, and more especially in cases of hemorrhage into the pons, they are contracted; often they are natural; they are sometimes irregular, sometimes insensible to light. The last are symptoms of considerable significance. In the early state the patient, though unable to masticate, is still able to swallow fluid or food placed in the back of his mouth; when, however, the case is going on unfavourably, the power of deglutition fails absolutely. Respiration is, as has been stated, usually slow, but it is often irregular, and is liable to cease completely for some seconds. Sometimes the patient breathes as quietly as a child. But when a fatal termination is impending, stertor (if it were not before present) comes on; mucus and other fluids accumulate at the back of the throat and in the air-passages; the breathing becomes attended with loud rattling sounds, and the respiratory movements are often accelerated. The character of the pulse varies: at first probably it is slow, full, and hard, but it may be of natural rate, and present no deviation whatever from the normal; but with the continuance of coma it is apt to increase in frequency, and may rise to 120, 140, or 160 in the minute. The face usually is flushed, the skin more or less moist; and towards the end of life profuse sweats generally if not always break out. The patient has retention of urine, and loss of control over his alvine evacuations. Inability to swallow, accumulation of fluids in the fauces and air-passages, indifference of the pupils to light, failure of the eyelids to close when touched, extreme rapidity of pulse, and the occurrence of profuse perspirations are phenomena of the gravest omen.

In some cases the stupor of coma passes in the course of a few minutes, a few hours, or a few days, into that of death. But in a considerable number of cases the patient, after a longer or shorter time, slowly emerges from it, regains his consciousness more or less completely, and probably is found to be paralysed on one side, and to present a greater or less number of other indications of cerebral mischief. From this point, sometimes recovery is rapid and thorough; sometimes more or less complete hemiplegia continues temporarily, or, after more or less improvement, for life; sometimes he has hemianæsthesia as well as paralysis; sometimes his speech remains indistinct; some-

times he has more or less complete aphasia; sometimes he complains of headache or giddiness; sometimes he has loss of memory, failure of intelligence, or emotional perversion; or he may be stupid or demented, and then not unfrequently fails to control his bowels or his bladder. It is obvious that the various conditions here described are in the main identical with those which are apt to follow hemiplegia coming on without insensibility; and that, in fact, but for the circumstances that the supervention of coma on the whole implies either a large effusion of blood, or effusion into some vital part, and that coma itself brings with it special dangers, there is no essential difference as regards their subsequent progress between those cases of cerebral hemorrhage which are attended with coma and those which are and have been free from coma.

The character of the symptoms will be determined largely by the seat of lesion, by the size of the clot, and by the rapidity with which the blood is effused. When hemorrhage occurs in the corpus striatum, or in the white matter or convolutions of the brain in relation with the motor tract, or in the crus cerebri, motor hemiplegia will almost necessarily follow, and will probably be more complete according as the amount of brain-substance destroyed or compressed is larger. If, however, the effusion take place in the white matter of the hemisphere, paralysis is more likely to be absent than if it occur lower down; if it take place in the crus, it is probable that the third or fourth or some other of the nerves on the same side as the clot will also be implicated. Aphasia generally attends right-sided paralysis. Hemianæsthesia alone is rarely if ever present; but it is not unfrequently associated with hemiplegia, especially perhaps with hemiplegia of the left side; in which case the hemorrhage probably involves the optic thalamus, internal capsule, or crus. If blood primarily effused into the corpus striatum or other neighbouring parts escape with sudden violence into the ventricular cavities and flood them, the pressure which is at once exerted on a large number of ganglia essential to life induces sudden profound coma with general paralysis and flaccidity of the limbs. Also, if the surface of the brain be suddenly deluged with blood, profound coma almost immediately ensues, which is often attended with convulsions, but by no means necessarily with paralysis, and in some cases, especially if it be at the base, with inequality of pupils. When hemorrhage occurs into the pons, there are often convulsions, and usually sudden profound coma and general paralysis, attended at the commencement with contraction of pupils; and the case is rapidly fatal. Sometimes, however, the effusion here is small in amount and unsymmetrical in position, in which case the paralytic symptoms will probably be more or less irregularly distributed; there will perhaps be hemiplegia, with more or less complete implication of various sensory and motor nerves, situated either on the side opposite to the hemiplegia, or irregularly on both sides; there is apt also to be more or less

serious interference with the muscles of speech and deglutition—the usual symptoms, in fact, of bulbar paralysis. Hemorrhage into the cerebellum is often attended with severe occipital pain, vomiting, and especially with vertigo. Paralysis is for the most part absent, but the patient, if able to walk, staggers like a tipsy man.

We have already pointed out that a patient who has had cerebral hemorrhage, whether he has had coma from which he has emerged, or whether he has had a simple attack of paralysis and has attained that stage at which all present fear of coma has passed away, may rapidly or slowly recover from all his symptoms, may recover imperfectly, or may remain without any improvement whatever. We have not, however, referred to the important fact that various complications may arise in the progress of the case. The principal of these may be briefly considered. *a.* The presence of a clot, and of the collateral œdema which always attends its presence, is very apt to induce at any time during the first few weeks after its formation some inflammation in the surrounding brain-tissue—an occurrence which is often indicated by more or less elevation of temperature, rapidity of pulse, return of paralysis, drowsiness, and impairment or loss of control over the emunctories, and may lead to coma and death. *b.* Bed-sores are apt to form. In some instances these come on when the patient is bed-ridden, or has continued for some length of time in a fatuous or semi-comatose condition, just as they may come on in any other persons who are confined to bed and of uncleanly habits. But they are also apt to appear, and then mainly upon the buttock of the paralysed side, from the second to the fourth day after the attack, apparently in consequence of some direct influence transmitted from the seat of lesion in the brain. The formation of these early bed-sores is always a bad sign, and almost without exception foretells an early fatal issue. *c.* Inflammation of internal organs, such as pneumonia, dysenteric ulceration, and the like, occasionally supervenes. *d.* Not unfrequently, after the second or third week, or later, if the limbs remain paralysed, rigidity and contraction gradually ensue, associated after a while, in some cases, with wasting of the muscles. This rigidity is not to be confounded with the temporary irritative rigidity sometimes observed at the commencement of paralysis, but is the consequence of secondary degenerative changes in the course of the lateral columns of the spinal cord, and is permanent. It is observed by Trousseau that in those rare cases of hemiplegia in which the arm recovers more rapidly than the leg, the prospects of the patient are very gloomy; that the leg becomes stiff and painful; that imbecility comes on, and the patient usually dies within the year. Whatever the explanation of the imbecility in these cases may be, it seems pretty certain that the arrested recovery of the leg is sometimes due to the fact that degenerative disease has already commenced in the cord. *e.* All patients who have had one attack of cerebral hemorrhage are specially likely to have subsequent attacks; and sometimes two or three of these occur

at irregular intervals, previous to the fatal issue of the case, adding complexity to the patient's symptoms. Lastly, partly from the effect of the primary lesion, partly from the associated diseased state of arteries, partly from pressure, œdema, inflammation, or degeneration of surrounding parts, many additional symptoms are liable to come on—among others athetosis, epileptiform attacks, delirium, mania, or dementia.

2. *Cord.*—Hemorrhage into the arachnoid cavity or subarachnoid tissue, or into the substance of the cord is so rare, except as a consequence of injury or pre-existing disease which has already caused serious symptoms referrible to the cord, that it is scarcely necessary to discuss the symptomatology of these lesions. It is sufficient to say that hemorrhage around the cord will naturally cause the symptoms of pressure—namely, more or less loss of voluntary motor power, associated with little or no impairment of sensation; and that the effects will vary according to the seat of the effusion, its extent, and the degree of pressure exerted by it; and that hemorrhage into the substance of the cord will be attended with precisely those symptoms which occur in inflammatory softening involving the whole thickness of the cord.

Treatment.—When a patient is seized with sudden paralytic symptoms due, as we suppose, to hemorrhage, there is little to be done save to keep him perfectly quiet, mentally and bodily, to make him lie down with his head somewhat elevated, in a room of equable but not elevated temperature, and to feed him sparingly with milk and farinaceous food. There is no harm, probably, even if there is no good, in giving him cooling drinks, and in administering medicines which are supposed to check hemorrhage. A powerful purgative is often given, but it is questionable whether the straining which attends its action is not more injurious to the patient than the retention of fæcal matter in the bowels. If coma have come on, again there is little to be done beyond leaving the patient at rest. Some bleed, but bleeding will not benefit those who have large effusions of blood in the pons or ventricles, or on the surface; and those who have large hemorrhages elsewhere for the most part recover from their coma without any such assistance. Further, bleeding is probably quite incompetent to arrest cerebral hemorrhage. Nevertheless, we are inclined to believe that the guarded removal of blood in these cases may sometimes prove beneficial by diminishing pressure within the skull, or, as Sir Thomas Watson suggests, by relieving the congestion of the right side of the heart, which is often manifested by engorgement of the veins of the head and neck, and lividity of surface. At all events a single bleeding will probably have no injurious effect whatever. It is customary to give powerful purgatives in these cases, such as a couple of drops of croton oil alone, or mixed with a little castor oil; and on the whole the practice appears to be good; purgation tends to derive (as the

expression is) from the head; and in cases of profound coma does not induce that powerful straining which is so great an objection to its employment when the patient is sensible. Other measures which may be adopted are the application of cold in the form of evaporating lotions or ice to the shaven head, or of counter-irritants, such as mustard plaisters, to the head, back of neck, and legs. During the further progress of paralysis following hemorrhage, the chief things to do are still to keep the patient quiet, and free from either mental or bodily excitement, to regulate his hours and employments, to keep his bowels regular, if necessary by the use of opening medicines, to relieve all discomforts and secondary affections under which he may happen to labour, to counteract, as far as possible, the effects of any renal or other organic disease of which he is the subject, and to attend very carefully to his diet. As a rule, all alcoholic beverages should be interdicted, or, if circumstances render their use necessary, should be allowed only in small quantities, and in a dilute form. The patient should be well nourished, but the amount of food given him should not exceed what is essential for his well-being. The food, moreover, should be wholesome and readily digestible. It is often recommended that the patient should be restricted to a vegetable diet and milk—a diet which is doubtless very appropriate if there be any chronic renal affection. But if his abdominal viscera be healthy, we do not see how such diet should have any superiority over a diet containing a fair proportion of animal food. As regards the affected limbs, friction and faradism are sometimes efficacious, when the acute symptoms have passed away, in promoting the restoration of the impaired motor powers; and when late contraction is occurring it may be relieved or prevented by galvanising the contracting muscles and faradising their opponents.

X. OBSTRUCTION OF CEREBRAL ARTERIES.

(Thrombosis. Embolism. Softening.)

Causation and morbid anatomy.—We have drawn attention to the facts: that the group of arteries supplying the brain, although anastomosing freely in the circle of Willis, have no further communication with one another excepting by means of the capillary vessels situated at the periphery of their several areas of distribution; that the same arrangement holds good with respect to every branch of these arteries, down to their smallest twigs; and that hence any obstruction, however produced, whether in a primary or in a subordinate branch, no matter how small, puts a stop to the circulation of the blood in the district to which the obstructed vessel leads, and involves its degeneration and

death. The same rule obviously does not apply with equal force when obstruction takes place in the basilar or either internal carotid artery below the anastomosis, since by means of that anastomosis blood for the most part finds its way readily from the pervious trunks to the branches of the obstructed vessel. Nevertheless, such obstruction, or even obstruction of the common or internal carotid in the neck, occasionally influences seriously the nutrition of that portion of the brain with which the obstructed vessel is in relation. The arteries of the cord, on the other hand, are small, are derived or reinforced from many sources, and rarely, so far as we know, become obstructed, or if obstructed, instrumental in the production of degenerative changes in the substance of the cord.

The causes of obstruction are various. *a.* In many cases the arteries at the base of the brain in persons advanced in life get rigid, thick-walled, and the seat of atheromatous or calcareous degeneration; and as a consequence of the advance of these processes it sometimes happens that one of the diseased vessels becomes reduced in calibre or altogether impervious. *b.* In a considerable number of cases, again, one of the arteries at the base of the brain or one of the primary branches becomes obstructed in a greater or lesser portion of its length by a clot or thrombus, which fills it, adheres to its surface, and after a while undergoes degenerative changes, in which also the vascular parietes probably share. The causes of such thrombosis are not always obvious. Occasionally it is due to the fact that the affected vessel leads to some diseased tract in which the smaller vessels are involved and obstructed; and the coagulation of blood in it is therefore secondary. Sometimes, possibly, it is due to the special tendency which the blood appears to have in some dyscrasic conditions to undergo spontaneous coagulation. Sometimes it is determined by disease of the arterial walls, such as roughening from atheromatous or other chronic processes, inflammatory thickening, or syphilitic growth. *c.* But perhaps the most interesting cause of obstruction is the detachment of granulations from diseased valves on the left side of the heart, their conveyance to the arteries of the brain, and their impaction, usually at the point of bifurcation of an artery, or at some other spot where the vessel is too small to allow of their further transmission. The embolus usually forms the nucleus for the development of a thrombus extending to a greater or less distance in either direction along the channel of the obstructed artery.

Obstruction of the arteries at the base from atheromatous or earthy degeneration is observed mainly in persons advanced in life, and especially in those suffering from arterial disease elsewhere, or who are the subjects of chronic renal disease, or have led laborious or debauched lives. The obstruction usually occurs in one of the arteries forming the circle of Willis, or in one of the trunk-vessels below this anastomosis.

Thrombosis is not uncommon in the vertebrals, the basilar, the internal carotids, and their several primary branches; and indeed not very unfrequently involves two or three of these vessels one after the other at irregular intervals. Obstruction from thrombosis connected with arterial degeneration is an affection of advanced life; as a consequence of syphilis, it is mainly a phenomenon of early adult life and middle age.

Embolism is in the great majority of cases the consequence of rheumatic inflammation of the valves of the heart; it may, however, follow degenerative lesions of the same parts and the detachment of masses of calcareous or atheromatous matter, or of fibrinous tubercles which have become developed on the degenerate surfaces. Embolism may occur at almost any period of life, yet is certainly most common from puberty up to the age of forty or fifty. It almost always affects the middle cerebral artery, some part of its course, and usually the middle cerebral of the left side.

The changes in the brain-substance which result from arterial obstruction are (excepting when this takes place below or in the circle of Willis) almost accurately limited to the district which the obstructed vessel supplies. The affected region becomes opaque white, yellowish, or greenish, mottled with light red patches, or even minute extravasations of blood, and softened—often so soft as to break down readily into a pulp under the finger, or to admit of being washed away under the impulse of a stream of water. The microscopic characters which it presents depend on the appearance of granule-cells in greater or less abundance, on the degeneration of the nervous elements, more especially the white substance of Schwann, and its conversion into masses of refractive globules, and on the accumulation in the walls of the vessels and perivascular sheaths of more or less numerous fatty granules. Patches of softening from obstruction, especially if of small size, may, like apoplectic clots, become absorbed, and leave behind them a mere scar or a cavity containing serous or milky fluid. If of large size, they may undergo more or less diminution of bulk, and involve obvious shrinking of the mass of brain in which they are contained. Not unfrequently inflammatory changes go on in the brain-substance around them.

Symptoms and progress.—The symptoms due to obstruction of one of the cerebral arteries so closely resemble those caused by hæmorrhage that, if there be no appeal to other facts than those afforded by the cerebral symptoms which are present, it is utterly impossible in the great majority of cases to distinguish the one affection from the other. It is stated by Recamier and by Todd, and their views are supported by Trousseau and many others, that whenever hemiplegia, complete and absolute, occurs suddenly without loss of consciousness, it is due to softening and not to hemorrhage. And in reference to this statement, we may point out that the mere sudden loss of function in a

limited portion of brain-tissue (as occurs in softening) is likely to be attended with less general disturbance of the cerebral functions than the extravasation of blood into a similarly limited space, which not only destroys the tissues which it infiltrates, but, from its bulk, causes more or less serious pressure on surrounding parts. The rule may doubtless, within certain limits, be accepted as the expression of a fact; but it is a rule to which there are frequent exceptions; for in many cases of softening from arterial obstruction, the hemiplegia, if sudden, is not complete, and in some the attack is ushered in by loss of consciousness; while, on the other hand, hemorrhagic hemiplegia, as we know, is in a large number of cases unattended with insensibility.

Various prodromal symptoms of cerebral softening are often enumerated; but it is clear that in most of the varieties of softening now under consideration no symptoms of the kind are likely to be met with. They can attend neither embolism nor thrombosis. And any that may be referred to disease of the arterial walls are equally indicative of future hemorrhagic effusion. As a matter of fact, the symptoms due to arterial obstruction are always sudden in their onset, and for the most part occur unexpectedly at a time when perhaps the patient seems to be in perfect health. The seizure comes on in various ways: sometimes the patient, who is walking or making some exertion, or perhaps even sitting down quietly, is attacked with sudden vertigo, and more or less confusion of thought, and tumbles or throws himself forward on the ground; sometimes he is seized with sudden pain in the head of such severity that he cries out; sometimes he becomes suddenly faint, and occasionally this faintness is attended with a slight convulsion. But, however various these initial symptoms, it is almost always discovered so soon as the momentary attack has passed that more or less complete hemiplegia is present. It is almost needless to say that the character of the symptoms which present themselves and their severity must depend largely upon the size of the vessel obstructed and the part to which it is distributed; in other words (as also in hemorrhage), upon the amount of brain-substance which becomes incapacitated, and on its situation. Thus affections of the posterior cerebral lobes and of the cerebellum are always more obscure in their symptoms than those which involve the anterior parts of the brain, and more especially than those which involve its base. And hence it will be readily understood that obstructions arising in the course of the posterior cerebral and cerebellar arteries lead to less definite, if not less serious, consequences than obstructions in the course of the anterior and middle cerebrals. It is very important, however, for the purposes of diagnosis to refer to the exact distribution of the various intra-cranial vessels which has been given on a former page. And especially is it important to bear in mind: that it is from the basilar artery that the pons mainly receives its vascular supply; that the posterior cerebral artery is distributed not only to the posterior part of the cerebrum, but especially

to the posterior part of the optic thalamus, and to the corpora geniculata and quadrigemina; and that in the great majority of cases, and certainly in almost all cases of embolism, the obstruction occurs in the middle cerebral artery or in one of its branches, and that the tract which then undergoes softening is the district to which this vessel is distributed, or some part of that district, which includes the greater portion of the corpus striatum, the internal capsule, the anterior half of the optic thalamus, and nearly the whole of the antero-lateral region of the brain, inclusive of the island of Reil and the convolutions which surround the fissure of Sylvius. It is hence obvious in the case of embolism: that the main symptom which the patient would be expected to present is more or less complete (generally complete) hemiplegia, not improbably associated with some degree of anæsthesia, and more or less profound impairment of intelligence; and that if the disease occupy the left side of the brain, either marked aphasia, or total inability to utter articulate sounds, will be present. Other symptoms, however, which are not special to softening, are generally associated with these, namely vertigo, headache, sickness, rigidity of the affected limbs, loss of control over the bladder and rectum, and the like.

The subsequent progress of cases of obstructed cerebral arteries depends largely upon the extent of softening and its situation. If the patch be small (even if it be in the distribution of the middle cerebral) recovery as complete as occurs after some cases of effusion of blood may be expected. In most cases, however, recovery does not take place; in some the patient improves up to a certain point; in some he remains, so far as his mental and motor failures are concerned, much as he was immediately after his seizure. Occasionally, and especially if the case be one of thrombosis and not of embolism, several of the cerebral arteries, and even the main trunks of these vessels, may be obliterated at successive intervals, each attack adding its own special symptoms to those which had resulted from previous lesions. It remains a fact, however: that the patient rarely recovers completely from the effects of thrombotic or embolic softening; that if he has become aphasic, the aphasia continues in a greater or less degree; that hemiplegia for the most part persists, and is followed ere long by that form of contraction which results from secondary lesion of one of the lateral columns of the cord; and that occasionally arthritic effusion or inflammation ensues, or wasting of muscles. Further, the intellect, already probably impaired, is apt to fail, and the patient after a while to become bed-ridden and childish. It must be added that bed-sores occasionally form rapidly after softening, as they do after hemorrhage; and that inflammatory processes may go on around the softened patch and bring with them special symptoms. It follows on the whole that the prognosis in cases of softening is very unsatisfactory, and that even if patients survive they are apt to survive in a more or less maimed or wrecked condition. Death may occur at any period. Sometimes it

comes on early, the patient dying from coma, bed-sores, or failure of nutrition, or from pneumonia, or some other such complication. Death at a later period may be consequent on the recurrence of apoplectic attacks or on the supervention of inflammation around the focus of disease; or it may be due to asthenia or intercurrent disorders.

There is for the most part extreme difficulty in determining of any case which comes before us whether it be one of sanguineous effusion or one of softening from arterial obstruction. In many cases, indeed, there is nothing whatever to aid us in coming to a differential diagnosis. The chief points on which reliance must be placed are the following:—First, our knowledge of the relative seats of hemorrhage and of softening and of the different symptoms which they are hence likely to evoke. Second, the clinical history of the patient and the state of his various organs: thus the case is likely to be one of embolism if there be heart-disease present, or if there be a history of his having had former cardiac mischief, or even if he have had an attack of acute rheumatism; it is not unlikely to be one of thrombosis if the patient have had a chancre, or if he be suffering, or present traces of having suffered, from the secondary or later symptoms of syphilis; it is almost certain to be hemorrhagic if we discover the presence of albuminuria or chronic renal disease, arterial degeneration, or hemorrhage into the retinæ. And, third, the age of the patient—cerebral hemorrhage being on the whole a disease of advanced life, embolism occurring indifferently at all ages from puberty upwards. We need scarcely repeat that the occurrence of sudden and complete hemiplegia without loss of consciousness and without premonitory symptoms points strongly to arterial plugging, while the gradual development of symptoms culminating in hemiplegia and coma is strikingly characteristic of cerebral hemorrhage.

Treatment.—It is needless to lay down any specific rules of treatment. We cannot reopen an obstructed artery; we cannot hope that the area to which it leads shall be fed by collateral channels. The softened part remains necrosed, and the best thing that can happen is that it shall shrink into an inert mass or undergo absorption. It may, however, during this process induce inflammatory mischief in the parts around. This contingency should be guarded against as much as possible. For this and various other reasons, the patient should be kept quiet and cleanly, his bowels should not be allowed to become constipated, and his food should be nutritious, but easy of digestion, and not too abundant. But, indeed, the same general treatment is applicable to these cases as to cases of paralysis after hemorrhage, and need not be more particularly considered.

XI. HYDROCEPHALUS AND HYDRORRHACHIS.

(Cerebral and Spinal Dropsy.)

Causation and morbid anatomy.—Dropsical accumulations in the cavities connected with the brain and cord are not uncommon, their chief seats being the subarachnoid space and the ventricles. A relative excess of subarachnoid fluid, which has been mistaken for dropsy, is generally observed in connection with the shrunken brains of old persons and of those who die demented or fatuous, or the victims of certain other chronic forms of insanity. Whenever any portion of the brain, whether from congenital defect or as a consequence of disease, wastes, the space which it formerly filled becomes occupied either by an excess of subarachnoid fluid or by fluid accumulated in a local dilatation of one of the ventricles. Further, effusion of serum attends the progress of many morbid conditions, such as inflammation, morbid growth, and softening: thus in some cases of meningeal inflammation, inflammatory products with excess of fluid accumulate in the subarachnoid space; in some cases the substance of the brain becomes wetter or more succulent than natural, or serous infiltration (collateral œdema) occurs in the vicinity of foci of disease; and in some cases (and these are the most frequent and important) the lateral ventricles, the third ventricle, or the fourth ventricle, or all of them together, get largely distended with fluid. In the last series of cases the accumulation of fluid in one situation is balanced by its removal from other situations; and hence, as a rule, excess of fluid on the surface is attended with comparative absence of fluid from the ventricles; and dropsy of the ventricles or substance of the brain causes flattening of the convolutions, obliteration of the sulci, and dryness of the subarachnoid tissue. Dropsical effusion plays a more or less important part in the production of symptoms in the cases which it complicates; but by far the most important, in this respect, is the intra-ventricular dropsy, which is so commonly associated with the presence of tubercles or other tumours of the brain, or of meningeal inflammation, and which occasionally arises (both in children and in adults) as an independent malady. This is often referred either to compression or obstruction of the venæ Galeni, or to closure of the communication with the subarachnoid tissue which exists at the posterior extremity of the fourth ventricle.

The most important and striking forms of dropsy are congenital or come on without obvious cause shortly after birth, sometimes associated with malformation, sometimes independently of it.

1. Among the former of these classes must be included: in connection with the brain, hydro-meningocele and hydrencephalocele; in connection with the cord, spina bifida. *a.* In *hydro-meningocele* and

hydrencephalocele, a congenital perforation is present, either in the occipital bone (which is most common), or in some other part of the vault of the cranium; through which protrudes, in the form of a tumour, either the membranes of the brain alone, with a circumscribed accumulation of serum (hydro-meningocele), or a portion of brain nipped off, as it were, from the rest, and usually containing within it a dilated dropsical diverticulum from one of the ventricles (hydrencephalocele).

b. Spina bifida generally occurs in the sacral or lumbo-sacral region, but may be met with in the neck or any other part of the spine. It forms a rounded tumour, usually with a central dimple, and is due partly to the fact that the arches of the vertebræ in the situation of the tumour have remained ununited, and partly to the fact that the membranes of the cord are there expanded and distended with dropsical fluid, and protrude through the abnormal fissure. In some instances the membranes alone protrude, and we have then a condition which is equivalent to hydro-meningocele. But much more commonly (especially if the disease be at the lower end of the spinal canal), the cauda equina is prolonged into the cavity. The filum terminale is then attached to the centre of the concavity of the cyst, causing the dimple to which reference has been made; and the nerves of the cauda equina accompanying this to the posterior aspect then arch forwards across the cavity—double upon themselves—to reach their several foramina. If (as sometimes happens in such cases) the central canal of the implicated portion of the spinal marrow be dilated into a cyst, we have a condition which is the exact counterpart of hydrencephalocele.

2. Congenital or early developed dropsy (independent of malformation) probably always occupies the ventricles; although in some cases, apparently by accident, fluid becomes effused also into the cavity of the arachnoid. *a. Chronic hydrocephalus* (as it is generally called) sometimes commences during the later period of intra-uterine life, and the child is born already hydrocephalic. More frequently, however, the first manifestation of the disease occurs between the time of birth and six months after that event. But it may come on at any time previous to the union of the cranial sutures; and a few cases are recorded in which the supervention of dropsy shortly after this union has caused the bones again to separate. The fluid of hydrocephalus is of higher specific gravity than cerebro-spinal fluid, and contains albumen, chloride of sodium, and urea. It mostly occupies the lateral, third, and fourth ventricles; and its gradual accumulation leads to their dilatation, to the flattening of the various projecting ganglia, to the rounding of the several cavities, and to the enlargement of their orifices of communication. In this way the lateral ventricles may become enormously dilated, the lateral walls of the third ventricle may be opened outwards, until they become horizontal, and lost, as it were, in the common floor of the general ventricular cavity, and the foramen of

Monro and the fissure passing thence backwards beneath the fornix may be so much dilated as to form a free arch-like communication (of which the expanded third ventricle forms the floor) between the two lateral ventricles. The dilatation is not, however, always uniform or general. In some cases one lateral ventricle is much more enlarged than its fellow, or one part of a ventricle much more expanded than another part. In some cases, indeed, the posterior cornu becomes isolated from the rest of the cavity, and forms an independent cyst. Sometimes the third or fourth ventricle remains unaffected; sometimes the dropsy is confined to one of these cavities.

The effect of the gradual distension of the lateral ventricles upon the cerebrum is remarkable. We have pointed out that the various elevations and depressions in the ventricular walls become effaced, and that the lateral ventricles tend to communicate freely with one another in consequence of the displacement upwards of the corpus callosum, septum lucidum and fornix. Concurrently with these changes the convolutions on the surface of the organ become unfolded, until finally, in extreme cases, their grey matter forms a continuous smooth plain over the dilated hemispheres, which at the same time become reduced in thickness to a half or quarter of an inch, and in some situations, probably to that of writing paper. Under these circumstances the dilated ventricles not unfrequently contain several pints of fluid; indeed, cases are quoted by Trousseau in which 30, and even 50 lbs. were found in them. Occasionally, as in the well-known case of the man Cardinal reported by Dr. Bright, rupture of the surface of the brain or of its meninges occurs in the course of the disease, so that the fluid originally contained in the ventricles accumulates in the cavity of the arachnoid, and the brain lies collapsed and empty on the floor of the skull.

The effects of hydrocephalus on the skull, on the nervous functions, and on the development of the child, are very important. As the dropsy increases, the head gradually enlarges at the expense of its lateral and upper part; the two halves of the frontal bone, the parietal bones and the occipital bone open (as Trousseau expresses it) like the petals of a flower, and are thrown outwards, while the intervals between them become proportionately widened. Consequently the forehead, the sides of the skull, and the occipital region all protrude, while the head becomes somewhat flattened at the top. At the same time, some want of symmetry is usually apparent. The inordinate size and strange shape of the skull impart to the comparatively small face below it a peculiar aspect, which is aggravated partly by the emaciation which is usually present, and partly by the influence of the enlarging skull on the orbits and eyelids. The upper walls of these cavities are displaced downwards by the pressure to which they are subjected from above, while the upper eyelids, with the eyebrows, are drawn upwards over the forehead by the tension of the stretched pericranial integuments. The

eyes consequently become prominent, and present a peculiar staring character, due to the fact that the sclerotic coat is habitually visible above the upper margin of the cornea.

The integuments of the head become attenuated and tense, the superficial veins remarkably distinct, and the hair scanty and poor. The displaced bones also become thin, and the serrations of their edges irregular and straggling. Further, as the case progresses, nuclei of ossification appear irregularly in the tense membrane which intervenes between the separated bones, and these grow into irregular osseous plates, termed ossa triquetra. After a time, with the aid of these intercalated bones, the sutures and fontanelles may become entirely closed. This closure, however, may not take place for twenty years or more. The patient almost always emaciates notwithstanding that his appetite may continue good, the frame remains undeveloped, and the limbs are puny and shrunken.

b. Internal hydrorrhachis, or dropsy of the central canal of the spinal cord, is probably, like hydrocephalus, an affection of congenital origin or of early infancy. As has already been stated, it is sometimes associated with spina bifida; it is sometimes also an accompaniment of hydrocephalus. The canal may be dilated more or less irregularly in its whole length, or may present circumscribed dilatations only, and may vary from a quarter of an inch to an inch in diameter.

As regards the ætiology of chronic hydrocephalus and hydrorrhachis commencing in fœtal life or early infancy, we can only say that it is said to occur specially in rickety children, and in children of scrofulous or unhealthy parentage, and that its immediate cause is probably chronic inflammation or some condition allied to inflammation, involving the lining membrane of the affected cavities.

Symptoms and progress.—1. The symptoms of chronic hydrocephalus are to a large extent comprised in the pathological account of the disease which has already been given, or may be surmised from the anatomical facts in relation to it there considered. As regards the invasion, it may be observed that in some cases progressive enlargement of the head, and the gradual supervention of the characteristic physiognomy of the disease, are the earliest indications of the presence of hydrocephalus; while in some instances epileptiform convulsions, repeated from time to time, or other symptoms indicative of brain-disturbance, precede the appearance of any obvious change in the form or size of the skull. We may arrange the symptoms of the disease under three heads: namely, those dependent on the progressive enlargement of the skull; those connected with the general nutritive functions; and those which depend on the involvement of the nervous centres. *a.* The general shape which the head acquires, and the peculiarities presented by the stretched integuments, the eyes, the eyelids, and the face have already been sufficiently described. We may mention, however, that fluctuation can generally be easily perceived in

the course of the open sutures, and that these parts may often be seen to collapse with inspiration and to dilate with expiration. Occasionally, as in the case of Cardinal, the dilated head, like a hydrocele, is more or less transparent. The increasing size and weight of the head render it before long difficult for the child to support it, and tend, among other things, to delay the acquisition of the power of walking. The young babe is apt to rest its head constantly on the pillow, or on the nurse's lap, rolling it about from time to time; and even when the child can walk, it still has frequently to lay its head down, or to support it with its hands, and, under any circumstances, walks with a slow and cautious gait. The latter peculiarities may be continued throughout adult life. *b*. The general nutritive functions are almost always seriously impaired. The child probably takes food well—nay, greedily; but, notwithstanding this, it remains undersized and weak, and its face, trunk, and limbs become, as a rule, emaciated and shrivelled. The bowels are often confined. *c*. Not only are epileptiform convulsions often among the earliest symptoms of hydrocephalus, but similar convulsions, or attacks of laryngismus stridulus, are very apt to come on at a later period of the disease; and even if they have been absent before, they may supervene at the time of puberty, or later. These, however, are not the only nervous phenomena present. The child is generally fretful and dull, its sight becomes impaired and sometimes lost, and occasionally also deafness ensues; the limbs are liable to spasmodic twitches; and not unfrequently the muscles, and more especially those of the lower extremities, become rigid; they may also undergo atrophy. With the advance of age, we generally find gradually increasing hebetude or idiocy—loss of memory, incapability of mental exertion, or some special incapacity for learning; we probably find, too, that the patient becomes irritable, passionate, or morose. Nevertheless, he occasionally remains fairly bright and intelligent.

The duration of life is various. Hydrocephalic fœtuses not uncommonly die in the act of birth. Death usually occurs, however, during the first or second year, from either convulsions, coma, or intercurrent disorders. But life may be prolonged for five or ten years, or longer. In two cases quoted by Trousseau, from Frank, the ages at death were seventy-two and seventy-eight respectively. The prospect of life, no doubt, depends, to a considerable extent on the bulk to which the skull and its contents have attained, and on whether the disease has become stationary or not. It not unfrequently, indeed, comes to a stand-still at a comparatively early stage, and the patient survives with a large head, a protruding forehead, and other more or less obvious indications of the affection which he laboured under in infancy. But the prospects of life do not depend wholly on these conditions, for the man Cardinal, who lived to the age of thirty, had an enormously large head, and the ossification of his skull was not completed until two years before his death.

2. The symptoms due to dropsy of the ventricles, coming on after the consolidation of the skull, are necessarily obscure, and none the less so that the dropsy is almost without exception dependent on the presence of some other grave lesion which has already produced cerebral symptoms. The special symptoms to be expected are those which would arise from pressure on the important ganglia situated on the floor of the ventricles; or, if the accumulation be acute and abundant, and in these respects resembling intra-ventricular hemorrhage, those of almost sudden and profound coma, with general paresis. There is probably always more or less impairment of the mental functions, loss of memory, dulness and stupidity, attacks of unconsciousness or convulsions, more or less want of control over the evacuations, and finally coma. But, besides these phenomena, there may be more or less marked hemiplegia, and not improbably some interference with the conductiveness of some of the cranial nerves, or some impairment of speech.

3. The symptoms referrible to internal hydrorrhachis are also exceedingly vague. In some cases there is nothing either in the history or in the symptoms to indicate the presence of any affection whatever of the cord. In a case of Sir W. Gull's, and in some others that have been recorded, dilatation of the canal in the neck induced paresis of the upper extremities, with wasting of the muscles. It is natural, indeed, to assume that the symptoms of this affection should be those of pressure on the grey matter of the cord; and the symptoms which have been presented by published cases accord in the main with this assumption.

The clinical history of hydro-meningocele, hydrencephalocele, and spina bifida, and the treatment of these affections, belong rather to surgery than to medicine, and need not further occupy our attention.

Treatment.—The treatment of dropsy of the brain or cord is exceedingly unsatisfactory. If indeed the dropsy be in the adult, and secondary to some organic lesion, the probability is that it will not be diagnosed. If it were diagnosed it would not lead us to adopt any specific treatment. In the chronic hydrocephalus of children, however, we so easily recognise the presence of the disease, there is such a field for treatment offered by the slowness of the case and the gradual evolution of its various symptoms, that it is difficult to believe that everything we do must be unavailing. Yet this is certainly true of the great majority of cases. The attempt has often been made to promote the absorption of the fluid by the application of counter-irritants to the surface of the skull, or by the compression of the skull by bandages, or better, by the use of long strips of adhesive plaister applied uniformly over its surface. Trousseau states that in a case in which he adopted this treatment, sudden death was caused by the yielding of the bones of the base of the skull and the discharge of the dropsical fluid by the nose. It has been recommended to tap the distended cavi-

ties by means of a fine trocar and cannula. In using these, the puncture should be made vertically, at the edge of the anterior fontanelle, but avoiding the situation of the longitudinal sinus. A small quantity of fluid only should be removed at one time, and external pressure should be used to counteract the diminished pressure within. The operation is not dangerous, and has often been performed with temporary benefit; though no doubt there is risk that inflammation may follow, or that a vessel may be wounded. For internal use, iodide of potassium, iodide of iron, and mercurials have been employed. Sir Thomas Watson suggests, on the recommendation of an old apothecary of his acquaintance, the exhibition thrice daily of about ten grains of a pill made by mixing two parts of crude mercury with one part of fresh squills and four parts of conserve of roses. It is stated that persistence in this for several weeks has cured more than one case of the disease. It is nevertheless questionable whether any of the above plans of treatment are of real efficacy; and whether any children, who would not otherwise have got well, have recovered under their influence. On the whole it seems to us that it is best to aim at promoting the child's general health by attention to his diet and to his secretions, and by the use of iron, cod-liver oil, or other tonic medicines calculated to fortify his vital powers.

XII. CHOREA. (*St. Vitus's Dance.*)

Definition.—Chorea is a peculiar convulsive disorder, for the most part of early life, characterised by disorderly movements, which in the first instance are usually unilateral, but soon become general, and which tend as a rule to subside spontaneously after a few weeks' duration.

Causation.—This affection occurs mainly among children between the ages of five and fifteen, or from the commencement of the second dentition to the end of puberty. It is not, however, very uncommon to meet with it in persons between fifteen and twenty-five; and indeed it may occur, but occurs with extreme rarity, at any subsequent period of life. Dr. Graves records the case of a chemist who had chorea at the age of seventy, and M. Henri Roger that of a lady who was seized with it at the age of eighty-three. Chorea attacks females far more frequently than males. This preponderance in favour of the female sex is manifested even in early childhood, but it becomes more pronounced as life advances; and of adults who are attacked very few are men. Other predisposing causes are: hereditary influence, childbirth, and especially a previous attack of the disease. Trousseau draws attention to its frequent association with chlorosis; but perhaps the most interesting fact in relation to the causation of chorea is the intimate connection which it has with articular rheumatism and cardiac

disease. Not only does chorea often come on in the course of acute rheumatism, not only does acute rheumatism occasionally come on in the course of chorea, but a large proportion of those victims of chorea whose cases do not fall into either of these categories have suffered from acute rheumatism at some period or other prior to the choreic attack. It has further been clearly ascertained: that by far the greater number of choreic patients present some cardiac defect; that either the action of the heart is irregular, or there is what is supposed to be an anæmic murmur at the base, or there is distinct evidence of endocarditis, pericarditis, or both; and that this cardiac defect (even if clearly of inflammatory origin) is often met with in cases in which there is no history of rheumatism, or comes on during the choreic attack without any associated implication of the joints. Rheumatism, therefore, and especially rheumatism attended with pericarditis or endocarditis, must be regarded as at least one of the most efficient of the determining causes of chorea. Other causes, which operate apparently independently of heart-disease or rheumatism, are overwork, anxiety, excitement, and, above all, sudden fright.

Symptoms and progress.—Chorea generally comes on insidiously; and not unfrequently before any convulsive movements are recognised the child is observed to mope, to avoid its companions, to take no interest in its accustomed amusements or games, and to be incapable of fixing its attention on its work, of committing lessons to memory, or even of readily recollecting. Indeed there is generally some real or apparent mental deficiency, associated with more or less emotional disturbance, indicated by a tendency to caprice and fretfulness, to cry, and to be suspicious or timid. These phenomena may go along with more or less general loss of health and impairment of the nutritive functions. The first indications of the special nature of the disease under which the patient is labouring are usually more or less restlessness or fidgetiness, and a certain clumsiness in his movements: he cannot sit long in one place, he is constantly shifting his position or the position of one or other of his limbs; he stumbles unaccountably in moving about the room or in going up and down stairs; and he has a tendency to spill his tea or coffee, or to drop, or to knock against something else, whatever he essays to carry. The choreic movements are mostly first manifested upon one side, sometimes in the face, sometimes in the hand and arm, less commonly in the leg; but soon they involve the whole side in a greater or less degree; and after a variable time, a few days or a few weeks, the affection probably extends to the opposite side of the body, and thus becomes universal, although there often still remains more or less distinct preponderance of the symptoms on one side. But this mode of access, though the most frequent, is by no means invariable. In some cases, when the affection comes on in the course of an attack of rheumatism, no obvious prodromal symptoms are presented. And sometimes, especially when the disease is induced

by violent emotion, its onset is sudden, and the symptoms may be general from the beginning.

The phenomena of the fully-developed affection, although varying largely in degree, differ but little in kind, and are for the most part exceedingly characteristic. The convulsions affect, in a greater or less degree, the whole body. They are remarkable for their disorderly character; they are not rhythmical, neither are they simple alternate flexions and extensions; but they consist in sudden impulsive movements, succeeding one another at irregular intervals, and involving now one group of muscles, now another, now one part of the body, now another, now several concurrently. The convulsions generally subside in some degree when the patient is sitting or lying down; and (if they are not very violent) he is sometimes able to restrain them for a few moments; but they become aggravated whenever he endeavours to execute voluntary movements, whenever anything occurs to excite him, whenever he feels that he is being observed. It hence happens that the medical attendant rarely sees him at his best. The choreic phenomena cease during sleep and under the influence of chloroform. The affection of the muscles of the face induces constant contortions of the features; the eyebrows are at one time elevated and the forehead is thrown into transverse wrinkles; at another time the brows are knit; the eyes move suddenly and without purpose in various directions; the mouth is now opened, now closed, now drawn into various odd forms by the influence of the orbicularis and surrounding muscles. The face, moreover, wears a strangely vacant imbecile aspect. The tongue shares in these tumultuous movements. If the patient be asked to put it out, he opens his mouth wide and protrudes it with a jerk, and then as suddenly withdraws it, the mouth and jaws closing upon it with sudden violence. If he endeavour to answer questions, the convulsive movements of the face and mouth become aggravated; and he has extreme difficulty in articulating his words, which come out in driblets as it were, slurred over, or uttered with a peculiar drawl, hesitation, or stammer. The difficulty of speech depends partly on the convulsive action of the lips and tongue; but not unfrequently also on spasmodic affection of the larynx and respiratory muscles, which compels him to draw his breath suddenly through the laryngeal orifice with a strange sound. In some cases, even when no attempt at speech is being made, odd croaking or grunting noises are thus from time to time produced. The actions of the muscles of the head and neck are probably as incoherent as those of the face, so that the head is sometimes jerked to one side, sometimes to the other, or thrown disorderly into various odd positions. No parts usually manifest choreic phenomena more strikingly than the upper extremity: all its segments are involved in a greater or less degree; the patient hitches his shoulder; he moves his upper arm to and from his side; his forearm becomes flexed, extended, supinated, pronated; his hands

and fingers execute the most grotesque and inco-ordinate movements. The general movements of the limb, when the patient uses it—when, for example, he endeavours to raise a glass of water to his lips—are curious to watch. By an effort of the will (if the case be not exceedingly severe) the glass ultimately reaches its destination, but it reaches it probably after many failures; its progress is not arrested by a series of undulatory, tremulous, or backward and forward movements of the limb, but the different segments are suddenly and violently plucked, as it were, by some invisible power first in one direction then in another, in the line of the intended movement, or in direct opposition to it, or at right angles with it. The primary movement is overlaid, as it were, during its course with innumerable uncontrollable secondary movements, which retard it, aggravate it, and distract it. The lower extremities are affected similarly to the arms. They are moderately quiet when the patient is at rest, but as soon as he begins to use them, as soon as he begins to walk, their movements become inco-ordinate, jerky, tumultuous. To quote Sir Thomas Watson's words: 'when the patient intends to stand or sit still, her feet scrape and shuffle on the floor, or one of them is suddenly everted and then twisted inwards, or perhaps is thrown across the other; and if she endeavour to walk, her progress is indirect and uncertain; she halts and drags her leg rather than lifts it up, and advances with a rushing or jumping motion by fits and starts.' The muscles of the trunk partake in the general convulsive movements, and the body is twitched and contorted with sudden violence into all kinds of odd and unaccountable positions. It must be added: that mastication and deglutition are often rendered difficult by the spasmodic movements of the muscles engaged in these operations; that respiration is frequently interrupted and rendered irregular, jerky, and noisy, by involvement of the diaphragm; and that sometimes in severe cases the sphincters of the rectum and bladder relax, and the evacuations escape involuntarily. In mild cases the patient is able to walk about, though with more or less difficulty or clumsiness, and it may be to feed and dress himself. In more severe cases locomotion is impossible, and he has to be confined to his bed; he becomes, moreover, quite incapable of using his hands for any purpose. In the worst form of the disease the condition of the patient is miserable in the last degree, and pitiable to behold. His features and head and neck are in constant motion; his arms are flung out first in this direction and then in that, his fingers and hands meanwhile executing the most varied and fantastic movements; his lower extremities are probably little less violently convulsed than his arms; and his trunk is constantly being twisted about in bed, now into the prone position, now into the supine, is now doubled up, now straightened out again, now caught by some strange contortion.

The phenomena above described are not, however, the only nervous

phenomena which attend chorea. There is always impairment of the strength of the affected muscles, some paresis—a fact especially easy of recognition in cases of unilateral chorea. In some cases indeed, the convulsive phenomena may be replaced by more or less complete hemiplegia or even paraplegia. Sometimes the hemiplegic or paraplegic symptoms precede the onset of the choreic movements. More frequently they come on in the course of the disease and supplant them. Some impairment of sensation is also observable in the great majority of cases; and its degree has more or less relation to the severity of the convulsions or to the degree of paralysis present. Occasionally the anæsthesia is almost absolute. The fatuous aspect of the patient in the early stage of chorea has already been referred to; this aspect continues and even becomes aggravated during the continuance of the disease. No doubt it depends largely upon the various spasmodic movements in which the muscles of expression and those that move the eyeballs are implicated; but there is good reason to believe it is to some extent governed by the fact that intelligence does actually fail to a greater or less extent during the presence of the malady. Emotional sensibility, on the other hand, is somewhat exalted. Sometimes the eyesight fails.

Subordinate symptoms of more or less importance are apt to attend the progress of chorea. The patient's appetite is often bad, or capricious, or fails. His bowels are confined. His nutrition becomes impaired. He suffers from palpitation, and, as has already been pointed out, he is liable to functional or organic disease of the heart, either of which may supervene in the course of his attack. There is a striking absence of febrile symptoms during the progress of the disease.

The issue of chorea is in the vast majority of cases favourable. Sometimes (if for example the choreic movements come on in the course of acute rheumatism and involve one arm only) the patient recovers in the course of a few days. More commonly the disease continues for a period varying between four or five weeks and three months. In some instances it is prolonged for two or three years or more. But in these cases it is usually continued by successive relapses, each coming on before the symptoms of the preceding attack have wholly disappeared. Indeed, patients who have had one attack of chorea are peculiarly liable to subsequent attacks, which come on at irregular periods and under the slightest provocation. Very rarely, indeed, chorea lasts for many years or for a life-time. When the disease is fatal, it rapidly assumes aggravated proportions. The spasms are incessant; their violence and continuance prevent sleep, or allow only of occasional short snatches of sleep; and they interfere seriously with the ingestion of food, and thus rapidly induce mental and bodily exhaustion. Further, the evacuations escape unconsciously, or at all events are uncontrolled; and partly on this account, partly in consequence of the constant friction to which the trunk and limbs are subjected by their never-ceasing

movements, the skin becomes chafed in innumerable places, and bed-sores form over the various prominences, more especially over the elbows, hips, and sacrum. Often also the child bites its lips until they bleed; and very frequently the red portions of both lips become split by numerous deep vertical fissures. Death, which may be preceded by delirium, is generally due to asthenia. But its immediate cause may be the supervention of erysipelas or the consequences of heart-disease.

The recovery from chorea (putting cardiac disease out of the question) is generally complete: the patient regains his muscular strength, and his intelligence is restored to him unimpaired. But it is not always so. Occasionally he remains more or less feeble-minded, or even becomes insane, or lapses into a fatuous condition. In some cases, too, the implicated muscles remain enfeebled; and they may then undergo slow contraction or atrophy, or both. In a chronic case which has been under our observation, and in which the general symptoms were undistinguishable from those of genuine chorea, the choreic movements of the lower extremities were associated with marked rigidity of the muscles, some degree of flexion at the hip and knee joints, with overlapping of the knees from the preponderant action of the adductors of the thighs, and a tendency to talipes equino-varus—facts which seem to indicate that degenerative changes of the lateral columns of the cord had supervened.

Morbid anatomy and pathology.—The pathology of chorea is confessedly obscure; it is not known either what parts of the central nervous organs are the seat of disease, or what is the nature of the morbid process going on in the affected parts. The facts of its unilateral commencement and general unilateral tendency point, however, to disease, either of the corpus striatum and optic thalamus, or of the corresponding cerebral hemisphere. And doubtless one or other of these parts is the main seat of the lesions on which chorea depends. But there are many features of the disease, such as the occasional implication of the muscles of phonation, respiration, and deglutition, and the frequent occurrence of functional disturbances of the heart, which would seem to imply involvement of the medulla oblongata. And the resemblance of the choreic movements to those of locomotor ataxy are certainly suggestive of implication of the cord. Then as regards the nature of the disease, its frequent connection with rheumatism and cardiac disease has suggested at least two hypotheses. One, originating with Dr. Kirkes, and since ably supported by Dr. Hughlings Jackson, is to the effect that the symptoms are due to obstruction by minute emboli of the smaller branches of the arteries supplying the corpus striatum and contiguous parts, with consequent scattered minute patches of congestion and softening. The objections, however, to this view are obvious. Obstruction of the arterioles has been observed only in a very small number of cases, and it is doubtful if in these

the obstructions were embolic or thrombotic. Besides which, it is not only difficult to believe that showers of minute emboli should be distributed throughout the minute vessels supplied to one corpus striatum or one side of the brain only, and that at some later period there should be a similar limitation of such embolic patches to the region supplied by the middle cerebral artery of the other side: but it is difficult to understand why large emboli should not be occasionally intermingled with the smaller ones, and cause sudden hemiplegia by obstructing a large vessel, and why small emboli shed simultaneously should not become blended by fibrinous coagulation around them into one or two concrete masses. The other hypothesis is that the same disease which affects the valves of the heart or the joints in rheumatism attacks also the smaller cerebral vessels or the ultimate tissue of the central nervous organs, a view which might well explain the supervention of cardiac disease in chorea, as well as the dependence of chorea on rheumatic fever. A main objection to this view is the fact that it is simply conjectural, and wholly unsupported by anatomical evidence. Moreover, it fails, as also does the embolic hypothesis, to explain those cases which are due to fright or other powerful emotions, and in which, so far as we know, the heart remains sound.

It seems to us, however, that the clinical phenomena of chorea cannot possibly be referred to affection of any circumscribed region of the nervous centres; and that, whether the seat of disease be thus limited or not, the embolic hypothesis is altogether inadequate as an explanation of the nature of the morbid processes to which the clinical phenomena are linked. The symptoms are partly intellectual, partly emotional, and referrible partly to the functions of the voluntary muscles, partly to the cutaneous sensibility, and partly also to the bulbar nerves, which subserve articulation, deglutition, respiration and the motor functions of the heart; they would seem therefore to be connected at the same time or successively, and in different degrees, with the cerebral convolutions, the ganglia at the base, the pons and medulla, and the spinal cord. The valuable paper recently read by Dr. Dickinson before the Medico-Chirurgical Society is strongly confirmatory of this view. He shows, from the results of careful post mortem examinations made on several fatal cases of chorea: that there is a general tendency to dilatation of the smaller vessels, more especially the arteries, throughout the substance of the brain and cord; that this dilatation is attended with exudation into the tissues immediately surrounding the vessels, and occasionally with small hemorrhages indicated by the presence of blood-crystals and the like, or patches of sclerosis; that these changes are most advanced in the corpora striata, in the nervous matter in the neighbourhood of the trunks of the middle cerebral arteries, and in the posterior and lateral portions of the grey matter of the cord, mainly at the upper part; and further, that in all these regions the morbid conditions tend to be symmetrically arranged.

And on the basis of these facts, and admitting that chorea is associated generally with rheumatism, in the larger proportion of cases with heart-disease, and in some cases with no inflammatory or structural disease of any organ, he comes to the conclusion (in which we are disposed to concur) that chorea depends 'on a widely-spread hyperæmia of the nervous centres, not due to any mechanical mischance, but produced by causes mainly of two kinds—one being the rheumatic condition, the other comprising various forms of irritation, mental and reflex, belonging especially to the nervous system.' The tendency which the vascular changes have (on Dr. Dickinson's showing) to induce sclerosis in the tissues which surround the vessels well explains the wasting of muscles, rigidity of limbs, and permanent paralysis, which occasionally complicate chorea or supervene upon it.

Treatment.—For few diseases have so many specific remedies been vaunted as for chorea; yet few diseases are really so little amenable to treatment. It must never be forgotten, in weighing the value of medicines in this affection, that the great majority of cases tend to get well spontaneously in the course of a few weeks. Sydenham recommended bleeding and tartrate of antimony, and cured his patients by these means; and even Sir Thomas Watson advocates local bleeding when there is a fixed pain in the head. Large doses of antimony, indeed, have been strongly recommended by many physicians. Iron is a favourite remedy; so is arsenic; and so also is sulphate of zinc, given in doses, to commence with, of a grain or two three times a day, which are slowly increased by successive increments, until from 20 to 40 grains are given at a time. Iodide of potassium is lauded by some; bromide of potassium by others; phosphorus by others. Of medicines derived from vegetable sources we may name turpentine, strychnia, cannabis Indica, opium, belladonna, and various anti-spasmodics. Exercise, frictions, and cold baths, more especially shower-baths, have all their advocates. We must confess that in our own opinion few, if any, of the above remedies have any real influence over the course of the disease; if, however, we have any bias, it is in favour of arsenic, given in small doses, and continued for some length of time. We believe, however, that real benefit accrues in a considerable number of cases from improvement of the general health; that in this point of view, tonics (among which iron holds an important place) are useful, as also are careful attention to hygienic measures, good wholesome diet, early hours, avoidance of excitement, gentle exercise, cold or tepid bathing, and change of scene and air. Again, our treatment may often be usefully directed by the nature of the malady (if any) with which the chorea is associated; thus when rheumatism is present, or chorea is a legacy left by rheumatism, anti-rheumatic treatment may be of great service. In those severe cases in which the convulsive movements are incessant, and the patient has little or no rest, and death consequently threatens, narcotics and stimulants would seem to

be indicated. The inhalation of chloroform arrests the convulsive movements so long as the patient is under its influence; opium, morphia or chloral in large doses has the same effect. But it must be admitted that notwithstanding the temporary ease they give, the progress of the disease towards its fatal end is rarely, if ever, retarded by their use. The patient should then be supported by food and stimulants. Further, every precaution should be taken to prevent the patient from injuring himself in his contortions, and all sores that form upon the surface of the skin should be at once treated, and protected from further injury.

It may be pointed out, in conclusion, that chorea is apt to spread among children, apparently by imitation; that choreic patients are often rendered worse by their association with patients of the same class; and that hence precautionary measures directed against such accidents should be taken.

XIII. EPILEPSY. ECLAMPSIA. INFANTILE CONVULSIONS.

A.—*Epilepsy.* (*Morbus comitialis vel sacer.*)

Definition.—It is difficult, if not impossible, so to define epilepsy as within the limits of a mere definition to include all the varieties of form which it assumes. Speaking generally, it is a functional disorder of the nervous centres, characterised by sudden seizures of temporary duration and occurring at irregular intervals, in which the patient either loses consciousness or presents some other form of mental disturbance, or has tonic or clonic convulsions, or all of these phenomena in sequence. For a true conception of the disease, it must be understood that, however mild the attacks may be, all the phenomena which have been enumerated are potentially present in them, and may be expected to occur in combination during the progress of the disease.

Causation.—The causes of epilepsy are very obscure. The disease has been attributed to all sorts of circumstances which have probably little or no influence in its production. Among those to which most importance has been attached may be mentioned sudden fright, the witnessing of an attack of epilepsy, long-continued anxiety, overwork, drink, abuse of absinthe, and venereal excesses, especially masturbation. But their importance as exciting causes has been greatly overestimated. It is considered by Trousseau that the real share of each one of them (excepting fright) in the production of the disease is yet to be proved; and Russell Reynolds remarks of excessive venery and masturbation, that far too much importance has been attached to them. Hereditary predisposition, on the other hand, exerts a remarkable in-

fluence over the development of epilepsy. It is to be observed, however, that it is not so much epilepsy itself which is hereditary (although no doubt it is so in a high degree), as that epilepsy becomes hereditary in families, among the members of which neuroses, such as epilepsy, insanity, hysteria and the like, prevail. It is not uncommon to find in such families that several of the children are epileptic, or that one is epileptic, one suffers from chorea, one is an idiot, and so on. But the predisposition to epilepsy may be acquired: for it is certain that many of those persons who subsequently become epileptic have suffered in infancy from convulsions, which were induced by teething or other accidental circumstances. Epilepsy occurs pretty equally in both sexes. The first attack may come on at any period of life—in early childhood or extreme old age; but it occurs far more frequently between the ages of ten and twenty (more precisely, perhaps, during the time of puberty) than it does at any other period of life. Dr. Reynolds points out that there is comparative immunity between the ages of twenty-five and thirty-five; but that the outbreak of the disease becomes comparatively frequent again about the age of forty. After this time its primary appearance is extremely rare.

Symptoms and progress.—The phenomena of epileptic attacks are so various, they differ so widely from one another in different cases, both in their characters and in their grouping, that it is impossible to give a comprehensive, and at the same time graphic, account of them, excepting by the aid of illustrative cases. Our space forbids the adoption of this course. We shall, therefore, begin with the description of a typical attack of the disease, and then discuss the variations to which attacks are liable.

The epileptic fit is not unfrequently preceded by a well-marked prodromal period, lasting in different cases from a day or two to a few seconds. But under any circumstances, the fit itself comes on suddenly; the patient probably utters a cry, loses consciousness, and, if standing, falls down as if shot on his face or on the back of his head; his muscles become rigid, especially perhaps, those of one side, and at the same time slowly contract; and respiration ceases. After these phenomena, which constitute the first stage of the attack, have lasted for a few seconds, the second stage supervenes. This is characterised mainly by return of respiration, lividity of surface, distension of the veins of the head and neck, clonic spasms, which are mostly unilateral, biting of the tongue, and continuance of unconsciousness. At the end of a minute or two this stage also comes to a conclusion, the lividity disappears and the convulsions cease, but the patient probably still continues insensible; presently, however, consciousness returns in some degree, and he either rapidly recovers, or remains confused or maniacal, or in a state of stupor, for some hours, or it may be a day or two, before complete recovery takes place.

The prodromal period is present probably in about half the total num-

ber of cases, and if present in one attack is most likely present in other attacks occurring in the same patient. Moreover, under such circumstances the premonitory symptoms continue probably, at any rate for a time, of the same kind. Those which precede the attack by some hours or a day or two are the least common, and although perhaps apparent enough to the patient or his friends, are on the whole slight in degree and vague. They consist, for the most part, in some modification of the patient's intelligence, feelings, or habits; he gets dull and incapable of mental exertion or of attention to business, sullen or low-spirited; or his manner and conversation become sparkling and lively, and his spirits unaccountably buoyant and jovial—he may even be furiously maniacal; or there is simply something in his look—a wildness in his eye, or a dulness and heaviness of expression—which is not natural to him. The more characteristic premonitory symptoms are those which precede the fit by a few minutes or a few seconds only. They are remarkably various. In some cases they consist in the spasmodic contraction of certain muscles: the expressional muscles of one side of the face twitch; or the hand and arm are convulsed, and gradually carried upwards towards the face; or the lower extremity is equivalently affected; or the muscles of one side of the head and neck contract and carry the face over the opposite shoulder; or the muscles of several or of all these regions are simultaneously involved. Sometimes the epileptic fit is preceded by vertigo, or sickness, or by severe pain or some undefinable sensation referred to the head, throat, chest, abdomen, or some other part. Not unfrequently the premonition is furnished by what has been termed the epileptic aura—a sense of coldness, heat, or pain, starting from some point, say the finger or toe, the abdomen or chest, or it may be from the seat of some former injury—which seems gradually to ascend until it reaches, as the case may be, the epigastrium, the præcordial region, or the head, when insensibility suddenly supervenes. In some cases the attack is ushered in by some hallucination of the senses: the patient perceives some peculiar smell; or he hears strange sounds, and, it may be, voices; or he sees definite forms before his eyes—animals, departed friends, witches, devils. Not unfrequently, again, the premonitory symptoms consist in some odd mental disturbance: the patient experiences a sudden horror or trouble, or finds himself engaged in some special train of thought or perplexed by some problem or the plot of some story or strange combination of circumstances. It is curious that these mental perplexities which seize upon the patient are often entirely forgotten after the occurrence of the fit, yet that the same perplexity is repeated (exactly as the drawing up of the arm or the occurrence of an aura is repeated in other cases) before every epileptic attack.

The most constant feature of the first stage of the fit is the sudden onset of absolute unconsciousness. This may be momentary only, or may be prolonged throughout the whole of the first and second stages,

and for two or three minutes or more. As we shall afterwards show, it is sometimes absent. This unconsciousness while it lasts is profound; the patient neither sees, nor hears, nor feels, nor can be roused by any means at our command. The convulsions which attend this stage are tonic; they consist in the supervention in the affected muscles of great rigidity, attended in the first instance with fibrillar movements, and a tendency for certain muscles gradually to overcome their antagonists. They are rarely general, or if general they affect one side more powerfully than the other; they are in fact almost always unilateral, and sometimes limited to the side of the face or head and neck, or to the arm. The face becomes hideously distorted, and the tongue probably thrust between the teeth; the head, from contraction of the sterno-mastoid and other muscles drawn down obliquely on one side, the face being thrust over the opposite shoulder; the trunk contorted; and the arm or leg flexed or extended. At the same time the respiratory and laryngeal muscles become fixed, and the acts of respiration cease entirely. These spasms are sometimes wholly absent. The epileptic cry which ushers in the attack occurs only in a limited number of cases. It varies in character, is sometimes a loud shriek, sometimes a hoarse groan, and is usually very distressing to hear. It occurs (as Dr. Reynolds points out) once only, and appears to depend on the sudden emission of breath through the constricted glottis caused by the spasmodic contraction of the expiratory muscles. The pupils are dilated and insensible to light. If the patient be closely observed at the onset of his attack, his face will usually be seen to be suddenly overspread with a death-like pallor, which persists for a few seconds, but gradually, during the progress of the first stage, becomes replaced by redness and turgidity. In some instances no change whatever of colour can be discovered. In association with the phenomena here enumerated there is usually extreme feebleness of pulse. Although the patient generally falls with sudden violence, he sometimes slips down quietly, almost as if by design; and if the loss of consciousness be momentary only he sometimes remains motionless, standing or sitting, or merely staggers. The first stage usually lasts from ten to thirty or forty seconds.

The second stage is attended with continuance of unconsciousness. The face has usually by the time of its commencement become livid and bloated, and the veins of the head and neck distended; but this lividity and over-distension of the vessels slowly subside during its continuance. The tonic spasms cease, to be replaced by clonic spasms. These, which consist in alternate powerful contractions of flexors and extensors or other groups of antagonistic muscles, may be general; but they are more commonly one-sided and limited to those parts which had previously been the seat of tonic contraction. The pupils oscillate, the eyelids and muscles of expression-work, the mouth is alternately opened and closed with violence, and the protruded tongue, caught

between the teeth, is apt to get severely bitten, the muscles of the head and neck and those of the trunk are convulsed, and the arm and leg execute powerful movements of extension and flexion. At the same time probably the fæces and urine are discharged involuntarily. The respiratory acts are resumed at the commencement of this stage, and during its course are violent, jerky, noisy, and laboured. The skin is cold; profuse sweats break out; the pulse is full; the heart beats violently. Mucus accumulates in the mouth and fauces, and mingled with the blood yielded by the bitten tongue, escapes from the lips. The symptoms of this stage, violent in the beginning, gradually subside; and at the end of a few seconds, a minute, or at most two or three minutes, the patient draws a deep sigh, and the second stage is completed.

The condition of the patient in the third stage varies. Sometimes he recovers almost instantaneously, and appears at once in his normal health; but more commonly he lies for some minutes or for half an hour in a condition of profound coma, from which it is impossible to arouse him. Sooner or later, however, consciousness slowly returns; he opens his eyes and gazes stupidly or wildly about him; he tries to speak, but mumbles unintelligibly or incoherently, or fails to produce any articulate sound; he tries perhaps to get up, and his movements and demeanour resemble those of a drunken man; sometimes he becomes wildly maniacal, sometimes falls into a state of trance or ecstasy. It not unfrequently happens that the patient lapses into a more or less profound sleep, interrupted it may be from time to time by slight convulsive twitchings. Muscular weariness, a sense of general bruising, headache, vertigo, restlessness, severe mental or emotional disturbance are apt to remain for some hours or even for a few days after the fit. After the attack the patient often passes a large quantity of limpid urine; and owing to the extreme distension of the vessels of the head and neck and upper part of the trunk, minute extravasations of blood are apt to occur during the fit, and the surface of these parts to become more or less thickly studded with persistent hemorrhagic points, or petechiæ.

The above account applies to those fits, typical in their severity and in the sequence of their phenomena, to which the names *epilepsia gravior* and *haut mal* have been given. But in a large number of cases either the fit does not pass beyond the prodromal stage; or various of the stages are absent, or so rapidly completed, or so blended with one another, that they escape observation; or some of the features of the malady are aggravated; or new features are superadded. Most of these attacks come under the denomination of *epilepsia mitior*, *petit mal*, or *epileptic vertigo*.

In some cases the patient is affected with an occasional sudden spasm of one side of the face, or of one sterno-mastoid; or his hand closes, and the arm is gradually drawn up or flexed; or he experiences some one of

those sensory hallucinations which have been previously enumerated; or he has an aura, or a sudden attack of headache, giddiness, sickness, or faintness. In other words, he is attacked with some one of the prodromal symptoms which are known to usher in epilepsy. Now it does not at all necessarily follow that the sudden occurrence of such phenomena, or even their occasional repetition, proves that the patient is epileptic; but it is certain that of those persons who suffer from them, some become epileptic sooner or later; and that those epileptics whose fits are preceded by warning symptoms, not unfrequently have such warnings without fits following them. There can be no doubt that such attacks must, under these circumstances, be regarded as epileptic. They are, in fact, abortive epileptic fits. In some instances the patient's seizures consist in little more than a momentary interruption to the continuity of his thoughts. He is engaged in talking, and suddenly for a second or two becomes quiet, and then resumes the thread of his conversation as if nothing had occurred; or instead of ceasing to speak he may utter some incoherent sounds, or words and expressions utterly alien to the subject in which he was engaged. If he be closely observed at this moment, his pupils will probably be seen to dilate, his face to become momentarily pale, and then perhaps with returning consciousness a little more congested than natural. During the momentary attack the patient may become absolutely unconscious, and his mind may be a blank; or, although unconscious to everything about him, he may be the subject of a sudden trouble, perplexity of mind, or horror—some momentary nightmare, as it were. Sometimes he utters a shriek and reels or staggers, or performs a rotatory movement, and then without falling to the ground recovers. Sometimes he is seized with unconsciousness lasting for a few seconds, or for a minute or more, and remains sitting or standing, or in whatever other position the fit surprised him in, his features meanwhile being perfectly passive or presenting convulsive twitchings. In some cases the wholly unconscious patient will go on during his unconsciousness with the work in which he was engaged at the time of seizure; if walking he will continue to walk, if running he will go on running. Trousseau mentions the case of a young amateur violinist who, during attacks of short duration, would go on playing with perfect accuracy as if he were still in his ordinary senses. There are other cases, again, in which the patient, during his attack of unconsciousness, performs strange actions, which have nevertheless an aspect of purposiveness about them, but of which he has no recollection whatever when consciousness returns. They seem in fact like the translation into action of the fragment of a dream. Thus, sometimes, while walking, perhaps in the street, he all at once begins to run rapidly, avoiding all obstacles, and then coming to himself discovers himself unaccountably far from his destination or from the place at which he lost his senses. Trousseau cites the case of a magistrate who, in such an attack, suddenly left the court

over which he was presiding, went into the council chamber, made water in a corner of the room, and returned to the court entirely ignorant of the strange act that he had committed. Sometimes the patient will dance or sing, or peer about in various directions as if in search of something which he had lost or mislaid. But perhaps the most important, if not the most remarkable, of these aberrant forms of epilepsy are those in which the patient is seized with sudden and unaccountable fury, tears his clothes, or destroys anything that is near him, belabours the friend or the servant that is with him, or rushes out of the house and attacks the first stranger that he meets, or jumps from the window, or in some other way maims or kills himself, and moreover not unfrequently accomplishes such acts with apparent definiteness of purpose. Thus a husband, apparently waking out of sleep, will beat or strangle his wife with the utmost ferocity; a man walking along the street will, at the moment when the impulse is upon him, make an unprovoked and violent onslaught on whoever chances to come near. Now it must be borne in mind that in all the varieties of seizure, in all the different forms of epileptic vertigo, which we have been considering, the only appreciable part of the attack may be the temporary unconsciousness, or delirium, together with the various specific motor phenomena which have been enumerated, and utter unconsciousness or forgetfulness of what has passed in the attack. There may be no premonitory symptoms, no tonic or clonic convulsions, no change of colour, no succeeding bodily or mental suffering. On the other hand, careful observation will often reveal the presence in a more or less modified form of some of the more ordinary features of the typical epileptic fit. There may be slight premonitory symptoms; there is generally a sudden pallor or ghastliness at the commencement of the attack, soon followed by more or less redness and lividity, and in connection therewith dilatation of pupils, rolling of the eyes, or twitching or more violent convulsive movements of the muscles of the face or one of the limbs; and, further, vertigo, confusion of mind, or other such conditions may remain for a longer or shorter time after the subsidence of the fit.

The first epileptic fit may also be the last; but in the great majority of cases it forms the prelude to subsequent attacks, which may come on at various intervals for months or years or during the whole subsequent lifetime of the patient. Sometimes the fits recur with more or less irregularity at intervals of a week, a month, two or three months, or a year, or more. There may then be a single fit at each recurring period, or there may be two or three, or a dozen, or more, succeeding one another more or less rapidly during a period of twelve or twenty-four hours. In some instances frequent fits occur habitually day and night. It occasionally happens that, as the general health improves or age advances, the fits become less and less frequent, and at length recur at irregular intervals of years, or disappear altogether. It must

be borne in mind, however, that those who have once been epileptic, even if five, ten, or a dozen years have passed since the last attack, are still not unlikely to have a relapse; and, again, that patients whose fits have hitherto occurred only at long intervals, not unfrequently suffer from aggravation of the disease—the fits rapidly increasing in frequency, and recurring in large numbers day and night for weeks together. Sometimes, when the attacks follow one another very rapidly, the patient falls into the *status epilepticus*—a condition in which he remains insensible for many hours, sometimes for a day or two, and which has often been referred to the persistence of a single fit; it is made up, however, of a succession of fits, linked together by persistent epileptic coma.

Convulsive fits recur as a rule much less frequently than attacks of epileptic vertigo; yet when they recur at long intervals they are very liable to be repeated several times within a limited period. Epileptic vertigo may come on habitually twenty, thirty, or forty times, or even as many as a hundred times in the day. According to Dr. Reynolds, cases of the haut mal are nearly twice as common as cases of the petit mal; there can be little doubt, however, that the petit mal is much more frequent than is generally supposed, and that many persons accounted healthy, and who never consult a doctor, are liable to occasional slight seizures. Attacks of the haut mal not unfrequently, however, alternate with those of epileptic vertigo; and still oftener patients who are subject to the former have abortive seizures represented by the aura only. On the other hand, although epileptic vertigo often constitutes the only form of seizure from which patients suffer, attacks of a severer kind are in such cases always liable to supervene.

The circumstances which determine the epileptic fit in those who are liable to fits are not generally discoverable. They often, at all events when they first appear, come on only in the night, either it is said, at the moment of going to sleep, or at the moment of waking; and, even when they take place both day and night, they often occur mainly at night-time. It is not uncommon for them to attack women at the monthly periods; yet it still more frequently happens that epileptic women do not suffer specially at the time of menstruation; and they escape as a rule during parturition, a time at which eclampsia has a special tendency to supervene. In some cases the fit seems to be induced by severe mental labour, by emotion, by a debauch. It has occurred during the act of coitus. Sometimes, when the attack is preceded by an aura starting from some accessible point, it may be induced by irritation of that point. Thus we knew one case in which it was invariably excited by compression of a certain tender spot in the abdominal walls; and we have met with another in which for many weeks fits were brought on day and night whenever the patient's legs were moved voluntarily or involuntarily whether he was awake or asleep.

The condition of epileptic patients in the intervals between their seizures is very various. In a large proportion of cases they appear to be in the enjoyment of perfect mental and bodily health. Not unfrequently, however, some peculiarities reveal themselves sooner or later in connection with their nervous organism. They become more or less low-spirited or taciturn, or querulous, fidgety, or excitable; or there may be a little failure of memory, or some slowness of apprehension, or difficulty of application. The most remarkable mental phenomena, however, are those which are included in the term 'epileptic mania.' The attacks of mania resemble those which have been already referred to as constituting a part of the epileptic paroxysm; but they may occur independently of the epileptic fit, and may last from a few hours or two or three days, to a week or two or more. They are remarkable, as a rule, for the suddenness of their invasion and the suddenness of their subsidence. They present two varieties which by Dr. Falret are termed respectively *petit mal* and *haut mal*. The latter is furious, attended with sudden attacks of uncontrollable violence and ideas and hallucinations of a terrifying character; the language of the patient is less incoherent than that of many other lunatics, and it is remarkable that each successive maniacal attack repeats the main features, in almost every detail, of the attacks that have gone before. In the *petit mal* the patient is morose and despondent, and mistrusts and fears those who are about him; he is impelled, as it were, by some superior power, in obedience to which he performs acts that he would not otherwise do; he leaves his home and occupation, wanders about, and is liable to sudden outbreaks of passion, in which he will attack, destroy, or kill whatever comes in his way, or commit suicide. In both forms of mania the comparative coherence of the patient might lead one to suspect that he was either malingering, or under the dominance of simple revenge or passion. Yet his memory of what has occurred in his attacks is exceedingly defective; sometimes he recollects nothing; more often he recollects fragments of what has happened, as of a dream; but he can rarely call to mind all that has taken place, and perhaps forgets the main incidents entirely. In some cases of epilepsy the patient's mind undergoes gradual deterioration, and he becomes imbecile or idiotic. It is said to be principally in the case of the petit mal that this result ensues. In reference to this point, however, it must not be forgotten that epileptic fits are apt to supervene in cases of chronic madness, idiocy, and dementia.

Epilepsy does not tend immediately to shorten the duration of life; nevertheless it materially increases the risks to life. The epileptic patient is liable to incur serious accidents: to fall into the fire and be burnt; to tumble into the water; to be drowned while bathing; or to fall from his horse, or from a scaffolding, or over a precipice; he may also be choked when eating, or asphyxiated as he lies in bed. Very rarely the fit itself proves fatal without extraneous aid; when it does,

the patient dies from asphyxia during its first stage, or from exhaustion or coma during the status epilepticus.

From the multiform characters which epilepsy presents, its diagnosis is often a matter of extreme difficulty. When occurring only at night-time, it not unfrequently happens that the patient is ignorant of the nature of his malady or even that he has anything the matter with him. Yet, even in cases of this kind, a hint, or an admission, or the statement of some special occurrence may awaken the suspicions of the medical man. Thus the patient on waking up is uneasy, with giddiness, headache, or confusion of mind, from which he slowly recovers; or he finds that his tongue is sore, and that there is blood upon his pillow; or he notices petechial spots upon his face, neck, and chest; or he finds that he has passed his evacuations into the bed, or that he has dislocated his shoulder, or otherwise injured himself. Now any of these accidents may occur independently of epilepsy; but if they recur from time to time, and especially if two or three be associated, the evidence in favour of epilepsy becomes very strong.

The actual epileptic attack may be confounded with apoplexy or with hysteria. The true apoplectic attack, in which the patient falls down suddenly comatose, is now generally allowed to be epileptic or epileptiform. The point, therefore, to determine in such cases is not whether it be apoplectic or epileptic, but what is the pathological condition on which the loss of consciousness depends. The distinctions between hysterical and epileptic fits are generally well-marked, and little doubt usually remains when the history of the case is obtained. Still the affections appear to run into one another, and the condition termed hysterical epilepsy forms the link between them. The main points to bear in mind in forming a diagnosis are (apart from the patient's history) the usually much greater violence and much longer continuance of the paroxysm of hysteria, the more general distribution of the convulsive movements, and the generally great and persistent noisiness of the patient. The hysterical patient, moreover, is seldom unconscious, can generally be roused without much difficulty, rarely bites her tongue, passes her evacuations into the bed, or injures herself; the skin, too, is hot, and the pupils act under the influence of light.

Few diseases are so frequently feigned as epilepsy. The coarser features of the haut mal are so striking that few persons can fail, with a little study, to imitate them fairly well. There are various points, however, about the real attack which the actor does not observe, or cannot copy. Thus he neither bites his tongue, nor passes his evacuations into his trousers; his pupils are probably not dilated, and certainly not insensible to light; and his skin becomes hot and perspiring with his violent muscular exertions. Further, when he falls he takes care not to hurt himself; he overacts the convulsive part of the attack, but probably fails in details; moreover, he is alive to what

is going on around him, takes furtive glances at the bystanders, and gives distinct evidence that he feels if he be hurt, or if a jugful of cold water be thrown over him. Still there may be real difficulty; and it behoves the physician not to commit the error of assuming that a real epileptic is malingering. The stage which succeeds the period of insensibility is one not likely to be copied by a cheat; yet it is a stage in which it is often not difficult to persuade oneself that the patient is shamming.

Morbid anatomy and pathology.—There are few diseases about the pathology of which we are so entirely ignorant as we are about that of epilepsy. It has been referred to anæmia of the nervous centres; it has been referred to hyperæmia; it has been assumed that the cerebral convolutions, the ganglia at the base, or the pons and medulla oblongata are mainly at fault; and the disease has been regarded as one involving the nervous centres as a whole. Morbid anatomy scarcely helps us; for in the rare cases in which death has occurred in a fit, little or nothing more than hyperæmia has been detected, with in some cases hemorrhage into the perivascular sheaths of the smaller vessels; and when chronic epileptics have been examined post mortem, either the brain has looked healthy, or it has appeared to have shrunk somewhat, or there has been some induration of the white matter, or some thickening of the walls of the minute vessels with traces of previous hemorrhage in their vicinity. These lesions, however, have been mainly recognised in the brains of those whose epilepsy was associated with chronic insanity or dementia. Experiment has clearly shown that anæmia of the brain, suddenly produced, causes epileptiform convulsions; but, on the other hand, extreme congestion of the brain as occurs during the prolonged paroxysmal cough of pertussis is also followed by insensibility, associated with convulsive twitchings. There is every reason on clinical grounds to believe that in the epileptic paroxysm the brain is successively anæmic and congested. The extreme pallor which overspreads the surface at the commencement of the attack, and which has been observed, we believe, by Dr. Jackson to pervade the retinal vessels as well, may be taken as a clear indication that the brain itself is also anæmic at that time. And the great venous and capillary congestion which almost immediately afterwards replaces that pallor, coupled with the presence of post-mortem congestion and capillary hemorrhages in the brain in fatal cases of epilepsy, show clearly that the early anæmic condition of the brain is soon succeeded by notable congestion. But, even if it be allowed that anæmia of the brain is the cause of the earliest epileptic phenomena, including the tonic spasms, it is obvious that it is not the cause of the clonic spasms which come on with congestion. Dr. Marshall Hall, who clearly recognised this sequence, referred the clonic spasms to the congestion which followed upon the cessation of the respiratory acts, and recommended the performance of tracheotomy with the object of preventing

their supervention, and so of robbing the disease of its chief horrors. But if the epileptic phenomena depend on mere congestion or anæmia, this must obviously originate in some functional disturbance at the source of the vasomotor nerves which are distributed to the cerebral vessels.

The medulla oblongata and upper part of the cord are regarded by Dr. Reynolds as the primary seat of epilepsy. And MM. Luys and Voisin, as the result of careful post-mortem investigations, conclude that the parts which mainly suffer in this affection are the medulla oblongata, corpora striata, cerebellum, and other parts at the base of the brain. On the other hand, it has been shown by Brown-Séquard that epileptic convulsions may be artificially induced in guinea-pigs as a consequence of section of one of the lateral columns of the cord anywhere between the medulla and tenth dorsal vertebra. It must be admitted, indeed, that both tonic and clonic convulsions may be of spinal origin, and that in epileptic convulsions the motor tract of the cord must necessarily be largely concerned; at the same time, from the special implication of the nerves at the base of the brain, there can be no doubt that the motor nuclei in the medulla oblongata and on the floor of the fourth ventricle and iter must be at least equally affected; further, from the general unilateral tendency of the spasms or predominant action of the muscles of one side when both sides are involved, there is great reason to suspect that, however much the various nuclei of the motor tract are involved, they are dominated by the corpus striatum. Still, when we look to the clinical facts of epilepsy, and recollect that convulsion is by no means the most frequent or the most important element of the attack, and that when it occurs it is usually preceded by some aura, sensation, spasm, or hallucination, and is attended from the beginning either with absolute loss of consciousness or with a dreamy condition in which there is often a total insensibility to external impressions, it is impossible not to acknowledge: that, however seriously the cord, medulla, and ganglia at the base of the brain may be implicated subsequently, the earliest phenomenon must be connected with some limited spot in the nervous centres, which, though different for different cases, is probably always the same for the same case; that the pain, sensation, giddiness, or hallucination, is probably of central origin; and that from this primarily affected spot a sudden influence is discharged over the sensorium and the sensori-motor regions of the cerebrum, which as regards the sensorium either annuls consciousness wholly or in part, or perverts it, and as regards the motorial system, either excites it to unwonted or perverted action, or arrests its operations. There is reason, therefore, to believe that the epileptic fit commences before the brain becomes anæmic, and room, therefore, to question whether this anæmic state of the brain is the cause or the consequence of the symptoms which accompany it. There is equal reason, we think, to doubt, whether the congestion which follows the anæmia is the cause of the clonic contractions and of the various phe-

nomena which attend their occurrence; and whether, finally, the after-symptoms are to be referred (as some suppose) to carbonic acid poisoning. The pathology of the affection is, we repeat, obscure, and we do not attempt to elucidate it.

Treatment.—During the epileptic attack there is usually little to be done beyond preventing the patient from injuring himself, and removing all sources of pressure from his neck. It is often well to prevent him from biting his tongue by inserting a pad between his teeth. Convulsions may often be allayed by the inhalation of chloroform; and it may be advisable, when the congestion of the face is extreme and long-continued, to remove blood from the distended vessels of the neck. Not unfrequently, when the attack is preceded by a warning of sufficient duration, it may by proper management be averted. Among measures which have been successfully adopted for this purpose are: the inhalation of chloroform or ammonia, the administration of a dose of sal-volatile or ether, the application of a ligature above the point from which the aura springs, or the forcible prevention of the closing of the fingers or the flexion of the arm, when such movements constitute the premonitory symptoms. Latterly Dr. Crichton Brown has, for the same purpose, had recourse, with success, to the inhalation of nitrite of amyl.

The measures which have been employed to cure epilepsy are innumerable. Many drugs have been administered with more or less success, among which may be enumerated the sulphate and oxide of zinc, arsenic, copper, iron, nitrate and oxide of silver, and the bromides of potassium and ammonium; belladonna, digitalis, strychnia, opium, and Indian hemp; as also musk, valerian, and assafœtida. The list might easily be extended. Of the above, those which have perhaps enjoyed the widest reputation are the salts of zinc, silver and arsenic, belladonna, and the bromides of potassium and ammonium. Belladonna has been strongly advocated by Trousseau, who recommends that it be given in the form of a pill containing $\frac{1}{3}$ grain each of the extract and powdered leaves; or that in its place the $\frac{1}{100}$th of a grain of the sulphate of atropia be administered. He recommends that during the first month one of the pills be given daily, and that a pill per month be added, until the daily allowance of pills amounts to from five to twenty. He strongly urges that the pills be given either night or morning, according as the fits are nocturnal or by day, and invariably at the same hour in the same case. Bromide of potassium has been the favourite remedy of late years, and there is no doubt that its use is often highly beneficial and sometimes curative. The dose should vary from 10 to 30 grains three times a day, and it should be given for a considerable length of time. But probably more important than medicine is careful attention to hygiene; the patient's habits should be ascertained, and, if in fault, corrected; masturbation and excessive venereal indulgence should be checked; over-eating, and especially

over-drinking, late and irregular hours, and excitement of all sorts should be avoided. He should live quietly, keep good hours, take nourishing wholesome food, eschew alcohol as far as possible, attend to the condition of his evacuations, and, if need be, have change of air and scene. It is often a question whether the patient should give up work—whether if a man he should cease to engage in his ordinary business pursuits, if a child, give up learning. The answer to such questions must depend on the special circumstances of the case. No doubt when the fits are severe and frequent, it may be well to cease at least for a while from all mental labour and sense of responsibility; but in the great majority of cases there is every reason to believe that a certain amount of mental occupation, and it may be added of bodily exercise, is beneficial to the patient, and that, on the other hand, entire cessation from work is injurious. As a rule, therefore, the child should pursue his studies, the adult his usual avocations; but neither should be allowed to push his work to excess. Lastly, counter-irritation, setons and issues behind the neck, shower-baths, cold baths, and ice along the spine, and even the removal of the clitoris or of the testicles have each had their special advocates. There are no sufficient grounds, however, for believing any one of these measures to be really beneficial.

B.—*Eclampsia.*

Definition and causation.—This is the name which is now commonly applied to all those varieties of epileptiform convulsions which occur accidentally, so to speak, in dependence on some specific lesion or the presence of some special pathological or physiological process. Eclampsia may be one of the phenomena consequent on fracture of the skull, effusion of blood into the brain, or obstruction of a cerebral artery; it may be developed in connection with the growth of an intracranial tumour, whether this be tubercular, syphilitic, carcinomatous, hydatid, aneurysmal, or other; it is liable to occur when there has been sudden and copious loss of blood, when the brain is deeply congested, or when certain poisons circulate with the blood—it thus attends poisoning by hydrocyanic acid or absinthe, the retention of effete matters in the blood from renal disease, and in young children is often one of the earliest indications of the operation of the scarlatinal poison, or that of other infectious disorders; further, it is often induced by reflex action, and thus sometimes occurs during parturition, and in children is a frequent consequence of teething, gastro-intestinal disturbance, and many slight local conditions which in older persons would cause little or no inconvenience.

Symptoms and progress.—The fits of eclampsia are not distinguishable from those of true epilepsy. They may be exceedingly slight, they may be robbed, as it were, of one or more of the recognised stages, or they may present in a typical form all the sequence of events charac-

teristic of the haut mal. But they are often less sudden in their invasion; the patients are less liable to lose consciousness absolutely than true epileptics are; the fits much more frequently have a fatal issue, either from coma or from exhaustion; and they are much more irregular in their occurrence—probably, however, becoming more and more frequent and severe if the affection on which they depend is a progressive one, or ceasing permanently if their cause is removed. Further, with the exception that children who have eclampsia sometimes become epileptic in after life, these accidental fits seldom or never merge into true epilepsy. The diagnosis of these cases must depend less on the phenomena of the attack than on their history and the circumstances which attend them—such as the presence of constitutional syphilis, the existence of renal disease, the fact that symptoms of cerebral disorder have been gradually creeping on before the convulsions attacked the patient, the evidences of abundant loss of blood, the progress of parturition, and the like.

Treatment.—The treatment of eclampsia will depend mainly on the diagnosis at which we arrive; thus syphilitic eclampsia will need to be treated with iodide of potassium and mercury; renal eclampsia will probably demand the use of powerful drastic purgatives; anæmic eclampsia will call for good nourishment and stimulants; eclampsia arising from accidental causes of irritation will require the removal of those causes; while that variety which is connected with progressive cerebral disease can only be treated by palliative measures.

C.—*Infantile Convulsions.*

Definition and causation.—These are rarely epileptic in the true sense of the term, and come therefore properly under the head of eclampsia. There are reasons, however, for giving a separate brief consideration to them.

Convulsions arise in young children, especially during the time of teething, with remarkable readiness and frequency; and indeed Dr. West observes that convulsions in children seem often to take the place of delirium, or rigors, in adults. It is certain that they are very often developed in the course of diarrhœa and other disorders of the gastro-intestinal tract; that they occur in bronchitis and other affections of the respiratory apparatus; that they come on not only at the period of invasion of scarlet fever and other like diseases, but that they may be induced in the course of these disorders by various accidental circumstances; that they often depend on mere innutrition or anæmia; that they are common in rickety children; and that they are peculiarly liable to occur in connection with the irritation of teething. Children are, of course, liable, as adults are, to convulsions in the course of the development of tumours or other diseases of the brain or its meninges.

Symptoms and progress.—The convulsive attacks of children do not differ essentially from those of adults. They may be equally numerous, equally violent, and the 'status epilepticus' may be equally developed. They vary also in their intensity between the widest extremes. They do not, therefore, need any special description. Slight fits or threatenings of fits are very often indicated, either when the child is awake or when he is asleep, by sudden spasm of one or both hands with turning inwards of the thumb upon the palm, or by a momentary fixedness in the child's look, attended probably with pallor, dilatation of pupils, squinting, or some convulsive twiches of the face or limbs. It not unfrequently happens in children that the incidents of the fit are mainly connected with spasmodic contraction of the glottis and respiratory muscles. Respiration suddenly ceases, the face becomes livid and bloated, the veins swell, there is some rolling of the eyes, some convulsive movements of the muscles of the face; then the head falls upon the chest, and the limbs become flaccid, the pulse gets feeble, quick, and perhaps imperceptible, bloody sputum issues from the mouth, copious sweats break out, and if respiration be not speedily restored death ensues. In some instances such attacks are ushered in with a kind of crowing inspiration (laryngismus stridulus); in many they are perfectly silent. They are sometimes brought on during the continuous holding of the breath, or continuous expiration, which occurs when the child begins to cry, or when he is coughing or about to cough, and especially in connection with the paroxysmal attacks of hooping-cough. The number of fits which children suffer from and the frequency of their recurrence vary greatly. Sometimes the child has a single fit, and never any more; sometimes the fits recur many times a day, and the child may experience many hundreds of them in the course of a year. Not unfrequently, as before stated, he may pass into the status epilepticus and remain in that condition for some hours or even a day or two. Infantile convulsions are always, and on good grounds, a matter for serious alarm; it is astonishing, however, how children will suffer from almost innumerable fits occurring off and on for months and years, and yet recover perfectly. On the other hand, they are often fatal. The most dangerous are those which chiefly implicate the respiratory organs, and those which by their rapid succession render the child comatose for a long period. The immediate cause of death is either suddenly or slowly induced asphyxia, asthenia, or coma. Fits often repeated have in some instances similar results to those occurring in adults: they are sometimes followed by more or less permanent hemiplegia or some other form of paralysis, or by failure of intelligence or idiocy. Stammering, squinting, and other such defects are sometimes attributed to fits in early life.

Treatment.—The child's general health must be carefully maintained or improved; all affections, all causes of irritation which are present, must be removed. Bronchitis must be cured, diarrhœa checked, irri-

tability of the stomach assuaged; if the gums are congested and swollen and the child is evidently suffering in consequence, they should be freely lanced, and the operation should be repeated whenever the indications of irritation return; if the child has been having unwholesome or insufficient food, or if he has been over-fed, these conditions must be obviated. The various specific modes of treatment are as applicable in the case of young children as in that of adults; and hence belladonna, bromide of potassium, antispasmodics, and other remedies have all been recommended, and in certain cases have been found serviceable. In the fit itself, there seems no reason to object to the ordinary practice of putting the child into a hot bath, and applying cold water or a sponge dipped in cold water to his head or face. Chloroform inhalations may also be had recourse to. Fits may sometimes be averted by applying ammonia to the nose, or cold water to the face, at the moment of their commencement, or when premonitory symptoms are heralding their approach.

XIV. HYSTERIA.

Definition.—It is difficult to describe, still more difficult to define, hysteria. It may, however, in general terms be said to be a functional disorder of the nervous system, occurring mainly in females from the age of puberty upwards, in which the will, the intellect, the emotions, sensation, motion, and the various functions which are under the influence of the nervous system, are involved, or apt to be involved, in a greater or less degree.

Causation.—As has already been stated in the definition of the disease, hysteria principally affects females and usually makes its appearance in them for the first time between the age of commencing puberty and that of five-and-twenty. It may come on, however, previous to puberty, and at any age after twenty-five; but in the latter case more especially about the time of the cessation of the menses. Males occasionally become distinctly hysterical; but there does not appear to be the same tendency in them as in women for the disease to come on in early life. The causes of hysteria, like those of so many other functional nervous disorders, are very obscure. There are two or three, however, which seem to have a very important influence, direct or indirect, in its causation; these are emotional disturbance, sexual conditions, and occupation.

Nothing is more certain than that hysterical phenomena and the hysterical fit itself are frequently induced by circumstances which affect the emotions powerfully, such as sudden fright or horror, powerful religious impressions, disappointed love or hope deferred, grief, jealousy,

and the like. And indeed in those who are strongly predisposed to the affection the most trivial disturbances of this kind are liable to provoke violent outbreaks. Hysteria, like chorea and epilepsy, is often contagious.

The name hysteria was given to the disease under consideration in the belief that the womb was its seat. The fact that it occurs amongst men shows that that view of its origin cannot, at least in all cases, be correct. As regards females, however, there can be no doubt that the reproductive functions or organs do exercise a greater or less influence over its production. It comes on usually about the period of puberty or that of the climacteric change. Though not by any means occurring only in unmarried women, and those who are unhappily married, it occurs in them much more frequently than in such as become the happy mothers of families. And again, in no inconsiderable number of cases there is distinct evidence of involvement of one or both ovaries in the facts that they are painful to pressure and that characteristic hysterical symptoms may be induced by applying strong pressure to them. There is, however, no necessary connection between the condition of the catamenial flow and hysteria, although it must be admitted that the catamenia are often at fault in hysterical women, and that occasionally their restoration to the normal condition is attended with the restoration of the patient's general health. Nor is there sufficient ground for believing that the mere default of sexual congress either in the male or in the female has, as a rule, any important influence in its causation; excepting perhaps in so far as it may be connected with the yearning for love, the sense of neglect, jealousy, and other such feelings. Sexual excesses, and especially masturbation, have been assigned as causes.

There can be little doubt that occupation and position in life have something to do with the production of hysteria; for it is a disease which affects the higher classes in a disproportionate degree; but if these conditions are concerned in its causation, it is owing to the accidental fact that wealth brings with it the needlessness for work and the capability of indulgence in frivolous amusements and idleness, with consequent neglect of the healthy exercise and discipline of the mind. Other causes which have been assigned for hysteria are hereditary predisposition, overwork, anæmia, debility, and other forms of failure of health; but any influence they may exert is at best remote.

Symptoms and progress.—In describing the clinical phenomena of hysteria we will first discuss the mental characteristics of those who suffer from it, and then consider *seriatim* the various motor, sensory, and sympathetic disturbances which are apt to be associated with them.

The *mental conditions* of hysterical patients present the greatest variety, and yet there are gradations between the extreme conditions which prove their relationship. In many cases women who are liable to hysterical attacks under occasional states of ill-health or excitement

are in the intervals between their attacks as healthy in body and mind, and as free from all caprice or peculiarities of temper, as we could wish to see them. They will often acknowledge, however, that at the moment when hysterical feelings come upon them, they feel compelled to yield to them, and indisposed to make any effort to restrain them; and that yet if anything occurs to incite them to use self-control, they are able to resist them successfully. In other cases the patient is nervous and excitable, with little control over either her emotions or her actions, apt to laugh or cry on the slightest provocation and incongruously, and apt also to suffer from time to time from the various complications of hysteria. But in a very large proportion of cases the whole moral character of the patient is more or less profoundly altered. She is apathetic and neglectful of her duties, or exacting, selfish, and suspicious, exaggerating all her trivial annoyances and discomforts or disorders, resenting all healthy advice or reasonable attempts to promote her welfare, and quarrelling therefore it may be with her husband or dearest friend, but pouring out profuse affection on all those acquaintances, however new they may be, who affect to pity her condition, make the most of her ailments, and adapt themselves to all her changing moods and caprices. Under such circumstances it is astonishing to see women, well-nourished, and apparently in the best of general bodily health, remain for months and years useless members of society, suffering from paralyses and other maladies which they profess to look upon with the utmost alarm (and which they declare perhaps to be family complaints) not only with quiet complacency, but with a studied resistance to all plans of treatment likely to be of service to them. They are probably only too willing, however, to put themselves into the hands of some fashionable charlatan, or to do anything else which will render their misfortunes in any degree notorious. It is but a step from hugging her ailments and exaggerating them to malingering. And although we cannot fairly accuse the great majority of hysterical patients of shamming, shamming is by no means uncommon. The craving for pity and notoriety increases by being fed; the greater the commiseration she excites, the more does she endeavour to be worthy of it, and the more serious become the ailments from which she is suffering; and soon perhaps new phenomena develop. It is an interesting and important fact that the nature of these phenomena is not unfrequently determined by the direction which the interest and solicitude of the doctor or friends happen to take. If they pity her failing appetite, she soon perhaps affects to live without food; if it be observed that her urine and motions are scanty, she finds before long that they cease altogether; if it be a matter of wonder or speculation what becomes of her evacuations, she will be found perhaps to vomit urine or fæces, or blood. It is by such persons, though not by these alone, that various other forms, more or less singular, of malingering are practised. Thus at one time a patient

will bring on hard œdema or spurious elephantiasis of the arm or leg by the constant application of a ligature round the upper part of the limb, and will even submit to its amputation; at one time she will, by the constant application of some irritant substance, fret her skin into ulcers, and thus even cause perforation of the stomach; at one time she will place lumps of coal up her vagina and pretend that she is suffering from vesical calculus; at one time she will affect to have communion with the Virgin Mary, to have the marks of the stigmata on her hands and feet and side, and at the same time, probably, to live devoid of all those natural appetites and wants which are inherent in mortality. Hysterical patients sometimes suffer from a form of insanity known as hysterical mania; and occasionally after the lapse of years pass into a state of dementia.

We will now proceed to describe the various phenomena which are so apt to go along with the mental states which have just been considered, and which form, as a rule, the more striking phenomena of hysteria.

1. *Convulsions and spasms.*—Hysterical convulsions vary in their severity and duration, and have a more or less general resemblance to those of epilepsy, from which, however, it is important to distinguish them. The patient is rarely attacked without warning. She has probably, for some little time previously, been suffering from hysterical symptoms; she has been laughing, crying, or sobbing, or talking wildly or gesticulating violently, or she has complained of a sense of constriction or of a ball in the throat, or has manifested, in a marked way, some of the mental or emotional phenomena which are characteristic of hysteria. Then suddenly, perhaps, she utters a loud scream, and falls upon the sofa or the ground violently convulsed. The fit may last for a few moments, or be prolonged for a quarter of an hour, or continued by successive attacks for many hours, interrupted from time to time by cries, and sobs, and laughter. Such phenomena generally also attend the subsidence of the attack; or, if the patient be worn out with her long-continued exertions she falls into a sound sleep. The main features by which the hysterical fit may be distinguished from the epileptic are the following:—the hysterical patient, no matter how severe her attack may be, is very seldom totally unconscious; she can generally be roused either by the voice of authority, or a douche of cold water; she is noisy—the epileptic utters a single cry, or none at all, while the hysterical patient probably screams and cries and laughs and groans, or talks volubly and incoherently off and on during the whole of her attack; her convulsions are much more general and extensive than those of the epileptic—she throws her arms and legs about in all directions, she twists her body into the most grotesque attitudes, she suddenly raises herself to the sitting posture, and then throws herself violently down again, but with all this violence and excess of muscular effort she rarely, if ever, injures herself; the con-

vulsions are rarely tonic at any period of the attack; they are rarely, if ever, unilateral, and the face (unless when the patient is crying out) is free from the hideous distortion of epilepsy; she does not bite her tongue; the eyelids are closed and tremulous, but the pupils respond to light, and there is no tendency to squint; respiration never ceases, but is from the beginning noisy and irregular, and consequently, although the skin may become hot and perspiring, the patient never presents that lividity of countenance which attends the true epileptic attack; she does not discharge the contents of her rectum and bladder; and lastly, if we investigate the history of the patient, we never find that she suffers from attacks of the petit mal or epileptic vertigo. Yet, though the distinction between epilepsy and hysteria is for the most part easy, instances are sometimes met with in confirmed and severe cases, in which the hysterical attack puts on some of the features of epilepsy. It is then attended with sudden and total unconsciousness, and it may be with tonic spasm, temporary arrest of respiration, lividity of face, and biting of the tongue; but even here the antecedent presence of the globus hystericus and other indications of hysteria, and the ultimate conversion of the attack into one of obvious hysteria are generally sufficient to render diagnosis easy. Charcot points out as a further distinction between these attacks and those of true epilepsy, that they never lead to impairment of the intelligence or dementia; he further points out as an important distinction between the status epilepticus and the corresponding condition in hysteria, that in the former case the temperature rises to 103° or 104° or more, while in hysteria it rarely exceeds the normal by more than one or two degrees.

But besides these general convulsive attacks, hysterical patients are liable to permanent or tonic contractions of groups of muscles or limbs. These, as will presently appear, are not unfrequently associated with paralysis. Among them may be mentioned spasmodic closure of the hands, trismus, and spasmodic contractions at the knee or other joints.

2. *Hyperæsthesia* is exceedingly common among hysterical women. It may be general, or hemiplegic, or paraplegic, or it may affect a limb or a joint, the mamma or the ovary, the spine, or indeed any part of the surface, or any organ. Pain varies in its severity, is sometimes induced only by pressure, but often occurs independently of all external sources of irritation. It is a curious and suggestive, but not invariable, characteristic of it, that the patient will shrink from the slightest touch when she is expecting it, and yet will allow the painful part to be compressed and handled violently when her attention is directed to other matters. A common pain of which hysterical women complain is that which is termed *clavus*; it is generally referred to the forehead just above the eyebrow, and is likened to the effect of a nail driven into the skull. The most interesting variety of hyperæsthesia, however, is that of which the *globus hystericus* forms a part. The globus

hystericus is a sensation as of a ball rising into the throat and impeding respiration; it is of frequent occurrence in hysterical patients, and is commonly present before and during paroxysmal attacks. It often seems to spring from the iliac fossa. The patient then complains of pain or tenderness on pressure in this situation, whence from time to time the hysterical aura, as it may be termed, seems to spread: first to the epigastrium, causing nausea and vomiting; then to the chest, provoking violent action of the heart and palpitation; then to the neck, constituting the globus hystericus, which is often associated with sobbing, choking, and other such symptoms; and thence finally, according to M. Charcot, to the head, when it induces noises in the ears, dimness of vision, and clavus, all on that side of the body from which the aura started. These phenomena constantly precede the occurrence of the hysterical fit, and, according to the older writers, with whom M. Charcot is completely in accord on this point, are referrible to some peculiar condition of one or other ovary, generally the left. He states: that in a large number of hysterical women there is a tender point which may be discovered on deep pressure made directly backwards at the point of intersection of the horizontal line drawn between the two antero-superior iliac spines, and the continuation downwards of the vertical line which marks the lateral boundary of the epigastrium; that this point represents the ovary, which may, in fact, when the abdominal walls are flaccid, be often distinctly felt in this situation; and that continued pressure upon it will induce all the phenomena above described of the hysterical aura. This iliac or hypogastric pain varies in severity; in many cases it can only be discovered by hunting for it; but in many extreme pain and tenderness, so great as to forbid the slightest pressure, occupy not only the ovary but the superposed muscles and skin; and occasionally these phenomena become so widely diffused as to simulate the local symptoms of peritonitis. Intolerance of light, intolerance of sound, and intolerance of certain sapid or odorous substances, often associated with extreme acuteness of the special senses, is very common in hysterical women. But here again the phenomena generally present that marked characteristic of hysteria, namely, that the patient will complain bitterly of the slightest impression when her mind is directed towards it, but will endure the most discordant sound or the brightest light when her attention is distracted by other objects.

3. *Anæsthesia* is frequent among hysterical persons. It may occur in various parts of the body, and be limited to the distribution of a single nerve; it may affect the sense of smell, or taste, or may implicate the eye, causing dimness of vision or difficulty in recognising colours. The most remarkable cases, however, are those of hemianæsthesia, with or without co-existing loss of motor power. In this variety the loss of sensation as a rule involves uniformly the whole of one side of the body—leg, trunk, arm, and head and neck—ceasing abruptly at the

median line; and it involves not merely the skin, but the mucous membrane of the mouth and the organs of sense, so that taste and smell are lost upon the affected side, and the eyesight probably fails. Further, it usually implicates the deeper-seated tissues as well, namely, the muscles, bones, and joints. It may be complete and profound, or it may be merely insensibility to pain, with or without insensibility to variations of temperature. The anæsthetic parts are usually pale, and their temperature more or less considerably reduced. Hemianæsthesia is apt to come and go, and occasionally shifts to the opposite side of the body. Sometimes the anæsthesia becomes bilateral.

4. *Paralytic conditions* are probably the most common of the complications of hysteria. Like anæsthesia, paralysis may affect any part; it may involve the hand, the forearm, the entire upper extremity; it may affect the leg or some part of it; in some cases it assumes the form of paraplegia, in some that of hemiplegia; or it may be irregularly distributed, or general. It seldom implicates the muscles of expression. The paralysis may be complete or incomplete. The affected limb or limbs may be flaccid or rigid. Faradic contractility remains for the most part unimpaired, but electric sensibility of the muscles is generally more or less completely lost. The muscles usually do not waste. In the majority of cases hysterical paralysis may be distinguished from other forms of paralysis with tolerable readiness, but not always.

If the paralysis be hemiplegic, it comes on probably after an hysterical fit; it involves the arm and leg, but neither the tongue nor the face; the affected limbs are probably rigid—the arm bent, the hand firmly closed—while the lower extremity, on the other hand, is extended—the toes pointed, and the limb and pelvis movable only in mass; it may be that the arm is flaccid while the leg is contracted, or conversely; the paralysis is probably associated with hemianæsthesia. It may be remarked that the hemiplegia of organic brain-disease is only occasionally associated with complete hemianæsthesia; that it is never attended with persistent rigidity from the beginning; and that if in this case there be any difference between the arm and leg in this respect, it is the arm and not the leg which becomes rigid. If the paralysis be paraplegic, the limbs are usually rigid and in a condition of extension; and the paralysis with rigidity is probably, as in the other case, suddenly developed.

Whether the paralysis be hemiplegic or paraplegic, or limited to a limb or part of a limb, it is apt to come and go, and to shift from limb to limb, or to involve more or less suddenly other limbs besides those first affected; and, above all, it is generally associated with other phenomena indicative of the presence of hysteria. It is important, however, to recollect: that, although hysterical paralysis generally presents variations in degree, in character, and in site, it is (especially

in its hemiplegic or paraplegic form) liable to continue for years or for life; and that although as a rule the muscles remain unaffected as regards their bulk and contractility, they may, in cases of long standing, undergo degenerative changes from disuse, in connection with which secondary lesions may also take place in the cord.

5. *Affections of the larynx and air-passages.*—Aphonia is very common; the patient loses her voice completely and speaks only in the feeblest whisper; she probably, however, has no soreness in the throat, no difficulty or pain in swallowing, no evidence whatever of local disease. The voice, moreover, is generally feeblest when the patient is asked to display her powers; and sometimes reappears with sudden force under the influence of momentary excitement or it may be of forgetfulness. In some cases there is actual dyspnœa, which becomes so extreme as to demand operative procedure. Attacks simulating those of ordinary asthma are occasionally observed. Not unfrequently a peculiar cough, which Sir Thomas Watson describes as 'loud, harsh, dry, more like a bark, or a hoarse bleat, than a cough,' is one of the special phenomena of hysteria; it is apt to come on in paroxysms, which may continue for hours without cessation, and may come on daily or nightly for weeks or months. In some cases, without apparent cause, and with a pulse but little exceeding the normal rate, the respirations will suddenly rise to 40, 50, or even 70 or 80 in the minute, and continue thus for some minutes, or on and off for hours, and yet without other evidence of dyspnœa or distress.

6. *Affections of the alimentary canal.*—In some instances patients suffer from well-marked trismus, which interferes seriously with both speaking and eating; occasionally they complain of difficulty of deglutition; and distension of the stomach, with rumbling and eructations, is of common occurrence. Hysterical patients often suffer from vomiting, and in some cases this constitutes the most serious part of their malady; the vomiting is apt to come on after every meal, or it may be at some particular time of the day, and to be continued day after day for months or years. This sickness is frequently associated with good or even voracious appetite; but the bulk of matters vomited often seems in excess of the ingesta, and after a time extreme emaciation and debility probably ensue. In some instances the symptoms almost accurately resemble those due to ulcer of the stomach. The bowels are usually constipated, and there may be more or less pain in defæcation.

7. *Affections of the urinary organs.*—Retention of urine often occurs. Doubtless it sometimes depends on paralysis of the bladder, contraction of the sphincter, or pain in the act of micturition; but not unfrequently, like other hysterical conditions, it is more or less within the control of the patient, who makes no attempt to relieve herself voluntarily so long as she can enjoy the morbid pleasure of having the catheter passed for her. But more interesting than this are the phe-

nomena connected with the secretion of urine. It usually happens, after an hysterical fit, or after other paroxsymal nervous disorders, that the patient excretes large quantities of pale limpid urine. And such profuse discharges are not unfrequent at other times. But the opposite condition may be present. The patient consecutively for many days does not pass more than a few ounces of urine. In a remarkable case published by M. Charcot, the sufferer, a woman, forty years old, for more than a couple of weeks passed every other day only five grammes of urine, and none on the intervening days, and for a continuous period of ten days secreted no urine whatever. During one month her average daily yield was only three grammes, and during another month only two grammes and a half. In this case the diminution and suppression of urine were unconnected with renal disease, but were associated with constant vomiting, the quantity of fluid vomited having some supplementary relation to the quantity of urine voided. Further, the vomit contained urea, yet the urea secreted daily by the kidneys and stomach together was very far indeed below the normal. For a period of twelve days it amounted from both these sources to only five grammes daily. M. Charcot remarks, in reference to such cases, that the escape of even a small quantity of urea in calculous obstruction of the ureters often serves to ward off dangerous symptoms, and that doubtless the same rule applies here; but he further observes that there is probably in hysterical ischuria an impairment of the functions of assimilation which diminishes the total amount of urea and extractives to be discharged from the body.

8. *Of affections of the reproductive system* little remains to say beyond what has already been said. Amenorrhœa, menorrhagia, and other menstrual disorders are no doubt frequent accompaniments of hysteria; but many hysterical women are quite free from them. Again, the hyperæsthesia which is so common in various parts of the body in hysteria may affect the vulva or vagina and render the act of coition intolerable; whilst on the other hand, lascivious feelings are occasionally strongly developed, and either induce in the patient a demeanour, probably towards certain individuals, which far transgresses the bounds of womanly self-respect, or give a motive for feigning disease of the sexual organs. It is not surprising that the mental obliquity of such patients should occasionally incline in this direction.

9. *Other affections* which hysterical patients are apt to mimic are those of the *spine*, of the *joints*, and of the *mammæ*. These have already been adverted to under the head of hyperæsthesia. It need only be added that they often closely simulate inflammatory disorders of the same organs, and are apt to be mistaken for them; and that we must not hastily assume that a suspected hysterical affection of these parts is not hysterical because we discover, in addition to pain and tenderness, more or less swelling.

10. *Spinal irritation* is the name which has been given to a group of hysterical phenomena which have been particularly described by Mr. Teale and the Messrs. Griffin, and is still by many regarded as a distinct affection. It is characterised by the presence of more or less considerable tenderness at some spot in the course of the spine, or more rarely generally throughout its whole length, and by pain or other nervous phenomena referred to those parts of the body whose sensory nerves are in relation with the tender spot, or to certain of the viscera. Moreover, pressure upon the tender spot aggravates, or it may be actually induces, the phenomena in question. If the tenderness occupy the upper part of the cervical spine, the neuralgic pain associated with it affects the occipital region, or it may be even the distribution of the trifacial; if it be a little lower down, the neck suffers; if it occupy the situation of the cervical enlargement the pain is experienced mainly in the arms; if it be present in the dorsal region then the parietes of the chest or abdomen suffer; if it implicate the lumbar enlargement, the pelvis and the lower extremities are the chief seats of pain. Further, the sensation of a lump in the throat, palpitation, dyspnœa, spasmodic cough, gastralgia, nausea and vomiting, irritability of the bladder, or suppression of urine are all apt to attend the spinal tenderness; but the particular group of these complications appears to be determined, like the neuralgic pains, by the situation of this tenderness. In all respects besides those which have been enumerated, the symptoms which the patients present are identical with those of other forms of hysteria, and indeed the phenomena of these affections are, if not common to both, inextricably interwoven. The course of the disease, moreover, is identical in all respects with that of hysteria.

The *diagnosis* of hysteria is not always easy; and yet if the patient be carefully watched from day to day it is difficult to remain very long in doubt. It is not, however, an unnecessary caution to remind the reader that not only does hysteria ape many diseases so as to be readily mistaken for them, but that other diseases often simulate the phenomena of hysteria and may be easily taken for it. There is always a great temptation to assume that nervous disorders which we do not understand, and obscure visceral affections, in females are hysterical. Among diseases which may thus be mistaken for hysteria should especially be named chronic inflammatory conditions of the brain and cord, and tumours of the brain. In forming a diagnosis we must carefully consider all the features which the special affection from which the patient suffers presents, and how far and in what respects they differ from those of lesions of the same parts which are not of hysterical origin. We must also look carefully to the various complications which attend the main affection, or which supervene from time to time, or alternate with it; for it rarely happens that a patient suffering from an hysterical joint or from hysterical hemiplegia or paraplegia, does not also at one time or another have an attack of aphonia, or retention of urine, or a

bout of intermingled laughing and crying, or a distinct hysterical fit, or that the original affection does not undergo some striking change, or shift to some other part. Lastly, we shall often be importantly aided in coming to a decision by careful observation of the demeanour and conduct of the patient, and of her general tone of thought and feeling.

Hysteria is very common; and varies from a slight affection of little importance to one of such gravity that it renders the patient a lifelong invalid, and her existence a burden and a misery to herself and those about her. Fortunately the milder cases are by far the most common; and in many of these complete recovery takes place, while in many recovery is so far complete that there only remains a liability to the outbreak of slight hysterical phenomena under special circumstances of ill-health or excitement. Not unfrequently, however, patients suffer from hysterical vomiting, alternating it may be or associated with other hysterical symptoms, for years; or they remain hemiplegic or paraplegic and bed-ridden for one, two, ten, or twenty years; or they suffer from urinary disorders, or aphonia, or joint-affections for an equally indefinite period; or they are the victims of constantly recurring violent fits. In some cases patients continue thus for life. It may be said generally that the longer the phenomena have persisted, the less likely is ultimate recovery to take place; but it must never be forgotten that (unless any organic complication has arisen) there is always a possibility that the patient will get well, and not only get well, but get well suddenly. The patient who has been confined to her bed paralytic for years will perhaps, under the influence of some sudden impulse or mental or emotional excitement, recover the complete use of her limbs; the patient who appeared doomed to lifelong voicelessness will suddenly speak aloud in her natural tone.

Pathology.—We do not pretend to give any account of the morbid anatomy of hysteria or of its pathology. On these heads little or nothing of any importance is known, and we do not care to speculate. It is, so far as we know, a purely functional disorder.

Treatment.—The treatment of aggravated hysteria is exceedingly difficult, and all the more difficult that the patient's condition excites in those about her that sympathy which she craves; and that consequently that judicious firmness of management which the medical man should exercise is apt to be resented not only by herself but by her friends. Nothing, indeed, is more injurious to such patients than the pity and attention they receive; they live for them, they lay their plans to attract them, and their moral and bodily conditions deteriorate under their influence. On the other hand, the exercise of a judicious firmness is essential for their successful treatment; and this it is impossible for the medical man to accomplish unless he acquires the confidence, if not of the patient, at all events of those under whose control she is. For this purpose it is not necessary to be harsh, indeed

harshness is likely to defeat its object; but the respect, and if possible the trust, of the patient should be acquired by the cultivation of kindliness and friendliness of manner with firmness of purpose. There should be on the part of the doctor a judicious blending of the 'suaviter in modo' with the 'fortiter in re.' No doubt hysterical patients are extremely disposed to exaggerate their symptoms. No doubt they do occasionally wilfully and grossly deceive those about them; but it must not be assumed that there is generally intentional exaggeration, still less that there is imposition. They do, as a rule, really suffer that of which they complain, and suffer more when their attention is directed to the ailing part. It is impossible in a brief space to lay down any rules with regard to the general treatment of these cases. No doubt it is important to improve the general health, to relieve dyspeptic symptoms, to cure anæmia, to regulate the catamenia, to see that the bowels act regularly, to insist on regular hours, good wholesome diet, and daily exercise, and it may be to order change of air and scene; especially it is important to make the patient take an interest and pleasure in some useful occupation or some intellectual recreation or study. But it must never be forgotten that, to use Sir Thomas Watson's words, 'behind the moody, reserved, and tricky behaviour there often lies some mental or emotional cause—some hope deferred or disappointed—which being ascertained, and capable of satisfaction and satisfied, the patient may be restored to her customary health.'

Among the drugs which have been employed with more or less success, or want of success, may be especially mentioned iron, zinc, vegetable tonics, assafœtida and other fetid gum-resins, and stimulants. Alcohol in various forms is often recommended by the medical attendant or had recourse to by the patient; but alcoholic beverages, chloral, opium, and other narcotic medicines, should be given or allowed with extreme caution, for the temporary relief which they give is very apt to lead to their habitual use and ultimate abuse. In the hysterical paroxysms very often nothing more is needed than to lay the patient down and unfasten her dress or anything tight about her neck; the paroxysm may, however, frequently be cut short or prevented by the free use of cold water—by dashing it in quantity over the neck and face—or as Dr. Hare points out, by firmly closing the patient's nose and mouth for a time, or until her dyspnœa is such that she is compelled to draw a long breath. Less valuable than these measures, though not altogether to be despised, are the inhalation of sal-volatile or smelling salts, and the exhibition of ammonia, assafœtida, or ether.

M. Charcot, besides pointing out the readiness with which hysterical paroxysms may be induced by pressure made in the region of one of the ovaries, shows that in the same cases powerful, regulated, and sustained pressure is generally efficacious in arresting the paroxysm, however violent it may be.

The removal or relief of the various local phenomena of hysteria

frequently demands special forms of treatment; aphonia may generally be cured by faradism of the throat, effected either by placing one pole of the instrument within the throat and the other external to it, or by placing the poles on either side externally. Paralytic affections are largely benefited by the same treatment, or by the frequent use of the cold douche. Dr. Reynolds especially recommends the application of narrow strips of blister around the affected limbs. Anæsthesia also is sometimes remediable by faradism. But for this, especially if there be at the same time coldness of surface and imperfect circulation, as also for the cure of hysterical contractions, galvanism is probably preferable.

It is mainly in cases of hysterical anæsthesia that Dr. Burq's[1] metallo-therapeutic treatment has come into vogue. This consists in the local application of some metal to which, by experiment, the patient is found to be sensitive. The metals employed are gold, silver, iron, copper, and zinc. To ascertain which of these is appropriate, discs of each must be applied in succession for two or three minutes each to the region about to be operated upon. This point having been determined, bands or groups of discs of the selected metal must be kept for a quarter of an hour or so in close contact with the affected surface by a bandage or other means. It would appear that the result is that the affected part (whether it be the skin or organ of special sense) gradually recovers its sensibility, and that associated with this there is a return of warmth and circulation, and of muscular power. But it would also appear: that whatever improvement there is on the one side of the body, is at the expense of the opposite side, which becomes anæsthetic in proportion as the other recovers; and, moreover, that the recovery is only temporary.

The sudden cure of hysteria in any of its forms is almost always possible under the influence of powerful emotional excitement. Thus a sudden alarm that the house is on fire will sometimes cause a woman who has been paraplegic for years to rush from her bed with the full use of her limbs; the unexpected infliction of sudden and severe pain generally suffices to make the dumb cry out at the top of her natural voice; the promise that if a long-closed hand opens by a certain day it shall have a valuable trinket placed in it generally calls for fulfilment.

XV. CATALEPSY, ECSTASY, AND OTHER CONDITIONS ALLIED TO HYSTERIA.

A large number of curious nervous phenomena—motor, sensory, emotional, and intellectual—occur, which are difficult to describe save by the help of illustrative cases, difficult to classify, and difficult to attach to specific lesions or specific conditions of the nervous system.

[1] See report by MM. Charcot, Luys, and Dumontpallier, quoted in the *British Medical Journal*, May 19, 1877.

In a large proportion of cases they originate in powerful mental excitement, and more especially in such as is connected with religious fervour; they sometimes also arise from imitation or moral contagion. Young persons, from the period of commencing puberty to the termination of adolescence, and more particularly females, or males of emotional temperament, chiefly suffer. The patients are often distinctly hysterical; and not unfrequently hysterical paroxysms and some of the various other phenomena which have been considered under the head of hysteria complicate some of the conditions we are now about to describe, or alternate with them. Indeed, if we look to the exciting causes, to the class of persons who are most commonly affected, to the character of the symptoms, and to their frequent association with hysterical phenomena, we can scarcely avoid regarding the affections under consideration as varieties of hysteria. We believe that they generally are so. In some cases, however, they seem to be related rather to chorea, epilepsy, or insanity.

1. *Rhythmical and other methodical movements.*—These present innumerable varieties of character. In some cases the patient performs unceasing oscillatory, undulatory or rotatory movements of the head and neck, or of the entire trunk. In some she is seized with an uncontrollable impulse to run forwards or backwards. In some she is impelled from time to time to leap into the air. To the same class must be referred the violent rhythmical movements which attended the 'dancing mania' of the Middle Ages.

2. *Catalepsy.*—By this term is meant an attack of loss of sensation and of consciousness, attended with remarkable stiffening of the muscles. The patient for the most part is attacked suddenly, after more or less mental or emotional disturbance; she becomes pale and corpse-like, the respirations being slow and tranquil, the pulse soft. She cannot be roused, and is entirely insensible to pain. But the most striking phenomenon is the stiffness of the muscles, which is such that the limbs, head and neck, or features, when forcibly put into any position, however constrained and unnatural it may be, or however difficult to be supported by the healthy muscles, retain that position for some length of time. But although the patient appears to be unconscious to external impressions, and to remember nothing of what happens during the attack, she will sometimes sing or talk whilst it is upon her, or indicate by her expressions the presence of pleasing or painful impressions. A cataleptic condition may also occur in patients who still retain full consciousness. Cataleptic attacks may last from a few minutes to several days; there may be a single attack only; or they may recur with more or less frequency.

3. *Ecstasy* is a condition in which the patient is absorbed in some all-engrossing fancy or delusion. It is the condition to which weak-minded persons are wrought under the influence of revivalist preachers, and in which they are sometimes impelled to plead fran-

tically for pardon for imaginary misdeeds, are sometimes in a delirium of complacency and joy at their supposed enrolment among the saved. It is the condition into which those persons fall who believe that they see visions of Christ, of the Virgin Mary, of saints, or of angels, or who hold familiar intercourse with them, or who receive divine messages. It is the condition into which the medium is not unfrequently brought under the mesmeric influence. It represents also the mental condition of the dancing maniacs of the Middle Ages. The nature of the fancies or delusions under which such patients labour may, therefore, present the widest range of variety, and their effects on the mind all degrees of intensity. Their influence over the actions of the patient, moreover, is very various. Thus, while one will gesticulate violently and roar or scream his prayers or denunciations; another will dance or sing or utter pious ejaculations; another will sit apart with an air of self-satisfaction or quiet happiness; and yet another will be transfixed or stunned, as it were, with intense anxiety or horror. In some of these cases the patient remains motionless and apparently insensible to every external impression for days together. But generally they are not wholly insensible; and although the mind may not be capable of being diverted from its engrossing thoughts, the pupils contract and the eyelids close under the influence of a strong light; sneezing and watering of the eyes may be induced by the application of ammonia or snuff; and the respiratory muscles may be made to act powerfully under the shock of a jugful of cold water.

4. *Double-consciousness.*—A curious condition, allied to the last, is sometimes witnessed, in which the patient appears to have, as it were, a double life—the one her normal state of existence, in which she is fairly sensible, and knows and understands, and perhaps takes an interest in, everything that goes on about her; the other a condition of ecstasy or somnambulism in which her mind is under the dominance of delusions, and in which the same lines of thought and feeling and the same delusions are continued through the successive ecstatic paroxysms; and in neither of which has she any recollection or knowledge of what occurs in her alternative condition. Occasionally these strange phenomena may be prolonged for years, the one state passing into the other almost suddenly several times a day. The waking condition, indeed, may form but a small portion of her existence, and may itself be attended with curious motor, sensory, or mental phenomena.

Treatment.—In treating the various cases which have just been considered it is important not to lose sight of the fact of their intimate relations with certain other nervous diseases, more especially epilepsy, hysteria, and insanity, of which indeed in the great majority of cases they may be regarded as mere varieties. Their treatment, therefore, resolves itself mainly into the treatment of these affections. Everything calculated to improve the general health of the body is indicated; but if a cure is to be effected it is rather by judicious management than by medicines.

XVI. TETANUS. (*Trismus. Lockjaw.*)

Definition.—Tetanus is an acute and generally fatal disorder, characterised by painful tonic spasms of the voluntary muscles, and usually traceable to some local injury.

Causation.—Traumatic tetanus may originate in a simple bruise, a trivial graze of the skin, the wound inflicted by a mere splinter, or a clean cut. But it is far more commonly due to compound fractures or other injuries attended with laceration or crushing. It is generally believed that injuries of the extremities are much more liable to be followed by it than injuries of the head and neck or trunk; but, as Mr. Poland justly remarks, the limbs are far more prone to accidents than other parts, and it is probably on this account alone that their wounds are credited with a disproportionate proclivity to tetanus. But climatic conditions also are largely concerned in the production of tetanus; for the disease is much more common in hot than in cold or temperate climates; and although it so frequently supervenes on wounds received in battle, it occurs much more frequently when the wounded are exposed to cold and wet than under opposite circumstances. Indeed the idiopathic form of the disease, which is somewhat unfrequent, is usually referred, and probably with reason, to the influence of these latter agencies—agencies which also induce rheumatism, pneumonia, and other internal inflammations. Tetanus may occur in either sex, and at any age. In the West Indies it is very common in new-born children, in whom it is supposed by some to be due to the division of the umbilical cord; and it occasionally happens in women after parturition. It has been attributed to intestinal irritation provoked by worms or other like causes. The supervention of traumatic tetanus appears to be wholly uninfluenced by the character of the changes going on in the injured parts.

Symptoms and progress.—Tetanus comes on after injury at periods varying between a few hours and three or four weeks—most commonly, according to Sir T. Watson, between the fourth and fourteenth day. When the disease is due to exposure, it always supervenes very quickly—occasionally in the course of a few hours.

The first symptoms of which the patient complains are usually pain and stiffness of the muscles of the jaws and neck—symptoms which he probably refers to cold, and describes as sore throat and stiff neck. He has difficulty in opening his mouth, in masticating, and in moving his head, which is soon followed by dysphagia, and by spasmodic attacks of pain and aggravation of his difficulties, provoked especially by every attempt to use the affected muscles. By degrees the stiffness and tendency to painful spasm extend to the other voluntary muscles: to those of the back, which by their action on the trunk tend to curve the body backwards; to the inspiratory muscles, especially

the diaphragm, the implication of which involves more or less difficulty of respiration, and occasional attacks of more severe dyspnœa, attended with acute pain striking through from the ensiform cartilage to the interscapular region; to the muscles of the abdomen, which get rigid and knotted; to those of the extremities, which become difficult of flexion, and from time to time powerfully and violently extended; and to those of expression, which by their tonic contraction impress upon the patient's features a fixed painful look (the *risus sardonicus*), which becomes intensified during each recurring spasm. The muscles of the tongue and eyeballs, and those which move the hands and feet, usually escape or are involved late and to a slight extent only.

As the disease progresses all the implicated muscles become more or less permanently stiff, and the stiffness gradually increases. But from the beginning the patient is liable to paroxysmal attacks, during which all his symptoms are enormously aggravated, and which come on at irregular but diminishing intervals, sometimes every quarter of an hour, sometimes every ten or every five minutes, and last individually from a few seconds to several minutes. These occur for the most part spontaneously, but are readily induced by any muscular effort, by moving the patient, or even by the slamming of a door and other such-like trivial causes.

In the fully-developed disease the patient, during the interparoxysmal periods, probably lies stiff in bed upon his back. The muscles of the trunk, limbs, and neck are hard and rigid; the jaws cannot be opened at all, or admit of being separated only to the extent of a few lines; the face wears a painful expression, the brows being knit and at the same time transversely wrinkled, the eyes somewhat closed, the angles of the mouth drawn outwards and upwards, the lips apart, and the grooves extending from the ala of the nose towards the angles of the mouth deepened; the mouth and fauces are clogged with saliva, which he has difficulty in swallowing; the voice is feeble, possibly reduced to a whisper; and the respirations are rapid and shallow. Further, he probably complains of more or less general pain or soreness, and especially of pain extending from the scrobiculus to the back. During the paroxysms his sufferings become extremely aggravated, and frightful to witness. His arms and legs (especially his legs) become more powerfully extended, and at the same time widely separated; the extensor muscles of the spine arch the trunk and head and neck powerfully backwards, so that not unfrequently the patient rests only on his head and heels; the respiratory muscles get more or less fixed, respiration difficult, and the face pale, livid, or ghastly; the distortion of the features, moreover, is now extreme—the forehead corrugated by the combined action of the frontales and corrugators, the eyeballs fixed and staring, the eyelids rigid and partly closed, the nostrils dilated, and the angles of the mouth drawn outwards and upwards so as to impart that peculiar appearance of grinning which has

been referred to. The lips moreover are retracted, exposing the clenched teeth; between which bloody saliva occasionally flows in consequence of the accidental wounding of cheek or tongue by their sudden closure at the commencement of the paroxysm. The paroxysms are said frequently to come on with increase of the diaphragmatic pain; and during their continuance cramp-like pains of the most agonising character pervade the contracted muscles.

Certain other phenomena to which it is desirable to draw attention present themselves in the course of tetanus. The pulse is for the most part rapid and feeble, and its rapidity and feebleness increase with the progress of the case, and are especially observable during the paroxysms. At such times also the skin, which is generally moist or perspiring, breaks out into profuse sweats. The urine is for the most part scanty, and the bowels are constipated; but the patient has entire control over bladder and rectum. According to Dr. Senator, there is no increase of excretion of the urinary solids. In the great majority of cases the patient retains his senses unimpaired throughout his illness, and is conscious up to the moment of death. He seldom sleeps, or he sleeps only by snatches. Sometimes the spasms cease entirely during sleep. The temperature in tetanus is generally somewhat above the normal, and liable to irregular diurnal variations. It does not usually exceed 100° or 101°, but may rise from time to time, even in cases which ultimately do well, to 102°, 103°, or more. Nevertheless, when the temperature reaches or exceeds 103°, the symptom must be regarded as of serious import. Occasionally, with the approach of death, the temperature rises rapidly, and it may then attain an elevation of 110° or even 112°. Sometimes in the course of tetanus the temperature becomes sub-normal.

When the tetanic spasms affect only or principally the muscles of the jaw, the affection is often termed *trismus* or *lockjaw*. When, as usually happens, the body during the tetanic spasms is arched backwards, the condition is termed *opisthotonos*. In those rare cases in which, owing to the predominant action of other muscles, the body is curved forwards or to one side, the condition of *emprosthotonos* or *pleurosthotonos*, as the case may be, is present.

The prognosis of tetanus is very gloomy; almost all traumatic cases, and the great majority of idiopathic cases, die. According to Mr. Poland, taking all forms together the mortality is at the rate of about 88 per cent. The most rapid cases, according to the same writer, die in four or five hours. But death has been delayed until the thirty-ninth day. More than half the total number of fatal cases perish during the first five days. Death is usually caused either by asthenia or apnœa, or by a combination of these conditions. It not unfrequently occurs suddenly in one of the spasmodic attacks, and is then probably due immediately to spasm of the respiratory muscles, and possibly to those of the glottis.

Tetanus may be simulated by hysteria, by inflammatory affections of the spinal cord, and especially by the effects of strychnia and other allied drugs. As regards the first two classes of disease, there can seldom be any real difficulty in distinguishing between them and tetanus, in consequence in the one case of the supervention of paralysis or other signs of organic lesion of the cord, in the other case of the association of various hysterical phenomena with the spasmodic muscular rigidity. Strychnia-poisoning, on the other hand, may be readily confounded with tetanus. The chief distinction between them lies (to quote Dr. Christison's words) in the fact that 'the fits of natural tetanus are almost always slow in being formed, while nux vomica brings on perfect fits in an hour or less.' Further, tetanus rarely if ever 'proves so quickly fatal as the rapid cases of poisoning with nux vomica.' It need scarcely be added that the history and ætiology of all cases in which tetantic spasms are present should be investigated with minute care.

Morbid anatomy.—Various lesions have been discovered in the nervous system. In traumatic tetanus the nerves proceeding from the injured region have been found swollen, hyperæmic, and inflamed, either in part or in their whole length. In many cases, however, no such lesions have been perceived. It was formerly believed that the spinal cord was healthy; but recent investigations, and more especially those of Drs. Lockhart Clarke and Dickinson, have demonstrated the presence, in some cases at least, of considerable dilatation of the small vessels (particularly the arteries and veins) with accumulation of blood within them and around them, together with more or less abundant translucent or finely granular exudation, infiltrating the tissues; and tending to accumulate here and there, especially in the fissures, and occasionally on the surface of the cord. With these changes are associated sometimes more or less disintegration of the proper nervous elements, sometimes local effusions of blood. In trismus neonatorum congestion of the spinal arachnoid is described, with effusion of serum and even extravasation of blood, into the subarachnoid tissue. It can scarcely be admitted, however, that these lesions are proved to be invariably present in tetanus. That the motor nuclei of the spinal cord and medulla oblongata are generally in a state of polarity or abnormal irritability, or that they are generally under the influence of some abnormal condition which excites them to unwonted action, is clear enough. But whether this excited action is due to some peculiar change in the nerve-cells themselves, or to the influence exerted upon them by the congestion and effusion which surround them, or to the presence in the blood of some endopathic poison (as is suggested by Sir T. Watson and by Dr. Richardson) resembling strychnia in its effects, are points upon which as yet we can only speculate. It is uncertain, therefore, at present, whether the lesions which have been discovered in the spinal cord are in any degree the cause of the tetanic spasms,

or whether they are merely secondary to them. Ruptures of muscular fibres are frequently seen after death from tetanus. They are common in the muscles of the back, but sometimes occur in the abdominal muscles and those of the extremities.

Treatment.—No treatment, so far as we know, has any curative influence over tetanus. A certain number of cases get well under the most unfavourable circumstances; the great majority die in spite of the most strenuous efforts to save them. Innumerable drugs have been employed, and, according to their several advocates, with more or less success. Among those which have acquired the greatest reputation are opium, mercury, wourara, Calabar bean, and chloroform. Many other medicines, for the most part sedatives, have also been recommended, especially, perhaps, aconite, belladonna, digitalis, tobacco, hydrocyanic acid, chloral, and turpentine. By some authorities, drastic purgatives have been lauded, by some, profuse stimulation by means of ether or alcohol. It is important to know that tetanic patients can take large doses of the most powerful sedative medicines, and drink large quantities of alcoholic beverages without being brought under the influence of these agents. Warm baths, cold baths, ice to the spine, bleeding, division of the nerves leading to the injured spot, and even amputation of the limb or part on which the injury was inflicted, are measures which have each in turn been adopted and abandoned.

As regards general rules of treatment we cannot do better than quote Sir T. Watson's words. He says:—'Since any, the smallest, movement or impression made upon the surface, or upon the senses, will bring on the severer degrees of spasm, it is of primary importance to protect the patient against those sources of trouble, so sure to aggravate his sufferings, and so likely to augment his danger. Hence, if blood-letting should be thought advisable, it should be done early, sufficiently, and once for all.' 'The same remark applies to the frequent use of purgatives. The bowels should be well cleared in the onset, and then let alone. The patient should lie in a darkened room, from which noise also should, as much as possible, be excluded. He should not be surrounded by a multitude of friends or attendants. He should be enjoined to speak, to move, to swallow, as seldom as he can. In the severe traumatic cases, the nerve, in my judgment, should be promptly divided, and as high up in its course as may be practicable; and in all cases, there being no special indications to the contrary, I should be more inclined to administer wine in large doses, and nutriment, than any particular drug. If the tendency to mortal asthenia can be staved off, the disturbance of the excito-motory apparatus may perchance subside or pass away.' The patient's sufferings may often be alleviated by the use of opium or chloroform inhalation.

XVII. CONGESTION. ANÆMIA. SUNSTROKE.

A. *Congestion and Anæmia.*

Symptoms.—So many nervous phenomena are commonly referred to congestion or anæmia of the nervous centres, that we can scarcely presume to pass these conditions over in silence. And indeed, although we are disposed to assert that the great majority of cases in which symptoms are referred to them in practice are not true examples of anything of the kind, it must be freely admitted that congestion and anæmia of the brain and cord do really play an important part in the phenomena of disease in these organs. Whenever inflammation or other processes of proliferation are in progress congestion is necessarily present. We see the evidences of former congestion in the condition of the blood-vessels and of the parts immediately bounding them in chorea, epilepsy, tetanus, and chronic insanity. In heart-disease, in chronic bronchitis, in cases in which tumours press upon the large veins at the root of the neck, during violent muscular efforts, in the attacks of hooping-cough, the brain also becomes more or less congested. Anæmia may be the consequence of abundant losses of blood; it may be due also to obstruction of the common or internal carotid, or of one of the arteries distributed to the brain. But in the majority of the above cases, either the symptoms which the patient presents are not those commonly attributed to congestion or anæmia, or the symptoms referrible to these conditions are intermingled and confused with others dependent upon causes of a different kind.

We shall not discuss the various symptoms, which on theoretical grounds might be attributed to cerebral and spinal congestion. It will be sufficient for our purpose to point out: that they must necessarily differ materially according as the congestion is acute or chronic, and according as it involves certain regions of these organs or pervades them generally; and that abnormal congestion, if it be not excessive is likely to be attended with exaltation of function, if it be excessive is pretty certain to induce perversion or abeyance of function—vertigo, headache, delirium, convulsions, coma, paralysis, muscæ and dimness of sight, noises in the ears, and dulness of hearing. The effects of temporary congestion are sometimes well seen in attacks of spasmodic cough, such as attend pertussis, and the presence of thoracic aneurysms. The patient becomes more and more livid in the face, suffers from vertigo, headache, muscæ, and noises in the ears, and presently becomes momentarily insensible, with probably some convulsive twitchings or spasms of the muscles of the eyeballs and of those of expression.

The symptoms referrible to anæmia, equally with those due to congestion, vary according as the anæmia is general or partial, acute

or chronic, slight or extreme. Moreover, they are very much of the same kind as those which attend congestion. Thus, in cases in which the supply of blood to the brain is suddenly interrupted either by obstruction of one or more of the arteries supplying it, or by temporary arrest of the heart's action, or by copious loss of blood, insensibility and convulsions frequently ensue; and again, upon anæmia more slowly produced, it is not uncommon for delirium resembling that of chronic alcoholism, or for acute mania, or for apoplectic symptoms to supervene.

In many of the cases in which cerebral anæmia or congestion has been diagnosed during life, the condition of the brain appears post mortem to have been perfectly healthy; and in some there has been found, from no obvious cause, accumulation of fluid in the ventricles or subarachnoid tissue. Hence it is possible that, at any rate in some cases, the symptoms referred to congestion or anæmia may really have been immediately due to the presence of serous effusion. It is possible, also, that in some cases they may have been due to the effects of undetected poisonous matters in the blood. Without venturing to decide upon what conditions of the brain each of them depends, we shall proceed very briefly to discuss two or three so-called functional disorders of this organ, which appear to belong to the group of affections we are now considering.

1. *Delirium tremens.*—We have already (page 610) fully described this affection as it occurs in drunkards; but it occurs also, though much less frequently, independently of alcoholism, and indeed in persons of abstemious habits, sometimes as a consequence of severe injury (*delirium traumaticum*), sometimes as a result of long-continued mental anxiety. The symptoms and progress of the disease are identical in all these cases, and need not be again detailed.

2. *Insanity.*—Various forms of insanity, especially perhaps mania, melancholia, and dementia, are apt to come on during convalescence from acute febrile diseases, and after profuse hemorrhages. Their symptoms are in no way distinguishable from those of the same forms of insanity occurring under other conditions. They generally, however, end in more or less rapid recovery.

3. *Eclampsia.*—The convulsive attacks which may attend these conditions vary from mere momentary spasms of the muscles of the eyeballs or face, or some other limited part of the body, attended with momentary loss of consciousness, to epileptiform seizures of the most violent kind. And, indeed, there is nothing in the attacks themselves by which they are distinguishable from those of true epilepsy. The differential diagnosis must rest upon the associated phenomena of the case, and upon its history and progress.

4. *Apoplexy and paralysis.*—The apoplectic attacks which appear to be due to merely functional disturbance of the brain have in many respects a close resemblance to those which are the result of hemor-

rhage. Their onset is sometimes sudden; but it is more commonly gradual, the patient becoming drowsy, then semi-comatose, and finally, perhaps after remissions, passing into a state of complete stupor. When the apoplectic condition is fully established, the patient is absolutely insensible; his pupils are probably dilated and inactive to light; he has lost the power of deglutition in a greater or less degree; his breathing is probably explosive or stertorous; his arms and legs are motionless and flaccid; he has no control over his emunctories; and his urine is retained. The phenomena may, however, be as varied as those due to sanguineous effusion; and scarcely any of the symptoms which have been enumerated may not fail in certain cases, or be replaced by others. Especially it must be recollected: that partial or general convulsions may occur; that the limbs, instead of being flaccid, may be rigid; and, indeed, that there may be distinct hemiplegia. The main distinctions between functional apoplexy and that from effusion of blood reside in their usually different modes of onset; in the paralysis, which in functional apoplexy is mostly general and attended with flaccidity; in the pulse, which is usually accelerated in the affection now under consideration; and in the temperature, which here generally rises from the commencement of the attack, whereas in cerebral hemorrhage for some hours at least, it usually falls.

These comatose attacks are not limited to elderly persons; they are somewhat common in lateral and disseminated sclerosis; and affections not clearly distinguishable from them are occasionally observed in young children, in whom they simulate the phenomena of meningitis.

In connection with the apoplectic attacks, and occasionally independently of them, hemiplegic and other paralyses may occur. Affections of the sensory nerves also may supervene; and the patient may have dimness or loss of vision, or analogous conditions involving the sense of hearing.

It need scarcely, perhaps, be said: that in each of the above cases the progress of the affection may closely simulate that of the malady which in its symptomatic phenomena it most closely resembles; that in many instances complete recovery takes place within a shorter or longer time; that in some instances relapses occur after such recovery; that in some permanent mental defect or paralysis follows; and that not unfrequently death ensues.

Treatment.—It is impossible to lay down any definite rules for the treatment of these various functional disturbances. In the majority of cases the treatment should no doubt be the same as that for the affections which they resemble. It is important, however, to bear in mind that if they be traceable in any degree to loss of blood, to want of food, or to anæmia, however produced, it becomes essential to support the patient's strength by food and tonics.

B. *Sunstroke.* (*Coup de Soleil. Calenture. Insolatio.*)

Definition.—By the term 'sunstroke' is usually implied a more or less sudden attack of unconsciousness, occurring in persons exposed, under adverse conditions, to high temperature. It seems highly probable, however, that more than one affection is included under this name.

Causation.—Sunstroke appears to result from prolonged exposure to intense heat, especially if the atmosphere be at the same time damp and impure, and the patient exhausted by long-continued over-exertion, and the wearing of clothes and accoutrements which impede the free action of his respiratory muscles. It often occurs, especially in tropical climates, from exposure to the direct rays of the sun; but it is common also even in the night-time among persons who are subjected to intense heat in close, overcrowded, and ill-ventilated barracks, houses, or ships. Soldiers engaged in long and toilsome marches under the glare of a tropical sun are especially liable to suffer.

Symptoms and progress.—Sunstroke is sometimes sudden in its onset, but is more frequently preceded by premonitory symptoms. In the former case, the patient, who is probably engaged in some laborious occupation and exposed to the sun, suddenly falls down insensible and collapsed, with pale, cold, moist skin, gasping respiration, and extreme feebleness and rapidity of pulse. There is no doubt that, in these cases, equally with those next to be considered, the internal temperature at the time of the attack is above the normal. Death under these circumstances not unfrequently takes place with great rapidity, or even quite suddenly.

The premonitory or early symptoms of the other variety of the affection comprise, in a large proportion of cases, great sense of weariness and prostration, vertigo, nausea, dryness and heat of skin, tendency to frequent micturition or even incontinence of urine, and restlessness or sleeplessness. The actual attack is sometimes ushered in with drowsiness, and the patient lays himself down to rest or sleep; in other cases he is seized with momentary delirium or mania, more rarely with convulsions. Under any circumstances the patient rapidly becomes comatose or apoplectic, and then presents most of the ordinary phenomena of this condition. He lies perfectly insensible; his pupils are contracted and unaffected by light, his conjunctivæ injected; he breathes rapidly, noisily, and sometimes stertorously; his pulse is frequent, small, weak, and often irregular; his face is pallid; and his skin intensely hot. In some instances the patient remains perfectly quiet during his comatose condition; in others he is attacked with local or general convulsions of more or less severity. If death take place it is rarely delayed beyond twenty-four or forty-eight hours.

The mortality from sunstroke is very heavy, exceeding 40 per cent.

Recovery is sometimes sudden and complete; but it is more commonly slow, and attended for some few days by feebleness of the heart's action and oppressed breathing, and is then not unfrequently followed by consequences of more or less importance, such as headache, chorea-like affections of the muscles, epilepsy, and some degree of mental imbecility. These sequelæ may be permanent.

Morbid anatomy and pathology.—In most cases of fatal sunstroke the blood is found to have remained uncoagulated; the lungs are intensely congested, and the right side of the heart is loaded with blood; further, there is generally more or less engorgement of the vessels of the brain. The proximate cause of the disease is uncertain. By some it is considered that the symptoms are due to the circulation of poisonous matters in the blood. It is suggested by Dr. Geo. Johnson that the intense heat of the body is attended with dilatation of the pulmonary capillaries, engorgement of the lungs, and apnœa, upon which the other phenomena of the disease presently supervene. The first symptoms, however, in many cases, are those of unconsciousness, and in all coma rapidly comes on with involvement of those organs, especially, which are in relation with the pneumogastric nerves. The symptoms differ indeed little, if at all, from those of the typical apoplectic state; and it is difficult therefore to believe that they are not primarily cerebral.

Treatment.—Indian practitioners are unanimous as to the danger of bleeding in these cases, and are equally unanimous with respect to the value of the cold douche, or of cold applied in other forms. Especially it seems advisable to apply cold to the head. Subsequently, if consciousness do not return, the head may be shaven, and counter-irritants used. The bowels should be made to act, but not violently purged. Generally, also, it is better to give nourishment and diffusible stimulants than to deplete.

XVIII. MEGRIM. (*Migraine. Hemicrania. Sick-headache.*)

Definition.—A form of headache, for the most part circumscribed, coming on in paroxysms, and frequently attended with sickness, affection of sight, and other nervous phenomena.

Causation.—Megrim appears in a large number of cases to be an hereditary disease; and, when of distinctly hereditary origin, not unfrequently commences during the period of the second dentition, from which age up to thirty it usually first declares itself. It rarely commences after thirty, and generally, even in those who are liable to it, subsides with advance of years. Patients do not often suffer from it after fifty. Females are somewhat more prone to it than males. The determining causes of the attack are very various. Amongst the

most common of them are, disturbance of the digestive organs, such as may arise from over-feeding or prolonged abstinence, uterine disorders and the catamenial period, sustained mental labour or excitement, emotional disturbance, bodily fatigue or want of exercise, insufficient or over-abundant sleep, overcrowded rooms, foul air, and meteorological conditions; and, besides these, impressions upon the senses, such as are produced by glaring lights, rapid successions of objects presented to the eye, loud or discordant noises, strong odours, and offensive smells. Megrim, or a condition undistinguishable from it, may arise also in the course of an ordinary catarrh, or be induced by exposure of the head to a current of cold air, or by malaria.

Symptoms and progress.—In the simplest and most common form of megrim, the patient is attacked, more or less suddenly, with dull pain, usually referred to a limited surface immediately over the eye or in the temple. This gradually extends in area, and becomes more intense, but usually still remains limited to one side of the head. It varies in intensity from time to time, is for the most part aching, but is not unfrequently attended with sudden shootings, and generally with throbbing, which is always greatly increased by bodily or mental exertion. It often involves the eye; and this, together with the scalp, is apt to become more or less hyperæsthetic or tender. The head is generally hot, and the arteries of the affected region manifestly dilated. In many cases the pain affects both sides of the head, although even then it is usually more intense on one side than the other; occasionally it attacks the occipital instead of the frontal region; and sometimes it becomes generally diffused. From the commencement the patient is dull and indisposed for or incapable of mental or bodily exertion, and with the continuance of the headache these conditions increase upon him, and he becomes pale and chilly, and looks heavy, dejected, and miserable. Not unfrequently he has a vague dread, or sense of impending evil; and especially he is apt to experience a general feeling of profound illness, attended with tremulousness, shivering, and weakness of the limbs. The pulse for the most part is small and weak, and often slower than natural. After a variable time a sense of nausea supervenes, and in a large proportion of cases culminates in more or less severe vomiting, during the attacks of which the headache generally attains its greatest degree of intensity. After the vomiting has ceased the patient probably goes to sleep, and at the end of some hours awakes in pretty nearly his usual condition of health.

In a large number of cases other symptoms are associated with those just considered, and then for the most part precede them. Among the most interesting of them are disorders of vision. When these occur, they are probably always the earliest in the sequence of events; and they generally vanish with the supervention of headache. They vary in character:—in some cases certain portions of the retinæ become simply insensible, and if the central spots of the eyes be

involved, the patient, who probably feels well in all other respects, notices that he cannot see the nib of the pen with which he is writing, or the letters which he is forming, or that, while distinguishing all other parts of the body, he cannot see the face of the person at whom he is looking; in some cases he observes a tremulous vibratile, or rotatory movement in some part or other of the field of vision; sometimes these tremulous areæ or spectra become variously coloured; double vision sometimes occurs. The patches of retinal anæsthesia, or derangement, appear always to occupy identical parts in both eyes, and are apt to vary in shape from time to time. The colouration of the spectra, when it occurs, is always secondary.

Other senses are apt to suffer, but on the whole much less frequently than sight; and the phenomena referrible to them, when associated with ocular derangements, always come on later. Occasionally the patient experiences deafness or noises in the ears or loss or perversion of taste or smell; but more frequently, perhaps, he is attacked with numbness, passing on may-be to complete anæsthesia, of the upper extremity, of the half of the head and neck and face, and even of the parts within the mouth, all on the same side as the cephalic pain. This loss of sensation is sometimes associated with more or less complete muscular paralysis. Further, the cephalalgia is not unfrequently associated with the presence of distinct neuralgic pains, not only at the back of the head, but in the back and side of the neck, and even in the shoulder and down the arm.

It has already been observed that the patient becomes more or less dull and apathetic and incapable of intellectual exertion; but psychological phenomena of a more remarkable kind are apt to ensue. Sometimes there is marked mental confusion or incoherence of thought, sometimes indeed typical aphasia. Drowsiness is very common: often forming one of the earliest indications of the attack; sometimes coming on during its progress, and culminating in a more or less prolonged semi-comatose condition; but much more frequently constituting the termination of the attack.

Megrim begins in many different ways. In some cases it supervenes immediately upon exposure to its exciting cause, as when the characteristic headache attacks the sufferer while he is at the theatre, or at a picture gallery, or during exposure to discordant noises or offensive smells, or while he is engaged in some laborious mental occupation. Not unfrequently it comes on during the night, the patient waking from time to time with the consciousness of heaviness in the head, and getting up with the attack well-developed; or it manifests itself when he wakes in the morning, or immediately after rising. In other instances it comes on at various times of the day, without obvious immediate cause. In a large proportion of cases the headache is the first symptom. In many, however, this is preceded by some of the prodromal phenomena above considered—the patient ex-

periences a sense of general illness or of depression or dread; or he has numbness, or confusion of thought, or extreme drowsiness, or other of the psychical phenomena which have been enumerated; or he has some affection of vision. It is curious, as we have already shown, that when these several phenomena, or any of them, manifest themselves, they almost invariably precede the headache. It must be added that the attack may be limited to any one of these symptoms.

The duration of megrim varies for the most part between twelve and twenty-four hours; it may, however, last an hour or two only; and it may be prolonged, but generally by successive relapses, for several days. The subsidence of the attack is generally gradual; in a large number of cases it is preceded by vomiting; and in the great majority (whether vomiting have taken place or not) the patient after a while sinks into a profound sleep, from which he wakes refreshed, and probably well. The patient during convalescence often perspires profusely, and excretes a large quantity of urine.

Megrim is essentially a periodical disease, and in those who are liable to it not unfrequently comes on with more or less regularity once a week, once a fortnight, or once a month. It not uncommonly, however, occurs less regularly, and more distinctly in response to certain definite causes to which the patient exposes himself. Moreover, it often ceases for a time under various circumstances, such as pregnancy, suckling, and change of air or occupation, and occasionally is developed only at long intervals in connection with special causes of ill-health.

Pathology.—Megrim has often been regarded as essentially a symptom of disorder of the liver or stomach: and no doubt affections of these viscera, but more particularly of the stomach, must be regarded as some of its exciting causes. But, on the other hand, the attacks so frequently arise independently of any morbid condition of the digestive organs that we are compelled to look elsewhere for its essential seat. It has latterly been referred to disorder of the cerebral circulation due to the influence of the vaso-motor nerves on the vessels of the part supposed to be implicated. Dr. Latham believes that its primary cause is some affection of the vaso-motor nerves, in virtue of which the vessels become contracted and the tissues anæmic; and that it is to anæmia thus produced of the central nervous organs that the defects of vision and other early phenomena are due. And he refers the headache, which is generally if not always attended with manifest dilatation and throbbing of the temporal arteries, to secondary hyperæmia. Dr. Liveing, however, points out that even if, as seems not improbable, some of the phenomena are referrible to anæmia and hyperæmia, there must still be some antecedent cause to which the vaso-motor affection itself is due. And he contends that the phenomena of the disease depend on the irregular accumulation and discharge of nerve-force; that the immediate antecedent of the attack is a condition of unstable

equilibrium, and gradually accumulating tension in the parts of the nervous system more particularly concerned; and that the paroxysm itself may be likened to a storm. He regards the optic thalami and all those parts which lie between these bodies and the roots of the vagi as the seat of disease. Megrim would seem, according to this hypothesis, to have a close pathological relation with epilepsy. The diseases do not, however, pass into one another.

Treatment.—In the first place it is important for the patient to avoid those conditions to which his attacks seem to be traceable; it is especially important also for him to live wholesomely, to avoid gastro-intestinal disturbance, to take plenty of exercise, to inhale the fresh healthy air of the country, and to refrain from too prolonged or intense mental labour, worry, or excitement. Various remedies are employed, reputedly with more or less success, to prevent the occurrence of attacks of megrim; among others, iron, zinc, arsenic, iodide, bromide and chloride of potassium, quinine, strychnia, belladonna, hyoscyamus, and valerian. During the paroxysms, nothing is so efficacious as complete rest in the recumbent posture, in a darkened and perfectly quiet room. Relief may often be afforded, however, by the administration of strong tea or coffee, or of caffeine, theine, guarana or croton chloral. Occasionally a full dose of brandy, ammonia, or one of the fixed alkalies benefits the patient. Evaporating lotions, belladonna, or aconite locally applied are often of great service. The aconitia ointment is especially valuable in many cases in warding off or subduing the headache. But local measures, although they relieve pain, do not prevent or curtail the other phenomena of the attack. The headache may sometimes also be soothed by pressure upon the carotid or temporal artery of the affected side.

XIX. MÉNIÈRE'S DISEASE. (*Aural Vertigo.*)

Definition.—A disease characterised by sudden attacks of vertigo in connection with lesions of the semicircular canals.

Causation and pathology.—Experiments performed on the lower animals, in the first instance by Flourens and subsequently by other observers, have distinctly proved that injury of the semicircular canals is followed by vertiginous movements, which have some definite relation to the particular canal operated upon. To this subject attention has already been directed. It was M. Ménière, however, who first, in the year 1861, recognised and described the phenomena due to disease of these organs in the human being.

Many cases have now been recorded in which the group of symptoms presently to be considered has been found associated with more

or less impairment of hearing; and in several of them post-mortem examination has revealed the presence of inflammatory exudation strictly limited to the semicircular canals. There is good reason, however, to believe that similar effects may be produced indirectly in diseases of the middle ear or other neighbouring parts, by the pressure which is apt to be exerted upon the contents of the labyrinth, and through them on the semicircular canals. Hence they may result from catarrh, and complicate ordinary forms of otitis.

Symptoms and progress.—The specific phenomena of Ménière's disease are sometimes preceded by deafness, earache, and other indications of aural mischief. But in a considerable number of cases they manifest themselves without any such prodromata. The patient is suddenly attacked with noises in his ears, or in one of his ears, and a feeling of vertigo—symptoms which are attended with faintness, pallor, perspiration, nausea, and probably actual vomiting. The attack is of short duration, sometimes lasting two or three seconds only; and usually the recovery of the patient from it is for a time complete.

The noise, as above stated, is sometimes referred to one ear only, sometimes to both; but in the latter case it is generally more pronounced on one side than the other. It differs in intensity in different cases, and is variously described as buzzing, humming, whistling, or singing, and is likened sometimes to the puffing of a steam-engine, sometimes to a sudden explosion. The sense of vertigo varies in severity and duration: in some cases there is a mere momentary feeling of giddiness, or a feebler but more prolonged sensation of swimming in the head which resembles that attending sea-sickness; in some cases the patient feels as if he were suddenly thrown forwards, backwards or laterally, or rotated, and he staggers or falls in the direction which corresponds with his sensation, clutching at neighbouring objects for support, or actually falling to the ground. The attack is always attended with a sense of anxiety or alarm and more or less faintness. The latter condition may reveal itself by a momentary pallor, præcordial anxiety, and failure of cardiac action, or by all the ordinary signs of well-marked syncope, followed by sweating, and extreme rapidity and feebleness of pulse. A feeling of nausea is probably always present in a greater or less degree; but not unfrequently vomiting ensues, and with its occurrence the attack usually comes to a close.

The vertiginous seizure, however severe it may be, is never attended with actual loss of consciousness; and there is never any convulsive movement, paralysis, implication of speech, squinting, sensation which can be likened to the epileptic aura, or indeed any phenomena, beyond those above described as constituting the attack, which in any sense point to the presence of cerebral mischief. Headache even is unfrequent.

The fits come on in the first instance at irregular and probably

distant intervals; but they tend gradually to increase in frequency and to approach one another; and sooner or later, probably, a time arrives when the patient, though still suffering from more or less frequent paroxysms, is never free from some degree of vertigo.

The noises in the ear which attend the early attacks of vertigo are not necessarily associated with deafness; indeed, sometimes hearing is preternaturally acute, and discordant or loud noises are peculiarly painful to the patient. Occasionally the range of audition becomes contracted. At this time also it not unfrequently happens that no affection of hearing is observable between the attacks. But by degrees the noises in the ear become constant, though still undergoing exacerbation when the paroxysms occur; and the sense of hearing grows gradually more obtuse, until ultimately absolute deafness of the affected ear probably ensues. It is a curious fact that, with the supervention of absolute deafness, not only do the paroxysmal attacks generally cease, but with them the continuous sense of giddiness which had probably also been present.

Slight and momentary attacks of giddiness, essentially resembling those above described, are by no means uncommon in connection with various temporary or permanent affections of the middle ear, and are then frequently induced immediately by loud or discordant noises, or by other powerful impressions on the senses. In such cases, however, complete recovery may be anticipated, and the disease seldom takes the course which has been above sketched.

Treatment.—It is impossible to lay down rules for the treatment of Ménière's disease. It is important, however, to bear in mind that the vertiginous attacks are relieved though not prevented by lying down, and in many cases by carefully protecting the patient from all noises and other such influences. Further, it is obvious that when the symptoms depend on the presence of disease of the accessible parts of the ear, treatment directed to these parts should be employed.

XX. LOCAL PARALYSES.

A. *Paralysis of the Third, Fourth, and Sixth, or Oculo-motor Nerves.*

Causation.—Paralysis of these nerves is probably always the consequence of some lesion involving them either at their origin or in some part of their course. Among such lesions may be named syphilitic disease of the base of the skull or contiguous parts of the brain; tubercle, carcinoma, or other forms of morbid growth, or inflammatory exudation, occupying the same situations; tumours or accumulations of fluid or blood situated in the superincumbent brain-substance,

and causing pressure; and aneurysms or tumours in the course of the cavernous sinus. Oculo-motor palsy is common in locomotor ataxy.

Symptoms and diagnosis.—In order to determine the situation of lesions causing the various oculo-motor paralyses, and to distinguish the paralyses due to individual nerves, it is essential, on the one hand, to have a clear view of the relations of the oculo-motor nerves at their origin and in the various parts of their course; and, on the other hand, to have an exact knowledge of the normal actions of the muscles which they supply.

In reference to the anatomical point, it must be borne in mind: that the nuclei of the third and fourth nerves are in close relation with one another in the floor of the iter, and that the sixth arises, in common with the facial, in the floor of the fourth ventricle; that the muscles supplied by these nerves are seven in number, namely, the

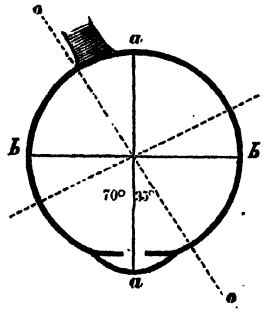


FIG. 12. Horizontal section of left eye seen from above.

a a, Antero-posterior diameter of eye; *b b*, transverse diameter of eye; *r r*, fixed axis of rotation for movements effected by superior and inferior recti; *o o*, fixed axis of rotation for movements effected by obliqui.

levator palpebræ superioris, the four recti, and the two obliqui; and that of these the external rectus is supplied by the sixth alone, the superior oblique by the fourth alone, and all the other muscles by branches of the third.

The muscles of the eyeball are six in number, and arranged in antagonistic pairs, which severally rotate the eyeball in opposite directions upon an axis perpendicular to the plane in which they respectively act, and passing as nearly as possible through the central point of the globe of the eye. These several pairs are the internal and external recti, the superior and inferior recti, and the superior and inferior obliqui. And the axes of rotation of the eyeball which correspond to them (see fig. 12) are (supposing the left eye to be under consideration, and its line of vision to be fixed directly forwards):—for the external and internal recti, a vertical straight line passing through the centre of the eye; for the superior and inferior recti, a horizontal

straight line (*r r*) passing obliquely through the centre so that its nasal extremity is a little in advance of its temporal extremity, and forming an angle of about 70° with the line of vision; for the obliqui, another horizontal straight line (*o o*) passing also obliquely through the centre, but in such a direction that it makes an angle of 35° only with the line of vision—its temporal extremity being just a little beyond the outer margin of the cornea, and its nasal extremity towards the back of the eye, a little internal to the optic disc.

It may be assumed as sufficiently accurate for all practical purposes: that the ball of the eye is globular; that it is lodged in a socket bounded by fat, connective tissue, and membrane, in which it moves as the head of the femur moves in the glenoid cavity; that its centre of rotation is the actual centre of the eye; and further, as Helmholtz shows, that in consequence of the fixed origins of the oculo-motor

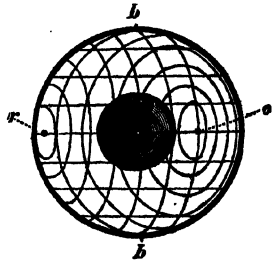


FIG. 13. Left eyeball with iris and pupil seen from the front.

o, Anterior pole of axis of rotation for obliqui; *r*, anterior pole of axis of rotation for superior and inferior recti; *b b*, upper and lower poles of axis of rotation for internal and external recti.

The concentric circles around *o* and *r* respectively indicate the direction and amount of movement of the different parts of the globe due to the action of the obliqui and superior and inferior recti respectively; the horizontal lines have a similar relation to the movements about the axis *b b*.

muscles, and their broad insertions, the three axes of rotation which have been indicated remain unalterable in their relation to the fixed points of the orbit, no matter how much the line of vision—the line in which the eye is looking—becomes altered.

It follows (see fig. 13) from the above considerations:— first, that the internal and external recti always cause the cornea to revolve around a vertical axis, to move therefore either in a horizontal equatorial line, or in proportion as it is elevated or depressed below this line in the arcs of smaller and smaller parallel circles; second, that the superior and inferior recti always cause the cornea to revolve around the oblique horizontal axis, whose position has already been defined, and hence in the arcs of circles which are parallel to a vertical equatorial section of the eyeball, made through or near the outer margin of the cornea (as the eye looks directly forwards) in front, and the inner

margin of the optic disc behind, so that the circles to which these arcs belong become smaller and smaller from the outer to the inner canthus of the eye, and the influence of the recti in causing vertical movements of the cornea correspondingly diminished; third, that the obliqui always cause the cornea to revolve around the oblique horizontal axis which has been referred to these muscles, and hence in the arcs of circles which are parallel to a vertical equatorial section of the eyeball, made through or near the inner canthus; the circles, to which these arcs correspond, commencing in a point a little outside the outer margin of the cornea, become larger and larger towards the inner angle of the eye, so that the influence of the oblique muscles upon the cornea varies from the production of simple rotation, when the eye looks outwards, to equatorial amplitude of movement when the eye is directed towards the inner canthus. It follows, further, that while all horizontal consensual movements of the two eyes can be effected by means of the inner and outer recti only, all vertical consensual movements require the co-operation of the superior and inferior recti and obliqui.

Paralytic affections of the muscles of the eyeball are attended for the most part with squinting and double vision—the direction and character of the squint being different for each muscle affected, and the position of the object, as seen by the squinting eye in relation to that seen by the normal eye, being either internal or external to it, above or below it, or tilted. The existence of double vision is sometimes not recognised by the patient when the axes of his eyes diverge very widely; moreover, the double image tends to merge into one, and the squint to become unapparent, in proportion as the patient turns his sound eye in the direction towards which the squinting eye inclines.

In testing the eyes with the object of discovering the existence of double vision and the peculiarities it may present, it is generally convenient to place a coloured glass before one of them, in order that the patient may be able to distinguish and indicate by their respective colours the two images which he sees.

Assuming the left eye to be affected, the following would seem to be the consequences of paralysis of its several muscles taken singly:—

Internal rectus.—Outward squint. Displacement of false image in horizontal line, to patient's right. Image not tilted. (See fig. 14.)

External rectus.—Inward squint. Displacement of false image in horizontal line, to patient's left. Image not tilted. (See fig. 15.)

Superior rectus.—Downward squint. Displacement of false image upwards. Elevation of pupil above horizontal line effected solely by inferior oblique; its upward movement, therefore, is in a curve directed upwards and to the left; it is ample when the pupil is directed inwards, but when the pupil is directed outwards, consists in a mere rotation of it upon its axis. Under these circumstances, the false image is tilted to the patient's right. (See fig. 16.)

Inferior oblique.—Downward squint. Displacement of false image upwards. Elevation of pupil above horizontal line effected by superior rectus; its upward movement, therefore, is in a curve, directed upwards and to the right; it is most ample when the pupil is directed outwards. The false image, when the pupil is above the horizontal line, is tilted to the patient's left. (See fig. 17.)

Inferior rectus.—Upward squint. Displacement of false image downwards. Depression of pupil below horizontal line, accomplished by superior oblique. Its downward movement consequently is in a

FIG. 14.

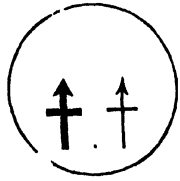


FIG. 15.

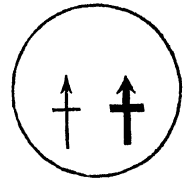


FIG. 16.

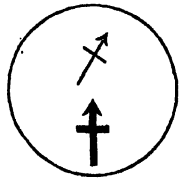


FIG. 17.

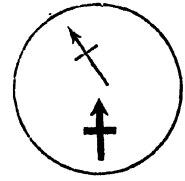


FIG. 18.

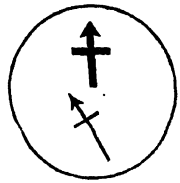
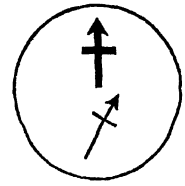


FIG. 19.



In the above diagrams the thick cross represents the *true image*, the thin cross the *false image*.

curved line, directed downwards and to the left, being a mere revolution upon its axis when the eye is directed outwards, but becoming more and more ample as the eye turns to the right. False image, when patient is looking downwards, tilted to his left. (See fig. 18.)

Superior oblique.—Upward squint. Displacement of false image downwards. Depression of pupil below horizontal line, effected by inferior rectus. Its downward movement takes place, therefore, in a curved line directed downwards and to the right, the movement being most ample when the eye is directed outwards, least ample when it

looks inwards. When the pupil is below the horizontal line, the false image is tilted to the patient's right. (See fig. 19.)

It does not generally happen, however, that one muscle only is affected, unless it be the external rectus, or that when two or three muscles are involved they are completely paralysed; and consequently it is often difficult to determine the respective shares which different muscles take in oculo-motor paralysis. Paralysis of the third pair very frequently causes ptosis, together with dilatation and immobility of the pupil. When the oculo-motor branches of the third nerve are all implicated, the eye assumes an external squint, and the pupil is commonly directed a little downwards. Paralysis of the sixth pair is not unfrequently bilateral, probably because, from the long course which these nerves take along the floor of the skull, and from their position between this and the pons, they are peculiarly exposed to pressure and involvement in intra-cranial inflammatory and other morbid processes.

Treatment.—The treatment of paralysis of the muscles of the eye must be determined by the opinion we form of the nature of the lesion causing it. Iodide of potassium and mercury are indicated in syphilitic cases. Electricity, applied to the muscles, is sometimes serviceable. However it may be explained, patients often recover from these paralyses.

B. *Paralysis of the Fifth Nerve.*

Causation.—This, like paralysis of the oculo-motor nerves, may be caused by various lesions implicating the nucleus of the nerve or the nerve itself in some part of its course. It is most commonly of syphilitic origin.

Symptoms and diagnosis.—The nerve may be implicated wholly or in part, and in the latter case the affection may be limited either to its ophthalmic, to its superior maxillary, or to its inferior maxillary division. When the affection is total, there is complete anæsthesia of all those parts to which the nerve is distributed, and at the same time complete paralysis of all the muscles of mastication which it supplies. The loss of sensation involves the anterior half of one side of the head and face as far back as the ear, inclusive of the conjunctiva, cornea, and eyelids, the mucous membrane of the nose, lips, cheek, gums, and palate, the tongue (excepting in the neighbourhood of the circumvallate papillæ), and the external auditory meatus. Consequently the conjunctiva is insensible to all external impressions, and liable, therefore, to get irritated and inflamed; its irritation, moreover, is unattended with reflex phenomena; the sense of smell is impaired, mainly from tendency of the Schneiderian membrane to inflame, but partly from loss of power to appreciate impressions made by pungent vapours or gases; the sense of taste is annulled in the anterior two-thirds of the tongue; and,

partly from the loss of ordinary sensation in one half of the oral mucous membrane, mastication becomes difficult on the corresponding side, and food tends to collect unknown to the patient between the cheek and gums, or between these and the tongue.

The chief muscles supplied by the motor portion of the nerve are the temporal, masseter, and pterygoids. The temporal closes the jaw, and at the same time draws it more or less backwards; the masseter and internal pterygoid also close the jaw, but tend to draw it forwards; the external pterygoid co-operates to some extent with the last, but is especially the muscle by which the jaw is thrown forwards. Collectively they close the jaw, and effect the various horizontal movements of trituration. If they be paralysed, neither the temporal nor the masseter can be felt to harden in contraction as do those of the healthy side, when the teeth are being firmly closed; when the lower jaw is retracted it is drawn back obliquely with an inclination to the healthy side; when it is protruded, it is protruded obliquely with an inclination to the paralysed side. The last deformity becomes especially remarkable when the patient opens his mouth wide, for not only is the jaw then displaced in a very high degree towards the paralysed side, but the oral orifice becomes lopsided, and the muscles connected with the affected side of the lower lip appear to act more powerfully than their fellows. The difficulty of masticating on the diseased side is necessarily largely dependent on the weakness of its muscles.

We have referred to the tendency which the affected side has to get congested and inflamed. These conditions may supervene in the mucous membrane of the mouth or nose, or in the conjunctiva. It is most commonly observed, however, in the cornea, which is apt within a few days of the occurrence of paralysis to become opaque, to ulcerate, and to slough. These nutritive lesions doubtless obey the law which has previously been considered; that is, they occur not so much when the continuity of the nerve is absolutely destroyed as when the implication of the nerve or of its nucleus is incomplete and irritative. Under the same circumstances, loss of faradic contractility of the paralysed muscles and wasting are likely to come on quickly.

If the affection of the fifth nerve involve only one of its main branches, the paralytic symptoms will of course be limited to the distribution of that branch. Accordingly, if the ophthalmic division be alone affected, the anæsthesia will occupy the front of the forehead, the upper eyelid, the conjunctiva, and a part of the mucous membrane and integument of the front of the nose; if the superior maxillary branch, the anæsthesia will involve the cheek, the lower eyelid, and upper lip, together with the side of the nose, a portion of the temple, the interior of the nose, and the mucous membrane of the cheek, upper gums, and palate; if the inferior maxillary, the lateral part of the head and face, with the ear, the lower lip and gums, the tongue,

and the muscles of mastication; if the motor portion, the muscles of mastication only.

Treatment.—If the disease be syphilitic, antisyphilitic remedies are indicated. But in other varieties of organic disease medicinal treatment can have but little influence. If the affection be hysterical, or due to inflammation or other removable causes, faradism or galvanism may be serviceable in hastening the restoration both of sensation and of voluntary movement.

C. *Paralysis of the Portio Dura. (Bell's Paralysis.)*

Causation.—This may be caused by any lesion implicating the nucleus of the seventh nerve in the floor of the fourth ventricle, or involving the nerve in any part of its course thence—either as it passes through the substance of the pons, or between its apparent origin and the internal auditory meatus, or in its passage along the aqueduct of Fallopius, or just after its emergence from the petrous bone. It may be caused: within the skull, by either extravasation of blood, inflammatory mischief, or syphilitic or other growths; in its passage through the bone, either by fracture, or by morbid growths originating in the bone-substance, or in connection with caries of the internal or middle ear, or in consequence of inflammation attacking the nerve itself. Externally to the petrous bone, it may be due to injury, as sometimes happens to the infant during delivery by the forceps, or to inflammatory or other lesions of the parotid or other tissues in the vicinity. The most common and interesting cause of Bell's paralysis is inflammation of the nerve within the aqueductus Fallopii—a condition which is readily induced by allowing a draught of cold air to play on the side of the face. Hence not uncommonly it is traceable to a railway journey in which the patient has been sitting facing the engine and next an open window, or results from exposure of one side of the face while sleeping to a current of air.

Symptoms and diagnosis.—Inflammatory, or so-called 'rheumatic' paralysis, is generally of rapid development, and is not necessarily attended with pain or constitutional disturbance. Yet not unfrequently the draught which caused the paralysis causes also earache, or neuralgic phenomena referrible to the fifth nerve. The symptoms of Bell's palsy are very striking: all the muscles supplied by the seventh of one side are more or less completely paralysed; the half of the face, consequently, is without motion and expressionless; the wrinkles are smoothed away; and the predominant action of the opposite muscles draws the mouth more or less powerfully over to that side. When the patient wrinkles his forehead in surprise, the healthy half becomes, through the agency of the occipito-frontalis, transversely furrowed; when he frowns, the corrugator supercilii contracts the same part into vertical folds; but in both cases the forehead on the affected side

remains perfectly smooth. The orbicularis palpebrarum ceases to act, so that the eye remains permanently open, and the conjunctiva, from the loss of its habitual protection, becomes watery and inflamed. M. Duchenne points out that this condition is sometimes associated with epiphora, and he attributes this circumstance to the fact that the tensor tarsi is then paralysed as well as the orbicularis itself, and consequently fails to retain the puncta in the position best adapted for carrying off the lachrymal secretion. The ala of the nose gets flaccid, and the corresponding nostril loses its rotundity. The cheek is motionless, and smooth and limp; the natural furrow beneath the eye and that descending from the side of the nose become indistinct; and when the patient coughs or blows through his mouth, the cheek, owing to paralysis of the buccinator, undergoes momentary distension. The mouth is drawn to the opposite side; when it is shut, the paralysed half closes less perfectly than the other; when it is opened, that half opens less completely; and the more powerfully he exercises his facial muscles, as in laughing and crying, the more extreme does its lateral distortion become. He has lost the power of whistling, and probably that of blowing out a candle; his utterance is somewhat impaired; he has difficulty in retaining fluids in his mouth, especially in the act of drinking; and food tends to collect between the teeth and the paralysed buccinator. There is no anæsthesia.

There are some important distinctions between paralysis due to direct implication of the portio dura, and paralysis of the same nerve of hemiplegic origin. In the first place, in hemiplegia the paralysis rarely involves materially those branches of the portio dura which are distributed to the eyelids and upper half of the face, while in primary affection of the nerve the paralysis is general. In the second place, in hemiplegia not only is there more or less general unilateral palsy, but the motor branch of the fifth pair and the hypoglossal are generally involved together with the facial; while, in the other case, the temporal, masseter, and pterygoids still act perfectly, and the movements of the tongue are in no degree compromised. Lastly, in hemiplegia the facial paralysis is rarely absolute even in the parts chiefly affected, and the paralysed muscles retain their bulk and faradic contractility, while in Bell's paralysis the loss of power is usually absolute, and the muscles lose their electrical contractility very rapidly, sometimes in less than a week. In neither case are the muscles of the eyeball and the levator palpebræ implicated.

The phenomena above enumerated are those which most commonly attend Bell's paralysis, and are the only ones which attend it when the lesion causing it is situated below the junction of the portio dura with the Vidian nerve; but other phenomena are apt to be superadded in proportion as the disease causing paralysis approaches nearer and nearer to the origin of the nerve. If the disease be so situated as to involve the chorda tympani and petrosal nerves, the patient is likely to

suffer: first, from more or less over-acuteness or painfulness of hearing, which has been attributed to relaxation of the membrana tympani from paralysis of the tensor, but is by Brown-Séquard believed to be due to hyperæsthesia of the auditory nerve dependent on involvement of the sympathetic branch supplying its blood-vessels; second, from dryness of the half of the tongue corresponding to the paralysed half of the face, and some impairment of taste, owing to implication of the chorda tympani and consequent interference with the salivary secretion, and to some obscure influence exerted directly on the tongue; and, third, from paralysis of the corresponding half of the soft palate, resulting from implication of the petrosal nerves. This paralysis is revealed: partly by the fact that from involvement of the corresponding half of the azygos uvulæ, the uvula when at rest, and still more when in motion, is so curved that its point is directed away from the paralysed side; and partly by the condition of the corresponding arch of the fauces, which is usually a little lower than its fellow, and owing to the tonic action of the muscles on the healthy side is drawn over in that direction.

When Bell's paralysis is due to disease situated within the skull, various other complications are liable to be associated with it; and our diagnosis of the seat of the disease will be mainly determined by the nature of these complications. Thus if the disease be within or near the internal meatus, the auditory nerve is likely to be involved and deafness to be produced; if it implicate the common nucleus of the sixth and portio dura, paralysis of the external rectus will go along with the facial palsy, and if it be irregular in distribution, or involve any considerable space, various other paralyses, referrible to implication of the nerves originating in the floor of the fourth ventricle, are liable to be present.

Occasionally both facial nerves are simultaneously affected, or affected within a short time of one another—a condition which may involve some difficulty of diagnosis.

The prognosis of paralysis of the portio dura will depend on the nature of the lesion to which it is due. That form of the disease which results from exposure to cold for the most part ends favourably, sometimes in a week or two, more frequently after four or five weeks, occasionally only after the lapse of some months. An element in the prognosis is the condition of the paralysed muscles as to faradic contractility; the more completely this has become annulled, the longer will recovery be delayed, and the greater is the fear that the paralysis may be incurable. M. Duchenne draws attention to the fact that not unfrequently permanent contraction of the muscles previously paralysed takes place, and that thus consecutive deformities are induced. This happens he says in those cases especially in which, either spasms supervene in the paralysed muscles under the influence of faradism or other forms of excitation, or a rapid return of tonic force takes place in muscles remaining paralysed and irresponsive to faradism. The

contraction affects sometimes one, sometimes several muscles. When it involves the lesser zygomatic it curves and deepens the naso-labial line and gives an expression of chagrin; when the greater zygomatic, it elevates the commissure of the mouth and imparts an aspect of gaiety; when the quadratus menti, it depresses and everts the lip; when the orbicularis palpebrarum, it causes diminution of the palpebral aperture; if all the muscles are involved, the side of the face becomes wrinkled, as if by cold. At the same time that the muscles contract, or it may be subsequently, they usually recover their voluntary power; but that is not always the case, and the contracted muscles may remain permanently paralysed.

Treatment.—In the treatment of paralysis of the seventh pair from cold, it is generally well (considering the serious results of permanent deformity) at once to adopt active measures; to apply a few leeches to the mastoid process, and to follow them up by fomentations, poultices, or equivalent applications. Subsequently blisters or other counter-irritants may be resorted to. If recovery do not follow these measures, electricity should be employed. Duchenne thinks it best in cases where the faradic contractility has wholly disappeared, to delay the use of faradism until after the lapse of two or three weeks. He recommends the employment of a current with rapid intermissions, and that the muscles should be directly and in turn excited. He points out that under this treatment the paralysed muscles often regain their tonic power, and the face its symmetry in repose, two or three weeks or more before there is any indication of the return of voluntary power; and that it is usually in the zygomaticus major that this power first returns—a fact which may be ascertained by making the patient smile. When the muscles begin to contract, he recommends that the intermissions should be few and the sittings short and unfrequent; and especially he recommends this, if any of the precursory signs of permanent contraction manifest themselves, in order that such contraction may be prevented. Galvanism also is efficacious in restoring the paralysed muscles.

D. *Paralysis of the Spinal Nerves.*

Causation.—Paralysis of these nerves may arise under various conditions; but we propose to refer only to those varieties which M. Duchenne speaks of as paralyses from cold, and in which the paralysis is due to inflammation of the trunk of the affected nerve. These affections are not uncommon, and may be readily mistaken for ordinary rheumatism.

Symptoms and diagnosis.—The symptoms comprise pain and tenderness along the affected nerve and febrile disturbance, together with the various consequences of disease involving mixed nerves: namely, on the one hand, burning or shooting pains in the course of its branches

and hyperæsthesia followed by tingling and numbness; on the other hand, muscular paralysis, followed by speedy loss of faradic contractility and wasting. The muscular paralysis for the most part comes on later than the sensory symptoms. During the early period of the disease, the temperature of the affected parts is augmented, later on it undergoes manifest diminution.

M. Duchenne singles out two forms of this affection for description, one of which he terms 'deltoid rheumatism,' the other 'paralysis of the radial nerve.' Affection of the spinal accessory is also not uncommon.

1. *Deltoid rheumatism* is essentially inflammation of the circumflex nerve. It is marked by the occurrence of violent neuralgic pains in the deltoid muscle, sometimes coming on in paroxysms, and augmented by any movement of the shoulder. In voluntary movements pain is especially excited in those fibres which are brought into contraction—a circumstance which will help to distinguish deltoid rheumatism from ordinary articular rheumatism. The symptoms may last for a few days only, or be prolonged for months. In many cases convalescence takes place without the occurrence of complications; but in some cases, atrophy of the deltoid, or of a part of it, supervenes after the pains have continued for some time; and when at length, under these circumstances, the pains have subsided, the muscle continues atrophic, although retaining its voluntary and electric contractility. In other cases paralysis, attended with more or less complete abolition of faradic contractility, supervenes.

2. *Paralysis of the radial or musculo-spiral nerve* is sometimes referred to pressure on the nerve, occurring, for example, during sleep, but by M. Duchenne is attributed (like Bell's paralysis) to exposure to cold, especially to exposure of the arm during sleep to a current of cold air, or to cold and damp. It generally comes on suddenly, without pain or tenderness, but with numbness and tingling, extending to the tips of the fingers. The paralytic symptoms have a close resemblance to those of lead-poisoning, and, like these, comprise, as an essential feature, dropping of the hand, and incapability of extending the fingers. The differences between them are, as M. Duchenne points out: first, that in paralysis from cold, the paralysed muscles retain their electrical contractility unimpaired, whereas in lead-palsy this quality rapidly diminishes or disappears; second, that the supinator longus, which never suffers in lead-poisoning, is invariably implicated in the present case. The following, as is shown by M. Duchenne, is the proof of implication of this muscle:—namely that when the patient has placed his forearm in the position of semi-flexion and semi-pronation, and attempts to flex it more completely (the attempt being opposed) the long supinator, which in that position is the flexor of the forearm, can be neither seen nor felt to contract. As in lead palsy, the flexor muscles of the forearm and hand and the interossei escape. Paralysis of the musculo-spinal nerve from cold is almost always followed sooner

or later by recovery. In some cases, however, progressive wasting of the affected muscles comes on; and occasionally, also, the opposing muscles and the interossei become manifestly enfeebled from want of use.

Treatment.—The value of electricity in the treatment of the above forms of paralysis is very great. When the deltoid pains are unattended with fever or local signs of inflammation, M. Duchenne strongly recommends the use of cutaneous faradism, effected upon a dry surface, with a feeble and slowly intermittent current. When, however, there is wasting or paralysis, faradism of the muscles or the interrupted galvanic current is especially indicated, both in the case of the deltoid and in that of the muscles of the forearm. In both cases, moreover, frictions, stimulant applications, and blisters are often serviceable. When there is distinct evidence of inflammation, the various forms of electricity are not only inefficacious, but injurious. The ordinary remedies for local inflammation are then called for.

XXI. LOCAL FUNCTIONAL SPASM AND PARALYSIS, WRITER'S CRAMP, WRY-NECK, HISTRIONIC SPASM, &c.

Definition.—The affections here referred to are limited to a single muscle, or part of a muscle, or to groups of muscles, and occur only or mainly at the time when certain accustomed specific actions in which they are engaged are in process of performance—the affected muscles apparently acting normally under all other conditions, and in other respects seeming fairly healthy.

Causation.—The causes of these functional derangements are exceedingly obscure. They are, however, for the most part induced by the long-continued exercise, in special motor combinations, and the consequent fatigue, of the muscles which afterwards become affected.

Symptoms and diagnosis.—The most common of the affections included in the present group are those which are known in this country as 'writer's cramp,' or ''scrivener's palsy,' and 'spasmodic torticollis,' or 'wry-neck,' and 'histrionic spasm.'

1. *Writer's cramp* affects, as its names imply, those who are engaged in writing, and more especially those whose avocations compel them to write for many hours a day continuously for long periods of time. It generally commences with a sense of fatigue or pain in certain of the muscles of the hand or forearm, which comes on shortly after the patient has begun to write. This condition increases slowly until pain or weariness attends all his attempts to write, and compels him to rest for a time or to desist altogether. Sooner or later, and sometimes from the very commencement, some spasm or loss of power, coming on

only when the patient is engaged in writing, seizes certain of the muscles which he is exercising, and renders his handwriting tremulous or jerky or arrests his operations completely. In the earlier stages of the disease, the patient sometimes resists its influence with more or less success. But its almost inevitable tendency is to go on from bad to worse, until at length the use of the pen becomes impossible. In some cases patients have learnt to write with the left hand; but in many of these, unfortunately, this hand has after a while become affected similarly to the other.

The affection is sometimes paralytic, the patient suddenly losing power over certain muscles, and dropping the pen from his hand; in most cases it is spasmodic, the muscles causing tremulous or choreic movements, or sudden flexion, extension, or rotation. Different muscles are affected in different cases. In some instances they are the extensors and flexors of the index finger; in some the interossei of the second and third fingers; in some the muscles of the thumb; in some the supinators of the hand. Occasionally the muscles of the hand and forearm are all more or less involved. In some cases the spasm or paralysis commences in the deltoid or other muscles of the shoulder; and in some it extends from the muscles of the hand and arm to those of the head and neck and trunk. As a rule more or less sense of fatigue or pain accompanies the functional motor disturbance, but occasionally the patient complains of muscular cramp or of neuralgic pains. However extensive the paralysis or spasm becomes, the muscles retain their functional activity for all other movements than those which have induced them; but there is for the most part distinct loss of muscular power.

2. *Spasmodic wry-neck* is an affection of adult life, and of either sex. It comes on for the most part insidiously with uneasiness or pain in the affected side, and a tendency to jerk the head as though to relieve some feeling of discomfort. By degrees the uneasiness increases, the spasmodic movements become more constant and more violent, and the head is habitually carried on one side. At first the patient can temporarily restrain his spasms by a voluntary effort, and temporarily hold his head erect, or he can counteract the spasmodic contraction of the affected muscles by the voluntary action of the healthy muscles of the opposite side. But after a while the head and neck become permanently twisted, and the clonic spasms which accompany this twisting are beyond even temporary control. The spasm of the muscles of the neck is apt to become associated with similar spasm of the facial muscles or of those of mastication, or of those of the shoulder or arm. In the great majority of cases the spasms cease during sleep, or whenever the head is supported.

The muscles which are affected differ in different cases. In some instances they are those which rotate the atlas and skull upon the axis, and the movements of the head are those of simple rotation. Some-

times it is the splenius capitis which suffers; in which case the head is inclined downwards and backwards towards the affected side, the face at the same time rotating towards the same side, and the skin of the side of the neck being thrown into deep transverse folds. Sometimes it is the clavicular portion of the trapezius which is implicated; in which case, as in the last, the head is inclined downwards towards the affected side, and thrown somewhat backwards, but the face is rotated towards the opposite side. If the fibres of the trapezius which are attached to the shoulder also are involved, the shoulder will be distinctly elevated. Sometimes the sternomastoid suffers; in which case, as when the trapezius is contracted, the head is inclined towards the affected side, and the face is rotated towards the opposite shoulder; but, contrary to what happens in either of the other cases, the head is thrown forwards. Although the several muscles which have just been named may be affected separately, it is more common to find groups of muscles implicated. But the affected muscles can generally be readily recognised not merely by their effect on the movements of the head, but also by their contraction, rigidity and spasmodic action.

3. Among examples of other similar conditions we may quote the following, chiefly from M. Duchenne:—A tailor whenever he had made a few stitches suffered from violent rotation of the arm inwards, in consequence of contraction of the subscapular muscle. A fencing-master, whenever he put himself into the posture of defence, was seized with rotation of the arm inwards and violent extension of the forearm. A turner whenever he attempted to work the lathe with his foot, suffered from spasmodic contraction of the flexors of the foot upon the leg. A gentleman, who also suffered from writer's cramp, became subject, when he attempted to read, to contraction of the rotator muscles of the head, which carried his head to the right. A literary man, who had been employed for some years in deciphering manuscripts, suffered after a while from double vision, coming on a few seconds after he had fixed his eyes intently on any object; the defect was due to spasmodic contraction of one of the internal recti. A student, who had overworked himself, became the victim of a strange affection which rendered reading impossible, and finally impelled him to commit suicide. As soon as he began to read, he was seized with a painful constriction of the forehead, temples, and eyes, during which the eyebrows were elevated by spasmodic contraction of the frontales, and the eyes closed by the powerful action of the orbiculares palpebrarum. Pianists are liable to the same affection as writers are. Singers occasionally become incapable of singing from involvement of the laryngeal muscles; soldiers of marching from implication of the peroneus longus. In some cases the spasms affect the muscles of expression (histrionic spasm), in some the platysma, in some the muscles of mastication, and in some those of respiration.

Pathology.—The pathology of these functional affections is very

obscure. Most writers believe that the primary fault is in the nervous centres; but Dr. Poore, in his able text-book of electricity, seems to prove conclusively that the disease is in many cases due to abuse of the implicated muscles, which 'become tired out, and degenerate into a condition of chronic fatigue or irritable weakness;' and he shows also that, contrary to the general belief, the affected muscles are absolutely weaker than their healthy fellows, and that their faradic irritability is diminished.

Treatment has not usually proved satisfactory. In Duchenne's hands faradism failed absolutely. Dr. Poore, however, has latterly obtained great success by the employment of the continuous current in combination with rhythmical exercise of the enfeebled muscles. His mode of using the current in writer's cramp is as follows:—'One pole (the positive) is placed, let us say, in the axilla, and the other over the ulnar nerve, just where it leaves the edge of the biceps muscle *en route* for the olecranon. The strength of the current is short of that which causes muscular contractions, but is just sufficient to make the patient conscious of a tingle in the end of the little finger when the circuit is made or broken. The patient is then made to exercise the interossei by separating and approximating the fingers rhythmically.' The nerve to be galvanised and the muscles to be exercised will of course differ in different cases. Liniments and douches may also be employed: and tonics are generally indicated. But in all cases it is of the highest importance for the patient to abstain in a greater or less degree from all those habitual actions with which the muscular default is especially linked, and never to attempt to overcome it by violent efforts. Rest is essential.

XXII. NEURALGIA. TIC DOULOUREUX. SCIATICA.

Definition.—By the term neuralgia is meant pain, for the most part paroxysmal, occurring in the course of nerves and in their areæ of distribution.

Causation.—Neuralgia is the result of numerous different conditions. It may depend on injuries to nerves, due to contusion, laceration, or the impaction of foreign bodies; on pressure, such as may take place when the bony channels through which certain nerves pass become contracted from any cause, or when nerves are pressed upon by tumours or other adventitious masses; or on the implication of nerves in disease, as, for example, when they are involved in rheumatic or other inflammations, in syphilitic gummata, or in carcinomatous or other tumours. In some cases it appears to depend upon, or to be connected with, certain constitutional conditions, such as the malarial cachexia, anæmia, and hysteria. In a considerable number of cases

no cause whatever, local or constitutional, can be discovered. Neuralgic affections are said to be hereditary. This is no doubt true of specific forms, such as megrim, and possibly of tic, but can scarcely admit of satisfactory proof in respect of the heterogeneous cases which make up the great bulk of ordinary neuralgiæ.

It need scarcely perhaps be pointed out that neuralgic pains, which are sometimes of extreme intensity, attend a large number of the diseases which have already been discussed: among others, tabes dorsalis, spinal caries, and more particularly carcinoma of the vertebræ or pelvic organs, certain inflammatory affections of deep-seated parts, such as abscess of the liver, calculous pyelitis, and hip-joint disease, and a large proportion of the cases of zona or herpes zoster.

Lastly, it is important to bear in mind that the lesion or local condition causing neuralgia may exist in the course of the implicated nerve, or in the spinal cord or brain, or (as above pointed out) may occupy some remote part from which it acts indirectly.

Symptoms and progress.—Neuralgia is characterised essentially by the occurrence of pain in the course and distribution of some one or more of the sensory nerves. The pain varies in character: it may be tingling, aching, burning, boring, crushing, cutting, stabbing, darting; it may be more or less continuous, but usually occurs in sudden lightning-like shocks, which come on either singly or in paroxysms made up of a larger or smaller number of such shocks; and even when it is continuous it is usually attended with exacerbations presenting more or less of the latter character. The pain varies also in intensity; in its severest paroxysmal form the patient's sufferings are horrible—sometimes he raves and stamps like a madman, sometimes utters half-suppressed groans, sometimes screams aloud; but under any circumstances is so absorbed in the intensity of his sufferings that he appears almost unconscious of everything which is going on about him; on the other hand, it may consist in nothing more than a little tingling, creeping, or burning. This is often the case during the inter-paroxysmal stage of those cases in which there is never entire cessation from pain; and such sensations often constitute the commencement of each paroxysmal attack.

It very commonly happens that more or less tenderness or hyperæsthesia is associated with neuralgia; there may be tenderness along the course of the affected nerve; or there may be general tenderness in the area of its distribution, or spots of special tenderness scattered here and there upon that surface. It is a fact of considerable importance, first established by Valleix, and since confirmed by numerous observers, that in neuralgia there are generally, if not always, specially painful spots, which are more or less characteristic for each nerve that may be involved, and are determined mainly by the emergence of the nerve or of some of its branches from a bony canal, or by their passage through some dense fascia. Trousseau insists that one of these pain-

ful spots is the spinous process of that portion of the spine from which the painful nerve escapes. The neuralgic paroxysm may often be induced by irritation of the hyperæsthetic parts, or even by touching them; on the other hand, firm pressure upon them may relieve or avert it.

Anæsthesia, again, is not unfrequent in connection with neuralgia. Sometimess more or less impairment of tactile sensibility or discrimination goes along with considerable tenderness or hyperæsthesia. But absolute loss of sensation in the affected area occasionally supervenes after a time.

The sudden darts of intense pain which so commonly attend neuralgia are generally associated with more or less sudden reflex movements or twitchings of the part affected; if the toe be attacked, the leg is momentarily drawn up by an uncontrollable impulse; if the finger, the arm; if the face (as in ordinary tic-douloureux) spasmodic twitching of the muscles of the painful region occurs. These convulsive movements may vary from mere twitching of the muscles to spasmodic contractions of considerable force.

The above phenomena are apt to be complicated with other local manifestations. In many cases the affected surface becomes during the occurrence of the paroxysm more or less red and congested; and not unfrequently obvious dilatation of the arteries and veins both in, and leading to or from, the implicated region takes place, attended with painful throbbing. In connection with congestion, there is apt also to be some temporary modification of function in the affected area, such as arrest or increase of secretion, which is especially obvious if the conjunctiva or the mucous membrane of the nose or mouth be the part involved.

Further, the various nutritive lesions, especially erythematous and herpetic eruptions, which have previously been referred to affections of the sensory nerves, are all apt to occur in connection with neuralgia. Occasionally also the hair over the affected region turns temporarily or permanently white.

An interesting feature of neuralgia is the tendency to shift which it presents in many cases. Thus in trifacial neuralgia the paroxysmal attacks not unfrequently wander either from day to day, or it may be at distant intervals, from one branch of the nerve to another branch; the pain may even pass over to the great occipital nerve or to branches of the cervical or brachial plexus.

Another important point connected with neuralgia, and one indeed which has been regarded as inseparable from true neuralgia, is its unilateral or unsymmetrical character. This characteristic, however, is not universal, and occasionally both arms or both legs are symmetrically and equally affected.

In a large proportion of cases neuralgia is essentially intermittent; the pains come on in paroxysms lasting probably from a second or two

to a minute, rarely longer, which recur every five or ten minutes, day and night, or manifest themselves at longer and more or less irregular intervals. Occasionally they remit for weeks or months together.

The general state of health of neuralgic patients presents considerable diversity, yet it is important in reference both to prognosis and to treatment to pay attention to this subject. Thus in some cases we find the patient anæmic, in some hysterical, in some labouring under the consequences of old syphilis; sometimes he is rheumatic, sometimes gouty, sometimes he is suffering from the effects of the malarious poison. But in a considerable number of cases, and these are often the most severe and intractable, no general morbid condition can be discovered beyond that which the persistent neuralgia itself induces. In these cases the disease is not unfrequently incurable.

Neuralgia may attack any of the sensory nerves, as well those supplying the viscera as those distributed to the skin. Among the former class may especially be named neuralgiæ of the heart, stomach, kidneys, uterus and ovaries, testes, and mammæ. Among the latter class the more important probably are trifacial neuralgia or tic douloureux, and sciatica.

1. *Tic douloureux*, or as Trousseau terms it, epileptiform neuralgia, is at once the most severe and the most typical variety of neuralgia. It comes on in adult life, and is for the most part of lifelong duration. Its causes are obscure: sometimes it is referred to carious teeth, sometimes to exposure to cold, sometimes to gastro-intestinal irritation, sometimes to old age or failing health or malarious influence. The neuralgic phenomena involve the branches of the fifth nerve of one side. In some cases it is the first division, in some the second, in some the third, occasionally the whole nerve; or it may be that certain portions only of its divisions are involved. The pains, moreover, are apt to shift from time to time from one division to the other, or from certain fibres to certain other fibres. They vary in character as other neuralgic pains vary; but usually are burning or shooting, and occur in sequences of sudden electric-like shocks. They vary also in intensity, from a mere sense of warmth or tingling to paroxysms of the most intense agony. They sometimes come on at rare intervals; sometimes, on the other hand, occur every few minutes, night and day, and are then apt to be brought on by any movement of the affected parts, by pressure, by a sudden shock, or even by a breath of cold air. Consequently, in cases in which the second or third division is involved, the patient finds it impossible to masticate, and almost impossible to take nourishment by the mouth. Under any circumstances the severity and frequency of the paroxysms are apt to vary from time to time; and occasionally, even in severe cases, the attacks intermit for comparatively short periods. In aggravated cases the paroxysms of pain are often attended with spasmodic contractions of the muscles of the affected region. Sometimes the patient smacks his lips, or chews,

or executes other movements which are apparently voluntary, and are performed with the object of relieving pain. More frequently he rubs his face during the paroxysms either with his hand or a handkerchief, or with a pad that he carries in his hand for the purpose. This constant rubbing not unfrequently wears down the hair of the affected side—the whisker, the beard, the hair in the neighbourhood of the temple—which then appears as if kept close shaven; occasionally it even modifies the form of the side of the face. Further, the frequently repeated spasmodic action of the muscles of the affected side produces after a while a permanent curiously wrinkled condition of the surface.

Tic douloureux, unbearable though it appears to be, does not tend directly or necessarily to shorten life. Patients nurse their agony for many years. The only ways in which it can be regarded as inimical to life are by the difficulty which it occasionally opposes to the ingestion of food, and by driving the patient to suicide.

2. *Sciatica.*—This is one of the most common varieties of neuralgia. It frequently arises from exposure to cold, but may be due to many other causes; it is occasionally attended or followed by some degree of anæsthesia, and occasionally, but mainly when due to structural disease, leads to wasting of the muscles. The pain is of true neuralgic character, and is greatly aggravated by movement of the implicated limb, or by pressure. It is in many cases exceedingly persistent and difficult of cure.

Treatment.—In dealing with cases of neuralgia it is always of great importance to ascertain, if possible, the cause on which it depends, and then, if it be within our competence, to obviate or remove it. If, for example, the pain be traceable to the influence of the malarious poison, quinine or arsenic is indicated; if it be connected with anæmia, iron is probably the best remedy; if it be a consequence of exposure to cold or rheumatism, the treatment suitable for these conditions should be employed; if it be referrible to syphilis, iodine and mercury are most likely to be serviceable; and further, if it depend on the existence of some local morbid process compressing or otherwise involving the nerve, our treatment must vary accordingly.

But in a large number of cases, no such hints for treatment are afforded us; we can then, so far as general treatment is concerned, only deal with them empirically. Among remedies which, under these circumstances, have been found useful, may be enumerated iron, arsenic, quinine in large doses, oil of turpentine, chloride of ammonium, phosphorus, croton-chloral hydrate, aconite, Indian hemp, belladonna, and opium. Of these, opium, or its alkaloid, morphia, is by far the most valuable. Indeed, the severest cases of tic, and of similar forms of neuralgia in other parts, often find relief only from large and repeated doses of this drug, which may then be given by the mouth, or, preferably, by subcutaneous injection. If given by the mouth, it may be necessary at length, having begun with small doses, to administer as

much as from twenty to sixty grains of morphia daily. Alcohol is not unfrequently serviceable in relieving pain. Cases of the less severe forms of neuralgia are occasionally cured by a few glasses of wine, or by a tumbler of strong brandy and water.

Local medication is often very valuable. Of course the several narcotics which have been enumerated, especially morphia and atropia, may be injected subcutaneously at the seat of pain. But, besides this, the application to the surface, or the inunction, of opium, belladonna, or aconite, often gives relief. The most valuable of these applications is aconite in the form of the unguentum aconitiæ. Counter-irritation also is frequently of much benefit, more especially by means of blisters, issues, the actual or galvanic cautery, and acupuncture. Electricity is especially valuable. Duchenne employed cutaneous faradism, rendering the affected surface dry by dusting it with some powder, and then applying to it for a minute or so faradism of considerable strength, and repeating the process from time to time, according to circumstances. But the continuous current is much more efficacious. In this case well-wetted sponges must be used, and the current employed must be of no greater intensity than the patient can readily bear; and, as has been before pointed out, the origin of the affected nerves should be included between the rheophores, of which one should be moved over the painful region, and especially applied to the painful points. Moreover here, as in the other cases, the applications should be of short duration, and frequently repeated. Lastly, division of the affected nerve has often been practised, especially in cases of tic douloureux. It cannot be asserted that this procedure ever absolutely cures the neuralgia; but there is no doubt that it very often effects a temporary cure—a cure lasting occasionally for a few weeks or even for several months.

INDEX.

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CONTENTS.

PART I.

PART II.

LIST OF DIAGRAMS.

Errata.

Page 134, line 41, *for* Obermeyer *read* Obermeier.

„ 475, line 3; 476, line 35; and 477, lines 3 and 7; *for* circo *read* crico.

„ 1040, line 22, for *Rythmical tremors* read *Rhythmical tremors.*

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