Lectures on Nervous Diseases.

E. C. Ramsey

LECTURES

ON

NERVOUS DISEASES

FROM THE STANDPOINT OF CEREBRAL AND SPINAL LOCALIZATION, AND THE LATER METHODS EMPLOYED IN THE DIAGNOSIS AND TREATMENT OF THESE AFFECTIONS.

BY

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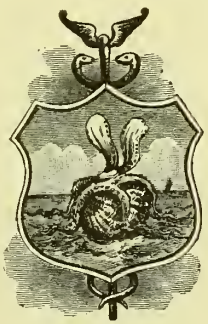
PROFUSELY ILLUSTRATED WITH ORIGINAL DIAGRAMS AND SKETCHES IN COLOR BY THE AUTHOR; CAREFULLY SELECTED WOOD-CUTS, AND REPRODUCED PHOTOGRAPHS OF TYPICAL CASES.



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ERRATA.

Page 18. Fig. 6. Text. For *shaded portions* read *gray* and *red masses.*

Page 156. 25th line For musculo-*spinal*, read musculo-*spiral.*

Page 166. 26th line. For *hyprocephalus*, read *hydrocephalus.*

Page 258. No. (5) occurs twice in the diagram The lower (5) should be (6).

Page 350. Under the "FUNCTIONAL DISEASES OF THE CORD," read *Thomsen's* Disease for *Thomson's* Disease.

I DEDICATE THIS VOLUME

TO MY FRIEND,

GEORGE T. STEVENS, M.D., PH.D.,

AS A TRIBUTE TO

HIS PERSONAL INTEGRITY AND GENERAL SCHOLARSHIP,

AND, ABOVE ALL, TO

HIS ORIGINAL INVESTIGATIONS RESPECTING THE
CAUSATION AND CURE OF FUNCTIONAL
NERVOUS DISEASES.

PREFACE.

WHATEVER of merit or demerit this course of lectures may possess in the opinion of its readers or critics, it must be conceded that it differs radically in arrangement and plan from others published upon this department of medicine.

The first section treats of those facts (anatomical, physiological, and pathological) upon which the science of cerebral and spinal localization of to-day is, of necessity, based.

The second section discusses more completely than most works in this field the various steps which should be taken by an aspirant in neurology during the clinical examination of a patient; and the deductions which may be drawn from the facts thus elicited. In many instances, authors have given a very incomplete *résumé* of this field or have omitted it entirely.

The third and fourth sections treat of individual diseases of the brain and spinal cord. Each is discussed from the clinical standpoint indicated in the first section, viz., the *localization* of the lesions described, as well as the recognition of the type which is encountered.

The section which treats of "functional" nervous diseases will, I trust, receive the careful attention it deserves. It comprises a full *résumé* of the researches of Dr. George T. Stevens respecting the bearings of "eye-defect" and "eye-

strain" upon the causation and cure of these imperfectly understood conditions. My own observations in this field have been quite extensive. They lead me to fully indorse all that has been claimed by this author. I can bear strong testimony to the value of the new methods of examination and treatment suggested by him for these distressing and obstinate maladies. Like other delicate procedures, they can only be intrusted to skillful hands, well versed in their intricacies and careful in respect to minute details. No other treatment has ever yielded me such satisfactory results in severe forms of epilepsy, hysteria, chorea, neuralgia, headache, insanity, and functional visceral derangements. As no drugs were employed by me in many of these cases, the relief obtained must be attributed solely to the method of treatment referred to.

The final section treats of electricity,—an agent which is to-day invaluable in neuro-therapeutics, but which is seldom if ever discussed in neurological manuals. To this subject the author has given much attention for some years past.

A glossary of neurological terms has been added, in order that the reader may not grope in the dark when uncertain respecting the meaning of a word which is new to him.

Much time has been devoted to the condensation of the matter presented within reasonable limits. To deal with an extremely difficult and somewhat new field in medicine in such a way as to bring it within the grasp of those who have devoted little or no attention to it, and at the same

time to avoid, from those more familiar with the field, the criticism of superficiality, has been no easy task. The author does not expect that his attempt will prove acceptable to all. An experience of fourteen years, however, in teaching medicine has governed the author in the presentation of this field as he deems most wise.

To those who have attended the author's lectures, delivered from year to year before his classes (either in the Medical Department of the University of the City of New York, the New York Post-Graduate Medical School and Hospital, or the Medical Department of the University of Vermont), much of the matter here contained will certainly be familiar, and he trusts acceptable.

The labor of preparing this volume from scattered lecture-notes has been greater than might appear on a cursory examination. Many of the illustrations are from the author's pencil, while others are from original photographs of his cases. Most of the colored diagrams incorporated are similar to those drawn by him upon the blackboard before his classes. Colors are of great service in making a diagram clear and easy of comprehension. To the author's mind, diagrams in this special field of medicine are of greater utility to the busy practitioner than microscopic sections, because very intricate mechanisms are discussed and interpreted which cannot always be shown.

Portions of this work embody extracts from two chapters upon Diseases of the Brain and Spinal Cord in the third edition of the author's treatise on "Surgical Diagnosis." Some other portions have appeared in print from time to

time, in the following journals: *New York Medical Journal*, *Medical Record*, *Archives of Medicine*, *Journal of Nervous and Mental Diseases*, *Medical News*, *Harper's Monthly*, *Medical Bulletin*, and *Medical Register*.

In closing, the author would acknowledge his great indebtedness to the original investigations of those who by their researches have been the source of much valuable information incorporated by him in this work. This acknowledgment must act as a poor substitute for frequent reference-notes, which are precluded on account of a want of space. A bibliography of some of the more important works and monographs on this field to which the author is indebted is, however, appended for the benefit of the reader. Special care has been exercised in selecting these, as far as possible, from sources which are easily accessible to American readers.

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156 Madison Avenue,
New York City, May, 1888.

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SECTION I.

ANATOMICAL, PHYSIOLOGICAL, AND PATHOLOGICAL DEDUCTIONS RESPECTING THE CEREBRO-SPINAL AXIS OF MAN.

SECTION I.

ANATOMICAL, PHYSIOLOGICAL, AND PATHOLOGICAL DEDUCTIONS RESPECTING THE CEREBRO-SPINAL AXIS OF MAN.*

Some thirty-six years ago, by a premature explosion of gunpowder, an iron bar three and a half feet long, one and a quarter inches in diameter, and weighing thirteen and a quarter pounds, was shot completely through a man's head and perforated his brain. This man walked up a flight of stairs after the accident, and gave his account of how it happened. Although his life was naturally despaired of for some time, he developed no paralysis, nor did marked impairment of his intellectual faculties follow convalescence. Eventually he recovered his health. Twelve years elapsed before his death; during which time he worked as a laborer on a farm.

The "American crowbar case" at once became famous. It startled the minds of the reading public, and confounded the medical fraternity. No satisfactory explanation of the remarkable features of the case could be given. Some prominent medical men pronounced it "an American invention," and laughed at the possibility of such an occurrence. The skull was exhumed, however, after death, and is to-day in the medical museum of Harvard University.

This case may be said to have been the starting-point of a new epoch in medical science. It rendered untenable all previous hypotheses that had been advanced regarding the organ of the mind. It proved conclusively that little, if anything, was known at that time respecting the architecture of the brain of man, and the functions of its component parts.

Since then, a large number of observers have published the results of various forms of experiments upon animals, made with a view of determining the physiology of the brain; but for some years the conclusions drawn from such investigations were contradictory, and nothing was definitely established.

We now are aware that serious defects existed in the early methods of research. By great ingenuity these have been gradually eliminated. We owe, however, to the discoveries of Türck, Fritsch and Hitzig, Waller, Flechsig, and Gudden, most of our knowledge of *new methods*

*The first ten pages of this chapter are quoted (with many important modifications and additions) from an article contributed by the author to *Harper's Monthly*, March, 1885.

of research which have simplified the study of the nervous system during life and after death. These methods of investigations have settled many points in dispute. They have also made our knowledge more accurate, and in accord with clinical observations.

The last decade has enabled us to bring many of the results obtained by vivisection into perfect harmony with pathological data. Those who have claimed that conclusions drawn from experiments upon animals are not applicable to man are, to-day, confronted with certain unanswerable facts to the contrary. Nature, through the agency of disease processes, is constantly performing experiments upon human brains; and the symptoms so produced may be recorded during life, and compared with the changes found in the brain after death. Physiology and pathology have thus proven valuable lines of research in this field *

To-day, the "crowbar case" is no longer a mystery to specialists in neurology. Bullets have been shot through the brain since then without loss of motion, sensation, or intellect; and, in some cases, they have been known to remain buried in the brain substance for months without apparent ill effects. Five years ago a breech-pin of a gun, four and three-quarter inches long, was forced into the brain of a boy nineteen years old, through the orbit, and its presence was not suspected for some five months. It was discovered during a surgical attempt to repair the facial deformity that resulted from the accident. Death followed the removal of the foreign body from the brain in consequence of inflammation created by the piece of iron, or possibly by its extraction. This case is quite as remarkable as the crowbar case, but it excited less interest in neurological circles because we are in possession of new facts.

We know, to-day, that if even a needle be thrust into one region of the brain (the medulla oblongata, Fig. 1), immediate death may follow; while a crowbar may traverse another portion of the organ, and recovery be possible. The effects of injury to the brain depend rather upon its situation than its severity.

In the light of our present knowledge the brain must be regarded as a *composite organ;* whose parts have each some special function, and are, to a certain extent, independent of each other.

* There are at the present time three distinct schools among the experimental physiologists respecting the subject of cerebral localization. Ferrier and Munk represent a faction which strenuously hold the view that the cortical gray substance can be mapped out into areas whose limits, as well as their individual functions, are clearly defined. Goltz stands at the head of a school which denies the accuracy of these views, and supports the conclusion, originally advanced by Flourens, that the brain can only act as a whole. Exner and Luciani (in common with their followers) occupy a ground which opposes very sharply-defined boundaries to cortical areas, functionally associated with the various senses. They believe that these areas overlap each other to a greater or less extent. At present, the latter view seems to be most perfectly in accord with clinical and pathological data.

One limited part is essential to vital processes; hence its destruction causes death. Another part presides over the various movements of the body; hence paralysis of motion is the result of destruction of any portion of this area. A third part enables us to appreciate touch, temperature, and pain; and some disturbance of these functions will be apparent when this region is injured or diseased. A fourth region presides over sight; disturbances of vision may follow disease or destruction of this area, in spite of the fact that the eyes escape. In the same way, smell and hearing are governed by distinct portions of the brain, and also the sense of taste. When a combined action of different parts is demanded —as in the exercise of the reason, judgment, will, self-control, etc.—the knowledge gained by means of the special senses can be contrasted and become food for thought.

The skilled neurologist can determine to-day, in many cases, by the symptoms exhibited during life, the situation and extent of disease processes that are interfering with the action of certain parts of the brain. So positive is the information thus afforded, in some cases, that surgical operations are now performed for the relief of the organ. A patient who had lost the power of speech from an accumulation of pus within the brain, was lately cured by the removal of a button of bone from the skull over the seat of the pus, and its prompt evacuation. Epileptics who suffer in consequence of brain-irritation may sometimes be cured of their fits by the mechanical removal of the cause. Paralysis can occasionally be alleviated by a removal of blood or pus from the surface of the brain through a hole in the skull. Only a few months ago a bullet, which had been shot into the head during an attempt at suicide, was removed from the skull, in one of our hospitals, by means of a counter-opening. The labors of such men as Meynert, Nothnagel, Ferrier, Flechsig, Wernicke, Munk, Luciani, Exner, Charcot, and others, have made neurology a science that would exceed the comprehension of its founders. Our ability to localize disease within the substance of the spinal cord is even more positive than in the case of the brain.

When we consider that it is by means of our nervous system that we move, feel, see, hear, smell, taste, talk, and swallow; that in our brains are stored all the memories of past events; that we digest and assimilate our food partly by the aid of nerves; and that, in fact, we perform every act of animal life by the same agency,—the utility of the latest information regarding the brain becomes apparent at once.

The nerves are but telegraphic wires that put the brain and spinal cord in direct communication with the muscles, the skin, and the various organs and tissues of the body.

The nervous centres may therefore be compared to the main offices of a telegraphic system, where messages are being constantly received

and dispatched. Every message sent out is more or less directly the result of some message received. So it is with our nerve centres. We are constantly in receipt of impressions of sight, smell, taste, hearing, touch, and other conscious sensations. These are called *afferent* impulses. As the result of the information so gained, we are constantly sending out *efferent* or motor impulses to the muscles. These create movements of different parts of the body. Respecting this view, Michael Foster expresses himself as follows: "All day long, and every day, multitudinous afferent impulses, from eye, and ear, and skin, and muscle, and other tissues and organs, are streaming into our nervous system; and did each afferent impulse issue as its correlative motor impulse, our life would be a prolonged convulsion. As it is, by the checks and counter-checks of cerebral and spinal activities, all these impulses are drilled and marshaled and kept in hand in orderly array till a movement is called for; and thus we are able to execute at will the most complex bodily manœuvres, knowing only *why*, and unconscious or but dimly conscious *how*, we carry them out."

Sometimes, however, the motor impulses sent out by the brain in response to sensory impressions take place in spite of our volition. Let us cite an instance in the way of illustration: a timid person sees perchance some accident in which human life is possibly sacrificed, or the sensibilities are otherwise shocked. His feelings overcome him, and he faints. How are we to explain it? Let us see what takes place. The impression upon the brain made by the organ of sight creates (through the agency of special centres in the organ of the mind) an influence upon the heart and (by means of vaso-motor nerve filaments) upon the blood-vessels of the brain. This results in a decrease in the amount of blood sent to the brain, and causes a loss of consciousness. In the same way persons become dizzy when looking at a water-fall, or from a height, through the effects of the organs of sight upon the brain.

Again, if a frog be deprived of only the upper part of the cerebral hemispheres, he is still capable of voluntary movement, breathing, swallowing, croaking, and all the other manifestations of frog-life. But when we observe such an animal with attention, we shall see that he is only a pure automaton, and that he differs from the normal frog in his behavior when left to himself and when disturbed. He will swim when placed in water, but only until he reaches a spot where he can safely repose. Then he relapses into quietude, evincing no desire to hop (as a normal frog would do) or to escape from his tormentor. Every time that his back is stroked the frog will croak. The same irritation will produce the same result over and over again. Such a frog, if placed upon a board which can be tilted, will climb up the board (in case he perceives that his equilibrium is endangered) in a direction necessary to render his position secure.

Otherwise he remains motionless. He is no longer a frog endowed with the normal attributes of that animal in health. He does not attempt to escape. He experiences no apparent alarm at surrounding objects. His movements can be predicted and repeated again and again at the will of the experimenter. He has been transformed into a machine in which every muscular movement can be traced directly to some stimulating influence from without.*

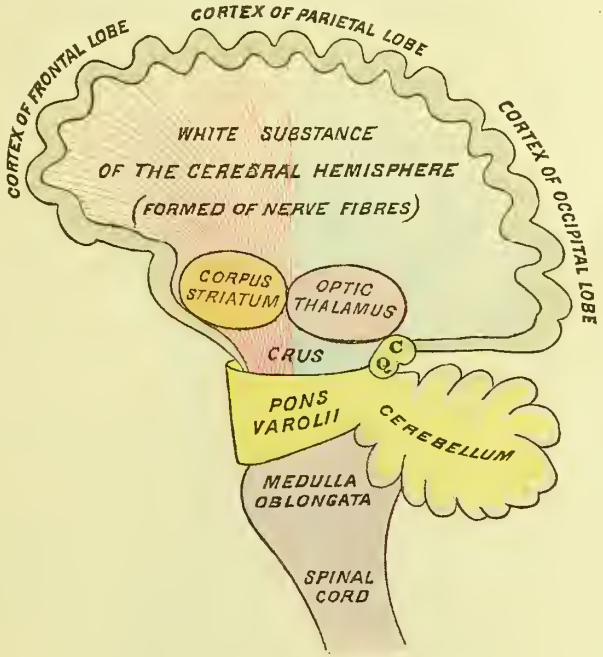


FIG. 1.—A DIAGRAM DESIGNED BY THE AUTHOR TO ELUCIDATE THE CHIEF COMPONENT PARTS OF THE HUMAN BRAIN.—The lettering upon the figure will be explained in the text. C. Q. the corpora quadrigemina. The lines within the white substance of the cerebrum or in the "crus" are not intended to convey any impression to the reader of the actual arrangement of the fibres; nor are the colors employed selected with special reference to the elucidation of the functions of the component parts of the organ thus diagrammatically shown.

Before we go farther, let us examine in a cursory way the anatomical elements of which the brain is composed. These are practically the same in all animals of the higher grades. We can then review the grouping of these elements, and study some of the structural details of that organ in man. Many of these have baffled all attempts at investigation until of late.

*The distinction between "*instinctive*" *or automatic acts* (which are governed by the spinal and cerebral ganglia) and "*conscious volitional acts*" (which are always of cortical origin) is not properly recognized by some experimental physiologists. Dr. M. Allen Starr has very happily shown in a late article on speech (*Princeton Review*) that this distinction helps materially to reconcile the antagonistic views now held by the opponents and supporters of cerebral localization.

We may start with the statement that the brain consists of two distinct anatomical elements,—*brain cells* and *nerve fibres*.

The number of brain cells in the cerebrum alone may be estimated at many thousands. Each cell, by means of its nerve fibres and the processes that spring from it, may be considered as a central station of an electric system. It can receive messages from parts more or less distant. It can dispatch messages in response to those received. Finally, it can store up such information as may be carried to it from time to time for future use, affording us, at the same time, memories of past events. It will simplify description if we consider each of the anatomical elements of the brain separately.

THE BRAIN CELLS.

These are placed chiefly upon the exterior of the organ, which is thrown into alternating ridges and depressions, somewhat like a fan when half closed. The ridges are called the "convolutions," and the depressions are termed "sulci;" or "fissures," in case they are deeper than the rest. The gray matter upon the exterior of the brain is called the "*cortex*."

The cerebral cortex is alone associated with consciousness and volition. Like gray matter found in other regions of the organ, the cortex consists of brain cells and a cement (formed of connective-tissue elements) that binds them together. This is called the "*neuroglia*."

Masses of brain cells are found imbedded within the substance of the organ; but their functions are less well determined than those of the cortical gray matter. The corpus striatum* and the optic thalamus are certainly the largest and perhaps the most important of these ganglionic masses.

If we study the appearance of the brain cells under the microscope, we find that different convolutions of the brain are peopled with cells that have individual characteristics of form and construction; hence we are justified (from an anatomical stand-point alone) in attributing different functions to individual areas of the cortex. This view is sustained, furthermore, by physiological and pathological investigation. We may consider each cell within the brain as possessing an individuality. Each is intrusted with and controls some particular function. Each is in telegraphic communication with other cells, and participates constantly in the growth and development of some special region of the body, acting in harmony with its fellows. Luys, who has carefully investigated the structure of these minute bodies, says of them: "Imagina-

*I apply the term "corpus striatum" throughout this work to its two halves (the caudate and lenticular nuclei, Fig. 6) collectively. Many of the German authorities employ it as synonymous with the caudate nucleus alone.

tion is confounded when we penetrate into this world of the infinitely little, where we find the same infinite divisions of matter that so vividly impress us in the study of the sidereal world; and when we thus behold the mysterious details of the organization of an anatomical element, which only reveal themselves when magnified seven hundred to eight hundred diameters, and think that this same anatomical element repeats itself a thousandfold throughout the whole thickness of the cerebral cortex, we cannot help being seized with admiration, especially when we think that

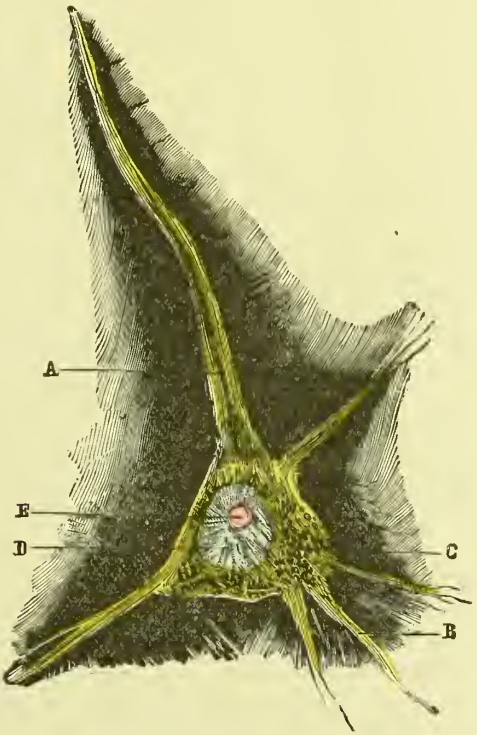


FIG 2.—CORTICAL CELL OF THE DEEPER ZONES AT ABOUT EIGHT HUNDRED DIAMETERS. (After Luys.) A section of the cell is made through its greater axis, its interior texture being thus laid bare. A, represents the superior prolongation radiating from the mass of the nucleus itself. B, lateral and posterior prolongations. C, spongy areolar substance, into which the structure of the cell itself is resolved. D, the nucleus itself, which seems only to be a thickening of this areolar stroma; it sometimes has a radiated arrangement. E, the bright nucleolus, which is itself decomposable into secondary filaments. The colors are only employed to aid in recognizing the various parts of the cell.

each of these little organs has its autonomy, its individuality, its minute organic sensibility, that it is united with its fellows, that it participates in the common life, and that, above all, it is a silent and indefatigable worker, discreetly elaborating those nervous forces of the psychic activity which are incessantly expended in all directions and in the most

varied manners, according to the different calls made upon it, and set it vibrating."

In the cortex of the brain we find the brain cells arranged in superimposed strata. The number of these strata varies in different areas of the brain surface.

Each stratum is composed of cells that have identical shapes, and whose structure is apparently the same. Delicate, hair-like processes are given off from the body of each cell, many of which subdivide like the branches of a tree, and become closely intermingled with those given off from neighboring cells. Some of these processes unquestionably serve to connect the cells that compose the various strata of the cortex; others serve as a means of attachment of nerve fibres to the cells. By means of these processes, *molecular movements* generated within any individual cell can probably be transmitted to other cells in the same stratum of the cortex, or to those composing other strata. Thus the *different layers of cells can probably act independently, or in conjunction with others.*

We may generalize respecting the purposes for which these minute bodies have been constructed, as follows:—

1. Some cells are unquestionably capable of *generating nerve force;* just as the electric battery, for example, generates electricity for the purpose of telegraphy.

2. Some are designed to *promote muscular contraction*, and thus to cause voluntary movements. They are enabled to do this by the nerve fibres. These conduct the current from the cells to definite muscles of the body. When, therefore, from any cause the generating power of motor cells, or the conducting power of motor fibres is interfered with, we have a symptom produced known as "motor paralysis."

Tumors, or inflammatory deposits sometimes press upon the motor cells to such an extent as to impair their function; inflammatory conditions may affect them directly, and cause their disintegration; blood may escape into the brain substance and plough up the delicate fibres that convey the impulses to the muscles (the condition known as "apoplexy"); and many other pathological conditions may derange or destroy this elaborate system of wires and batteries. Let me impress upon the reader that paralysis of motion is not a disease, as most people suppose. It is but one of the manifestations of disease.

3. Some cells of the cerebral cortex serve as *receptacles for nervous impressions.** Let us cite some examples. At birth the brain may be likened to the sensitized photographic plate before it has been ex-

* Disturbances of the memory may often prove a valuable aid in localizing the seat of a cerebral lesion. This fact has only been utilized of late; as new facts in cerebral physiology have been brought to light.

posed to the action of the lenses of the camera. Nothing has yet been recorded upon it. It may subsequently be beautified or disfigured by the impressions that are to be made upon it from without. At first the child stares stupidly about, unable to appreciate or properly interpret the pictures that are constantly being formed upon the retina by light. Loud noises frighten it, and softer sounds fail to attract its attention. It has not yet learned to determine the direction from which a sound comes. The appreciation of distance has not yet been acquired. The tiny hands are stretched out alike at remote and near objects.

Now mark the change that occurs when sufficient time has elapsed to allow the brain cells to accumulate *memories* of past events in numbers sufficient to admit of comparison with each other, and to form the basis of judgment.* The child soon begins to recognize familiar faces. It learns to discriminate between the voice and touch of the mother or nurse and that of a stranger. When only a few weeks old it begins to estimate distance, and to make voluntary efforts to grasp surrounding objects. Gradually its brain learns the meaning of articulate sounds, and by associating such sounds with definite objects it acquires a knowledge of language. The power of speech is developed later than the knowledge of language, because the complicated movements of the tongue, lips, and palate are difficult to perform properly, and also because articulation must of necessity be based upon a memory of the various sounds employed. Thus for many months the brain of a child is simply receiving and storing up in these wonderful receptacles, the brain cells, the impressions of the external world, that reach it chiefly by means of the organs of sight, smell, hearing, taste, and touch.

These facts become even more mysterious than they might at first appear to the reader when we reflect that the eye, for example, telegraphs the outline, coloring, and other details of every picture (focused by its lenses upon the retina) to the cells in the cortex of the occipital lobes of the cerebral hemispheres; and that these cells retain these impressions in such a manner that they can be recalled by a voluntary effort again and again as memories of what we have seen. The eye can thus go on taking photographs of external objects forever without fear of losing what it so elaborately duplicates. We have positive evidence to prove the accuracy of these statements. If the occipital lobes of both hemispheres be destroyed in animals, the sense of sight is lost immediately, in spite of the fact that the eyes have not been injured by the operation. I have had under my care several patients who have been rendered

*Clinical observation, as well as pathological statistics go to show that in right-handed subjects the left cerebral hemisphere is more intimately connected with the storage of memories than the right hemisphere. This is well illustrated in the reported cases of ataxic aphasia, paraphasia, word-blindness, and word-deafness.

totally blind in a lateral half of each eye by brain-disease; the other half retaining its normal power of vision. It is equally well proven that the memories of our conscious perceptions of odors, sounds, taste, and touch, are stored within the cells of different areas of the cerebral cortex, whose limits are already determined with approximate accuracy. These memories, as we all know, can be recalled at will with unimpaired vividness, just as picture after picture can be struck off the same negative when once made indelible upon a glass plate.

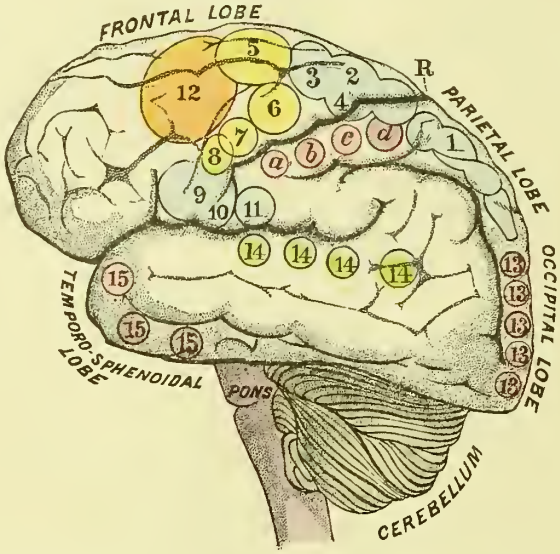


FIG. 3.—SIDE VIEW OF THE BRAIN OF MAN SHOWING THE AREAS OF THE CEREBRAL CONVOLUTIONS. (Modified slightly from Ferrier.) R, Fissure of Rolando. S, Fissure of Sylvius, divided into its two branches. 1 (on the postero-parietal [superior parietal] lobule). Advance of the opposite hind-limb as in walking. 2, 3, 4 (around the upper extremity of the fissure of Rolando). Complex movements of the opposite leg and arm and of the trunk, as in swimming; *a*, *b*, *c*, *d* (on the ascending parietal [posterior central] convolution), individual and combined movements of the fingers and wrist of the opposite hand; prehensile movements. 5 (at the posterior extremity or the superior frontal convolution). Extension forward of the opposite arm and hand. 6 (on the upper part of the antero-parietal or ascending frontal [anterior central] convolution). Supination and flexion of the opposite forearm. 7 (on the median portion of the same convolution) Retraction and elevation of the opposite angle of the mouth by means of the zygomatic muscles. 8 (lower down on the same convolution). Elevation of the ala nasi and upper lip with depression of the lower lip on the opposite side. 9, 10 (at the inferior extremity of the same convolution, Broca's convolution). Opening of the mouth with 9, protrusion; and 10, retraction of the tongue,—region of aphasia, bi-lateral action. 11 (between 10 and the inferior extremity of the ascending parietal convolution). Retraction of the opposite angle of the mouth, the head turned slightly to one side. 12 (on the posterior portions of the superior and middle frontal convolutions). The eyes open widely, the pupils dilate, and the head and eyes turned toward the opposite side. 13, 13 (centres of vision in the occipital lobes). 14 (of the infra-marginal, or superior [first] temporo-sphenoidal convolution). Pricking of the opposite ear, the head and eyes turned to the opposite side, and the pupils dilate largely (centre of hearing). Ferrier, moreover, places the centres of taste and smell (15) at the extremity of the temporo-sphenoidal lobe, and that of touch in the gyrus uncinatus and hippocampus major.

Professor Ferrier, of London, has mapped out, by means of a series of experiments upon the monkey tribe (the nearest approach to the type of man), a chart of the brain which shows the situation of certain groups

of cells or "centres" in the cortex that preside over particular functions. The cut introduced (Fig. 3), and its descriptive text, will make some of the conclusions of this author intelligible to the reader.*

Most of the conclusions of this investigator (excepting those relating to the visual centres, in which I think he is in error) have been partially verified upon man. It may interest the reader to know how these conclusions have been verified, since vivisection upon the human race is, of course, impossible.

In the first place, a careful study has been made of cases where Nature has performed the experiment of destroying or imperfectly developing portions of the brain, and where an opportunity of examining that organ after death has been afforded.† The clinical records of such cases have been collected from all reliable sources, and critically analyzed by competent medical men (Charcot, Ferrier, Nothnagel, Wernicke, Broadbent, Luys, Exner, Spitzka, Starr, Seguin, and many others).

Again, a large number of subjects who have suffered amputation of limbs, and who have survived the operation for some years, or who have manifested arrested development of limb, have been made to bear indirect testimony to the accuracy of the facts gained by vivisection and pathological research. When any part of the body is deprived of exercise, it will waste gradually from disuse. On this basis of reasoning, Bourdon and others have sought to determine the centres of motion of the limbs, by examining the cortex of the cerebral hemispheres of such subjects after death, with a view of determining the existence and exact seat of atrophy of definite groups of brain cells.

A third line of investigation, which has yielded brilliant results, consists in tracing the origin, course, and ultimate distribution of separate bundles of nerve fibres within the brain and spinal cord (Meynert, Flechsig, Gudden, Wernicke, Spitzka, Aeby, Roller, Starr, and many others). Some important discoveries have been made of late, which enable us to do this with accuracy,—a feat that was impossible by the older methods employed. A knowledge of the peripheral connections of certain groups of brain cells has shed much light upon their probable functions.

Finally, much has been learned by a microscopical study of the different layers of the cortex and the nerve-nuclei with reference to the

*The view upheld by Ferrier and Munk that the cortical areas have distinct lines of demarcation has been opposed by Luciani and Exner, who believe that the edges of these areas merge gradually into each other, and manifest less prominently than do their central portions the individual peculiarities of each.

† The study of microcephalic specimens, which bears somewhat upon this field, bids fair to become a very important line of investigation respecting the relations of certain parts of the brain to definitely recognized bundles of fibres within the crus, pons, medulla, and spinal cord. It is as yet in its infancy.

character of cells that compose them. It has been proven that the form and arrangement of the brain cells afford some clue to the special functions over which each preside (Luys, Arndt, Betz, Stephany, Spitzka, and others). Comparative anatomy has aided in this line of research.

Now, when we find that all of these methods lead us to an identical conclusion concerning any point in cerebral physiology, that conclusion becomes a fact beyond the possibility of dispute. Unfortunately for science, much still remains to be determined regarding this mysterious mechanism; but, on the other hand, much has been positively proven. Perhaps the day may never come when the human mind can fathom all of its mysteries.

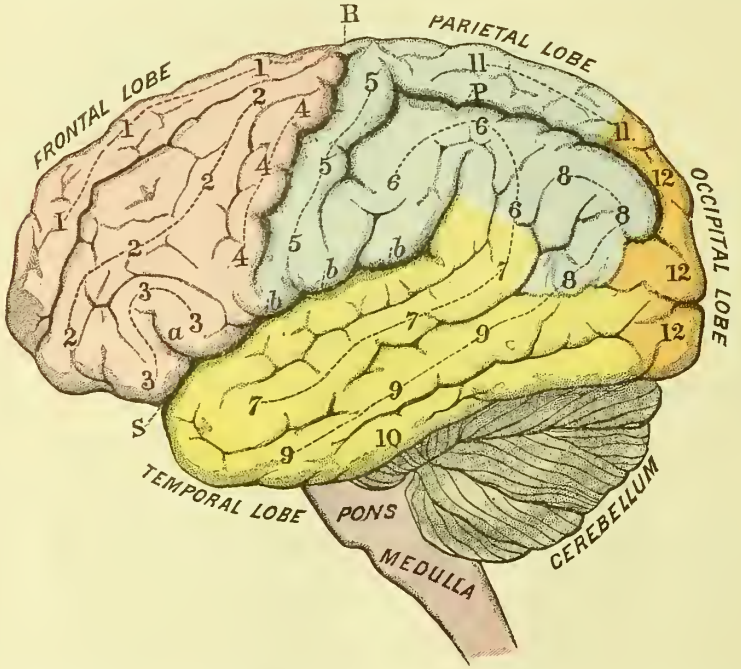


FIG. 4.—A DIAGRAMMATIC FIGURE, SHOWING THE CEREBRAL CONVOLUTIONS. (Modified from Dalton.) S, Fissure of Sylvius, with its two branches, *a*, and *b*, *b*, *b*. R, Fissure of Rolando. P, Parieto-occipital fissure. 1, 1, 1, The first, or superior frontal convolution. 2, 2, 2, 2, The second, or middle frontal convolution. 3, 3, 3, The third frontal convolution, curving around the ascending limb of the fissure of Sylvius (centre of speech movements). 4, 4, 4, Ascending frontal (anterior central) convolution. 5, 5, 5, Ascending parietal (posterior central) convolution. 6, 6, 6, Supra-Sylvian convolution, which is continuous with 7, 7, 7, the first or superior temporal convolution. 8, 8, 8, The angular convolution (or gyrus), which becomes continuous with 9, 9, 9, the middle temporal convolution. 10, The third, or inferior temporal convolution. 11, 11, The superior parietal convolution. 12, 12, 12, The superior, middle, and inferior occipital convolutions, called also the first, second, and third (the centres of vision). It is to be remembered that the term "gyrus" is synonymous with "convolution," and that both terms are often interchanged.

Before we pass to the consideration of the second anatomical element of nervous tissues—the nerve fibres—let me call the attention of the reader to the general form of the brain, and to a classification of the convolutions

that is now generally adopted. This will enable him to gain a clear insight into the functions of different areas of the cerebral cortex. Fig. 4 should be compared with Fig 5, as each will help to interpret the other.

The *lobes* of the cerebrum are named respectively the frontal, parietal, occipital, and temporal, from the bones with which they lie in contact. They are demarcated from each other by fissures or clefts that are clearly defined and more definitely placed than the sulci.

The fissures of Rolando and of Sylvius and the paricto-occipital fissure are of special importance. (Fig. 4.)

The diagram shows that the frontal and parietal lobes have four *convolutions* each, and the occipital and temporal lobes three each.

It must be remembered that the cerebrum has two hemispheres—a right and a left—only one of which is seen in profile. The right hemisphere is associated chiefly with the left lateral half of the body, and the left hemisphere with the right lateral half. Disease of one hemisphere of the brain may produce, therefore, a disturbance of some or all of the functions of the opposite side of the body below the head. There are exceptions to this rule, but it is a safe one to follow in the majority of cases.

Another diagram (Fig. 5) will be introduced later to show *certain areas of the surface of the brain* that are believed, in the light of our present knowledge, to preside over special functions, as, for example, those of speech, muscular movements of the extremities, sight, hearing, smell, and touch.

In summary, we are justified in drawing the following conclusions respecting the cells of the cerebral cortex from the results obtained by experimentation, clinical experience, and pathological data:—

1. The *surface of the brain* is the seat of all conscious mental action. It is the receptacle of all impressions made upon the organs of smell, sight, taste, hearing, and the tactile organs of the skin. Here, and only here, do such impressions become transformed into a conscious appreciation of external objects.

2. The *mental powers* are the result of different combinations of memories of past events stored in the cells connected with the special senses, and the activity of other groups of cells that are probably situated in the frontal lobes. Although the integrity of the entire organ is necessary to the unimpeded action of the higher mental faculties (such as judgment, will, self-control, reason, etc.), the cells of that portion of the frontal lobes that lies in front of the motor centres are perhaps more closely associated with these faculties than those of any other area. (Fig. 5.)

3. The *central convolutions** of the brain (a part of the frontal and

* Chiefly the *precentral gyrus*. The post-central gyrus appears to be associated with both motion and sensation to a greater extent than the precentral.

parietal lobes of each hemisphere) preside over motion and the memory of all motor acts of the limbs and body. The upper part governs the legs chiefly, the middle part controls the upper extremity, while the lower part presides over the complex movements of the tongue and lips necessary to speech. The memories of muscular acts are probably stored within the cells of the motor area. It is also probable that some forms of sensation are appreciated by the smaller cells of this area (Moeli, Tripier, and others).

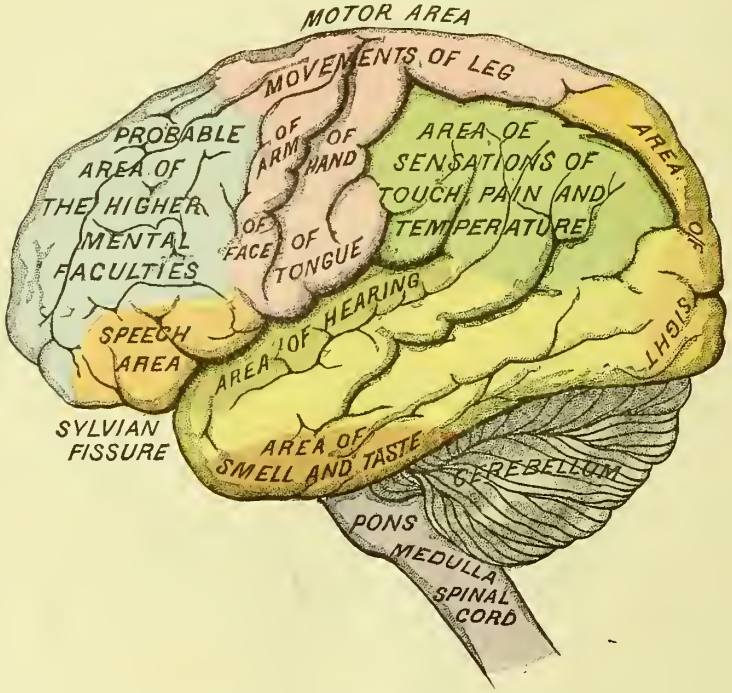


FIG. 5.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE PROBABLE FUNCTIONS OF DIFFERENT AREAS OF THE CEREBRAL CORTEX.

The limits of these areas must not be interpreted too literally by the reader as a basis for diagnosis. Each area probably merges almost imperceptibly into those which lie adjacent to it. The central portion of each are more clearly related to special functions than the peripheral portions.

The so-called "motor area" is probably connected not only with voluntary muscular contractions, but also with the conscious appreciation of all *sensory impressions connected with the muscles.* It might, therefore, be more properly designated as the "*muscular area.*"

4. The *occipital lobes** preside over the sense of sight and the memories of sight-pictures (Munk, Wernicke, and others). The recognition of familiar objects by the eyes depends on the activity of the cells in the cortex of these lobes. Hallucinations of vision point strongly toward a disturbance of the function of these cells. An inability to recognize familiar objects, such as faces, letters, words, etc., is one of the prominent symptoms of disease of the occipital region, provided the eyes are capable of performing their normal functions. Colored perceptions of objects and other ocular spectra often accompany irritation of these lobes. If the whole of the occipital lobe be not destroyed, the unimpaired part may slowly accumulate new sight memories, and the sense of vision may thus be slowly regained. This has been proven upon the dog by Munk.

5. That part of the *parietal lobes* which is not occupied by special centres of motion is probably associated with the conscious perceptions of various tactile impressions and the associated memories of touch, temperature, degrees of pressure, and pain.

6. The *temporal lobes* are the probable seat of our conscious appreciation of sounds, odors, and taste (Ferrier, Kussmaul, Gudden, and others). When these lobes are diseased, the memory of spoken words may be obliterated, and hallucinations of hearing or deafness may be developed. I once encountered an interesting case where hallucinations of smell (imaginary odors) existed in consequence of disease involving the apex of this lobe. Persons who have been suddenly deprived of their ability to appreciate a question when spoken, but who would reply promptly to the same question if written before their eyes, have been reported. In such the memories of sound have been obliterated by disease of the temporal lobe, but the memories of the form and meaning of letters have remained intact, because the occipital lobes were not involved. These patients can sometimes be made to repeat mechanically word upon word, in a parrot-like way, but the memory of their meaning has gone forever.

7. The *power of speech* (when regarded as a merely *mechanical* performance) seems to be governed by the inferior frontal convolution and the area adjacent to it around the lower part of the fissure of Sylvius.

* Ferrier originally placed the visual centres in the *angular convolution* of the parietal lobe (Fig. 4). I am led to believe that this is an error. This seems to be proven by an analysis of cases collected and published by Starr and Seguin. Wernicke has also lately shown that the visual fibres pass beneath the cortex of the angular gyrus in order to reach the occipital cortex. This discovery helps to explain the effects of destruction of the angular gyrus upon sight, as observed by Ferrier, Dalton, and others. Sight was destroyed by these observers, probably, by damage done to tracts of fibres lying beneath the cortex, rather than by a destruction of the cortical cells alone.

(Fig. 24.)* But it must be remembered that our remarks are usually called forth by some form of excitation, such as a spoken question, an impression upon the eye, or some form of irritation of the sensory nerves, as in the case of pain, tickling, etc., for example. Disease of this limited area of the brain surface causes patients to frequently interpolate wrong words in conversation, in spite of the fact that they grasp the meaning of all that transpires about them, and have the memories of past events perfectly at their command. Such a subject could write a reply to any spoken or written question with perfect accuracy, although he might speak it incorrectly. If he were asked to repeat words selected as a test of coördinated movements of the tongue and lips, he would probably fail to do so with his accustomed facility. This subject will be discussed in subsequent pages.

8. That we are endowed with *memories of muscular movements* is well illustrated by a case observed by Professor Charcot, of a gentleman who was rendered incapable, by disease of his brain, of recognizing either printed or written language, but who could grasp the meaning of both with ease by tracing out the curves with his fingers. The habit of writing had impressed the mind with the symbols of thought, through the agency of the muscles.

9. Some collections of cells within the deeper parts of the brain (the corpus striatum and optic thalamus of each cerebral hemisphere) are probably *distributing centres* for all impulses that pass either to or from the cerebral cortex.

They act as "middle-men," as it were. They are capable, as illustrated in the case of the mutilated frog previously referred to, of an automatic control over movements; but, as far as we know, there is no reason to think that they are associated in any way with the attribute of consciousness.

10. The functions of the cerebellum, the pons Varolii, and the medulla oblongata (see Fig. 1) are too complex to be fully discussed here. Their cells are called into action in a reflex manner, rather than by volition. There is reason to believe that the cerebellum is an "informing depot" for the cerebrum (Spitzka), and a "store-house for nerve force" (Mitchell). The medulla oblongata presides over acts that are chiefly outside of the domain of the will; such as the beating of the heart, the worm-like movement of the intestine, the regulation of the calibre of the

* Destruction of the centre of Broca and the island of Reil, seems to deprive the individual of those memories which are associated with the *proper coördination* of the apparatus of speech. Such patients cannot pronounce words which they may be able either to recognize by sight or to understand perfectly when spoken. The substitution of wrong words in conversation (paraphasia) is more commonly encountered than true ataxic aphasia when the island of Reil is involved. This subject will be more fully discussed later.

blood-vessels to the wants of the different organs, the modifications of blood-pressure, and other functions that are essentially vital.

THE NERVE FIBRES.

We now come to the second anatomical element of nervous tissues. If we pull a brain apart so as to expose its central portions, we shall be able to see that distinct bundles of extremely delicate white threads compose each "crus cerebri," or the leg of the hemisphere (Fig. 1), and that the thousand filaments which form each bundle diverge within the hemisphere and pass to its surface. We have grounds for the belief that each of these threads becomes united to a cell.

These are the nerve fibres. Each of these threads is insulated by a protective covering so as to prevent the diffusion of its currents to other fibres. The white substance of the brain is composed exclusively of fibres.

Of those that constitute the central portion of the cerebrum, one set serves to connect the cells of different areas of the cortex of each hemisphere (the "*associating fibres*"). These do not cross the mesial line of the skull. They allow of comparison of different memories, etc., and are probably essential to the *higher mental faculties*. The areas of sight, hearing, smell, motion, general sensibility, and taste, of each cerebral hemisphere, are thus brought into communication with each other. These fibres will be discussed at a greater length in connection with aphasia.

A second set serves to join the cortical cells of homologous parts of the two hemispheres of the cerebrum. They are evidently designed to promote a simultaneous action of the two hemispheres upon corresponding parts of the body, as illustrated in rowing a boat with two hands, swimming, etc. These are called "*commissured fibres.*" (Fig. 6.)

A third set comprises those fibres that pass from each hemisphere into the spinal cord. These are known as the "*peduncular fibres*," because they help to form the stem of the brain, or the crus cerebri (see Figs. 1 and 6).

A fourth set may be said to comprise those fibres that are associated directly with the *organs of special sense*, the nose, eye, ear, tongue, and skin. Some of these belong to the peduncular group.

Finally, a fifth set, known as the *fornix*, serves to connect the cortical cells of the temporal lobe of each cerebral hemisphere with a mass of cells buried deeply within the corresponding hemisphere, known as the optic thalamus. The function of these peculiarly arranged fibres is not yet determined with positiveness.

We have already discussed the *rôle* which the nerve fibres play in connection with the brain cells. They are the channels of transmission

of nerve impulses. Some carry impressions of a sensory character; hence their currents travel from peripheral parts to the cells of the brain. Others convey motor impulses from the brain cells to the muscles.

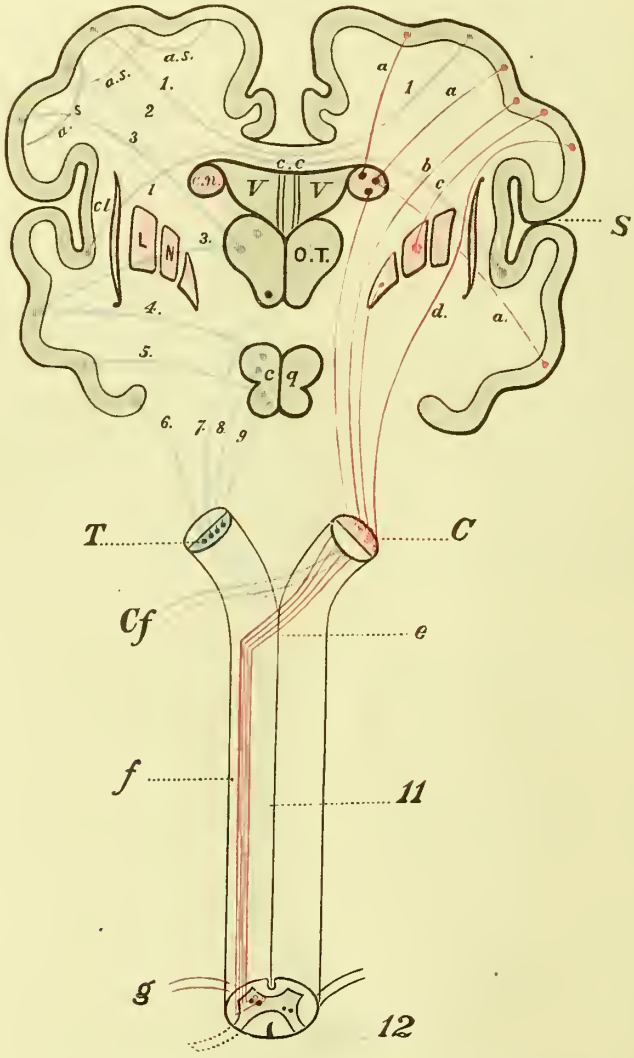


FIG. 6.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE GENERAL ARRANGEMENT OF THE FIBRES OF THE CEREBRO-SPINAL SYSTEM. (Modified from Landois.) The ~~shaded~~ portions represent the collections of gray matter. On the left side of the diagram, the *sensory fibres* of the crus are traced upward from the spinal cord to different portions of the cerebrum; on the right side, the *motor fibres* are similarly represented. Numerals are used in designating the *sensory* and *commissural fibres;* the *motor fibres* are lettered in small type. The cortical layer is shown at the periphery of the cerebral section, with commissural fibres (1) connecting homologous regions of the hemispheres, and associating fibres (*a.s.*) connecting different convolutions of each hemisphere. *c.n., Caudate nucleus* of the

CORPUS STRIATUM; L. N., *lenticular nucleus* of the same; O. T., OPTIC THALAMUS of each hemisphere, united to its fellow in the median line; *c. g.*, CORPORA QUADRIGEMINA; *c. l.*, CLAUSTRUM, lying to the right of the letters; *c. c.*, CORPUS CALLOSUM, with its commissural fibres; *S*, FISSURE OF SYLVIUS; *V*, LATERAL VENTRICLE, the fifth ventricle being shown between the two layers of the *septum lucidum*; *C*, the *motor tract* of the CRUS CEREBRI (*basis cruris—crusta*); *T*, the *sensory tract* of the CRUS CEREBRI (*tegmentum cruris*); *Cf*, the *cerebellar fasciculus*; *e*, the point of decussation of the motor fibres of the spinal cord; *f*, the course of the *decussating motor fibres* of the spinal cord below the medulla, showing their connection with the cells of the anterior horns of the gray matter, and their continuation into the anterior roots of the spinal nerves (*g*); *a*, fibres which radiate through the caudate nucleus; *b*, fibres of the "*internal capsule*;" *c*, fibres which radiate through the lenticular nucleus; *d*, fibres of the "*external capsule*;" 2, 3, 4, 5, 6, 7, 8, 9, sensory fibres radiating from the tegumentum cruris to the cortex by means of various nodal masses of gray matter; 11, course of the sensory fibres of the spinal cord (shown by dotted lines), intimately connected with the posterior root of the spinal nerve (12), and decussating at or near to the point of entrance into the spinal cord. This diagram may be studied in connection with Figs. 12, 15, 16, 36, and 37, with possible benefit to the general reader. In this diagram, the *direct pyramidal fibres* are not shown (see Fig. 29), nor the gray matter of the pons.

Different observers have been able to trace the course and terminations of the separate bundles with exactness by means of methods lately discovered. Nature, under certain conditions, makes the dissections during life; and we, after death, can study out the details of her work. In this way we have learned facts that no human dissection could have determined. The discovery of Türck that nerve fibres degenerate (as a result of mal-nutrition) when severed from the nerve cells, enables us to investigate the results that follow destruction of certain limited areas of the cortex of man by disease or mechanical injury. When sections across such a brain are made and examined under a glass (proper staining reagents being employed) the area of the degenerated fibres becomes as clearly depicted from that of healthy brain fibres as would an ink spot upon a table-cloth. An examination of successive sections enables us to trace the course of the fibres that were originally connected with the cells of the diseased area to their peripheral connections. Some years after Türck's original paper, Flechsig opened another field of investigation. He showed that during the development of the embryo, certain bundles of nerve fibres in the brain and spinal cord became completely formed before others. By means of sections of embryotic brains, he and his followers have been able to confirm many of the facts made known to us by Türck's method. Finally, Gudden has lately proven that extirpation of the eye and some other organs, as well as the divisions of some nerve-tracts, in the newly-born animal, are followed by a proximal degeneration of the fibres connected with the organ removed.

Let me remark here that every nerve impulse sent to the brain *does not travel along a continuous fibre* to reach the cell of the cortex that is capable of receiving it; and the same holds true of all motor impulses dispatched from the brain to the muscles. All impulses are *passed from cell to cell* by means of connecting fibres. In this way they eventually reach the cerebral cortex, just as water-buckets are passed up a ladder, in case of fire, to use an illustration borrowed on account of its aptness. The object of this arrangement is to allow of an independent action of certain collections of cells (that are subservient to the cortical cells of the cere-

brum) in case the required response does not necessitate volition or consciousness. Many of the vital processes (such as the beating of the heart) are governed by what is known as "reflex action." We cannot check them by the will, and, as a rule, we are unconscious that they are constantly going on.

THE GANGLIA AT THE BASE OF THE CEREBRUM.

Buried within the substance of each cerebral hemisphere, isolated gray masses (composed of nerve-cells) exist. They may be revealed by vertical or horizontal cross-sections of the hemispheres.

Among these may be prominently mentioned: (1) the *caudate* and *lenticular nuclei* of the *corpus striatum* (so named from the striped appearance which they present); (2) the *optic thalamus* (a term which signifies the "bed" of the optic fibres); (3) the *geniculate bodies*, connected with the optic tracts (Fig. 21); (4) the *amygdalæ*, each being formed by the tail-like prolongation of the caudate nucleus of the corresponding hemisphere (Fig. 9); and (5) the *basal ganglia of Meynert*.

The limits of this chapter will preclude more than a hasty and very imperfect summary of the functions of the corpora striata and the optic thalami.

In the *Journal of Nervous and Mental Diseases*, I published some years ago two lectures delivered by me upon these ganglia. In some respects, I have changed my views relating to a few disputed points concerning the structure and probable functions of these bodies since these lectures were published. I shall quote, however, some paragraphs from these articles from time to time, with modifications in the phraseology.

THE CORPUS STRIATUM.

Within each cerebral hemisphere, two nodal masses of cells are imbedded, known as the corpus striatum and the optic thalamus (Fig. 1). Because these bodies lie near to the base of the cerebrum, they are collectively called the "*basal ganglia*" of the hemispheres.

Each corpus striatum is divided (by the fibres which constitute the so-called "internal capsule" of each hemisphere) into two distinct portions; one of which projects into the lateral ventricle, while the other does not. These are known as the *intra-ventricular* portion, or the "*caudate nucleus*," and the *extra-ventricular* portion, or the "*lenticular nucleus*." Fig. 6 will make this apparent to the reader.

The *two nuclei of the corpus striatum become joined* both anteriorly and posteriorly; hence the separation of these masses is only partial. Horizontal and vertical cross-sections of the cerebrum show these nuclei as distinct from each other, as a rule.

Space will not allow of an anatomical description of these bodies.

I quote, therefore, a few paragraphs from two monographs of mine, relating to these nuclei :—*

"The clinical results of lesions of either nucleus are attributed by

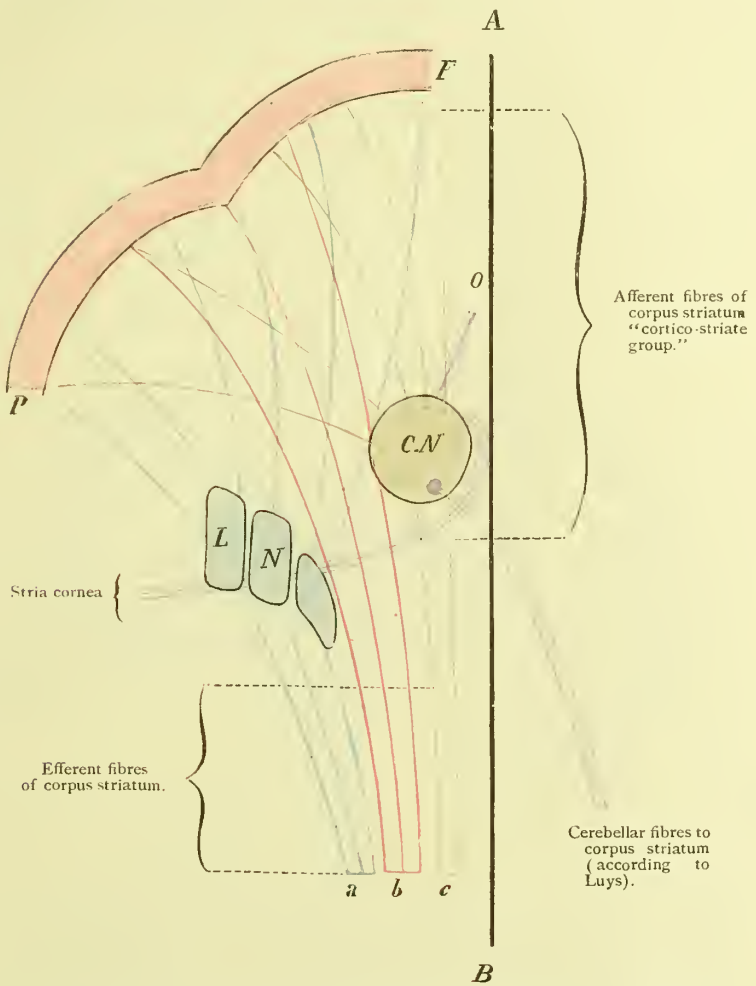


FIG. 7.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE AFFERENT AND EFFERENT FIBRES OF THE CORPUS STRIATUM. *C. N*, "caudate nucleus," or ventricular portion of corpus striatum; *L, N*, "lenticular nucleus," or extra-ventricular portion of corpus striatum; *A—B*, median line, separating cerebral hemispheres; *P—F*, psycho-motor regions of the cortex; *a*, peduncular fibres connected with *L, N; b*, fibres of the so-called "internal capsule;" *c*, fibres connected with *C. N; O*, olfactory fibres. (Luys.)

most authors to pressure effects upon the motor fibres of the internal capsule. In no instance, to my knowledge, has the destruction of these nuclei produced psychic manifestations.

* *Journal of Nervous and Mental Diseases*, 1883.

The *hemiplegia*, which follows injury to the corpus striatum, is confined chiefly to the side opposite to the lesion; in cases of extreme rarity, paralysis of motion on the same side has been clinically recorded. Flechsig has proved that such cases are to be interpreted as the result of an individual peculiarity in the relative number of decussating and direct pyramidal fibres (Fig. 29).

"The corpus striatum, like the optic thalamus, may possibly (as Luys suggests) be considered, as a territory in which cerebral, cerebellar, and spinal activities are brought into intimate communication. It probably acts as a 'halting place for voluntary motor impulses' emitted from the cerebral cortex. It enables these impulses to 'become modified and possibly reinforced by currents derived from the cerebellum; and, by its efferent fibres, it transmits centrifugal motor impulses along the projection system to different groups of cells within the spinal gray matter, whose individual functions they tend to evoke.'"

Luys states that this ganglion probably acts as a condenser and modifier of all motor acts which are the result of volition; and manifests, through the agency of its satellites (the cells of the anterior horns of the gray matter of the spinal cord), the outward expressions of our personality. Without the influence of the cerebral hemispheres, it is also capable, by means of cerebellar innervation, of governing all the complex muscular movements required in maintaining equilibrium (coördinated movements). Finally, it may be presumed to "possess the power of analysis of cerebral and cerebellar currents received simultaneously, and of materializing them by the intervention of its nerve-cells, projecting them in a new form, amplified and incorporated with the requirements of the general organism."

Experiments made upon the caudate and lenticular nuclei can hardly be said to have afforded results which can be made the basis for positive deductions respecting the functions of each. Nothnagel employed injections of chromic acid into the substance of each, and also destroyed them by means of an instrument devised for that purpose, but he arrived at no positive conclusions, save that the lenticular nucleus seemed to have a more decided influence upon motion than the caudate nucleus, when the nuclei of both sides were simultaneously destroyed. Observations in comparative anatomy seem to show a relationship of the caudate nucleus with the fibres of the leg and of the lenticular nucleus with those of the arm.

Some observers claim to have destroyed the entire ganglion without any marked disturbance of sensory or motor phenomena. Collected cases of lesions confined to either nucleus fail to show that any permanent symptoms have been produced which are diagnostic of such lesions.

THE OPTIC THALAMUS.

Efforts have been made by some of the later anatomists and physiologists who have specially investigated the brain, to subdivide the gray matter of the thalamus into circumscribed masses or nuclei, and to trace the fibres which appear to arise from these nuclei to special regions of the brain and spinal cord. Among the most attractive of these attempts may be mentioned that of Luys, whose views will be subsequently given in detail. Whether clinical research and physiological experiment will confirm all of these attractive theories, and place them upon a ground as worthy of credence as the deductions of Broca, Munk, and Ferrier regarding the functional attributes of other parts of the brain, time alone

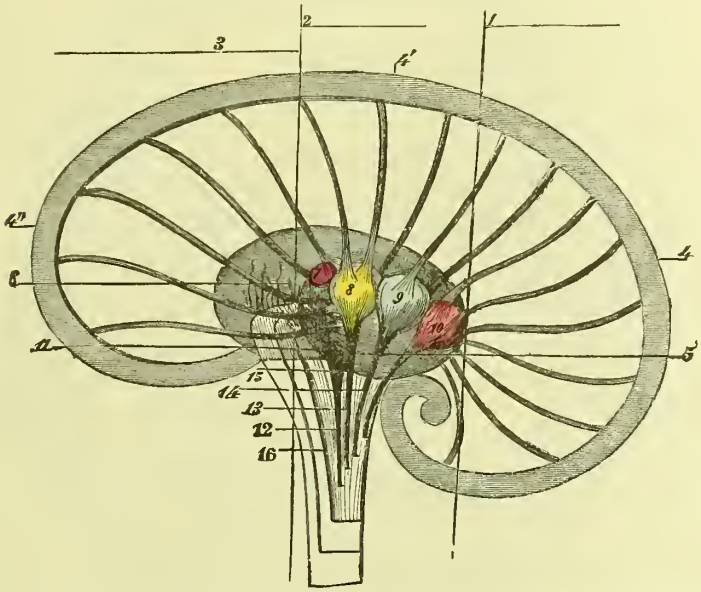


FIG. 8.—A DIAGRAM OF THE NUCLEI OF THE OPTIC THALAMUS AND THE CONVERGING FIBRES ASSOCIATED WITH THEM. (After Luys.) 1, converging fibres of posterior convolutions; 2, same, of middle convolutions; 3, same of posterior convolutions; 4, 4', 4'', cortical periphery as related to the central gray masses; 5, optic thalamus; 6, corpus striatum; 7, anterior (*olfactory*) centre; 8, middle (*optic*) centre; 9, median (*sensitive*) centre; 10, posterior (*acoustic*) centre; 11, central gray region; 12, ascending gray fibres of visceral innervation; 13, gray optic fibres; 14, ascending sensitive fibres; 15, ascending acoustic fibres; 16, series of antero-lateral fibres of the spinal axis going to be lost in the corpus striatum.

can decide. They are opposed to many of the conclusions of Meynert, Flechsig, Wernicke, Spitzka, Starr, and others.

"According to the researches of Luys, four isolated ganglions may be demonstrated in the thalamus. Arnold, in common with some other anatomists, has recognized three of these, and the fourth is now added by the author quoted. This author states that these ganglia are arranged in an antero-posterior plane, and form successive tuberosities upon the thalamus, giving that body the appearance of a conglomerate gland.

"*The anterior ganglion* of Luys (*corpus album subrotundum*) is especially prominent. It is said by this author to be developed in animals in proportion to the acuteness of the sense of smell. By means of the 'tænia semi-circularis,' this ganglion (according to this author) may be shown in the human species to be connected with the roots of the olfactory nerve. Respecting it, he says: 'Direct anatomical examination shows that there are intimate connections between the anterior centre

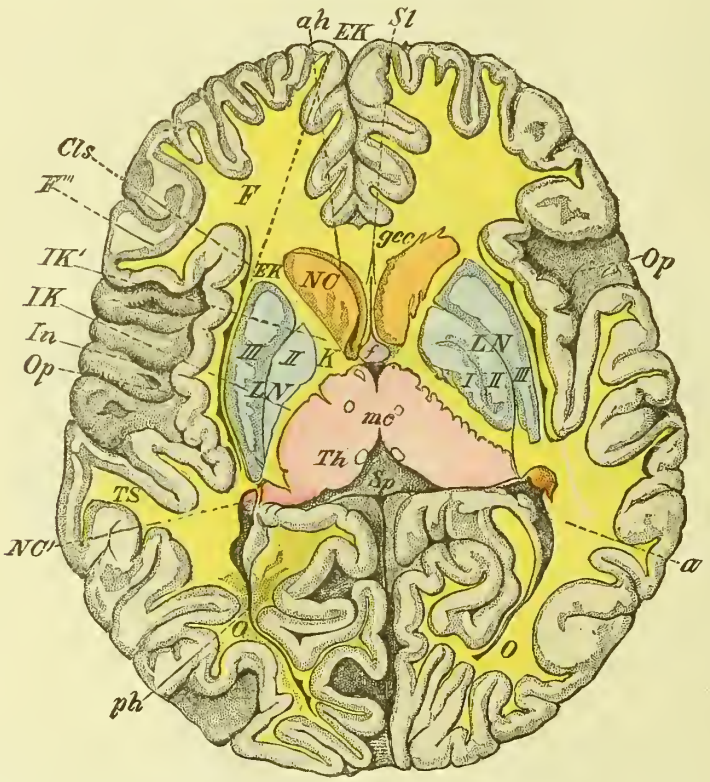


FIG. 9.—A TRANSVERSE SECTION OF THE HUMAN BRAIN FROM BEFORE BACKWARD. (After Flechsig.) *N C, caudate nucleus; N C'*, the *tail* of *N C*, cut across (the *amygdala*); *L N, lenticular nucleus*, with its three subdivisions (*I, II, III*); *TH, optic thalamus; F*, frontal lobe; *TS*, temporal sphenoidal lobe; *O*, occipital lobe; *Cls, claustrum; IK, thalmo-lenticular portion of internal capsule; K, knee* of same; *IK', caudo-lenticular portion* of same; *EK, external capsule*, lying between the lenticular nucleus and the claustrum; *f, fornix* cut across; *In, insula*, or *island of Reil; Op*, depth of Sylvian fissure beneath the operculum; *mc*, middle commissure of the thalamus; *ph*, posterior horn of lateral ventricle; *ah*, anterior horn of same; *Sl*, septum lucidum.

and the peripheral olfactory apparatus. On the other hand, in confirmation of this, in the animal species, in which the olfactory apparatus is very much developed, this ganglion itself is proportionally very well marked. Analogy has thus led us to conclude that this ganglion is

in direct connection with the olfactory impressions, and that this marks it as the point of concentration toward which they converge before being radiated toward the cortical periphery.'

"The *second or middle centre* is in apparent continuity, according to Luys, with the fibres of the optic tract. He considers it on the same grounds as those previously quoted respecting the anterior centre, as a seat of condensation and radiation of visual impressions.* There seems to be undisputable grounds for the belief that the thalamus, the outer geniculate bodies, the anterior corpora quadrigemina, and the cortex of the occipital lobes are, in some way, associated with the perceptions afforded by the retina. (Munk, Wernicke, Monakow, and others.)

"We know that extirpation of the eye is followed by more or less complete atrophy of the outer geniculate body of the opposite side, although the inner geniculate body seems to remain unaffected. The experiments of Longet, who destroyed the optic thalami upon both sides without being able to note any impairment of vision, or influence upon the movements of the pupil; and those of Lussana and Lemoigne, who found that blindness of the opposite eye followed unilateral destruction of the thalamus, may suggest the possibility, in the former, of the escape of this centre, and, in the latter, its destruction. It is difficult to devise any experiment which will positively settle the bearings of the thalamus upon vision; because it is almost impossible to destroy special portions with accuracy, or if this were insured, to avoid injury to adjacent structures. Fournié claims to have effected the separate annihilation of the special senses of smell and vision by injections made into different parts of the thalamus of animals; and his experiments, if subsequently verified, will tend to confirm some of the theories advanced by Luys.

"Ritti has pointed out that irritation of the thalamus may play an important part in the *development of hallucinations.*

"The *third centre* ('median ganglion' of Luys) is described as about the size of a pea, and situated mathematically in the exact centre of the thalamus. To it, the discoverer ascribes the function of presiding over and condensing all *sensory impressions.*

"The *fourth posterior centre* is stated to act as a halting place and condenser of *auditory impressions.* Two instances where the brains of deaf mutes were found to present a localized lesion of this centre are reported by Luys.

"The views here expressed are quoted on account of their originality; and because the author of them ranks high as an authority upon

*Luys states that it is scarcely visible in those animals (the mole as an example) where the optic nerves are rudimentary. The view is now more generally accepted that the posterior tubercle of the thalamus (the *pulvinar*) is functionally associated with the optic fibres.

the subject of which he speaks. The numerous cases of cerebral hemorrhage which have been reported, where the thalamus was apparently the seat of localized injury, are too often accompanied with a clinical history which points toward pressure upon the internal capsule, to be of value as confirmatory evidence of the existence of special centres in the thalamus.* The effort of Luys to adduce cases of hemianæsthesia in support of his views regarding the function of the 'median centre' of the thalamus, merely because a lesion of that ganglion was found in an area defined by him as the normal limits of that special centre, must not be deemed conclusive; because the same effect *might* have been produced by pressure upon the fibres within the *posterior third* of the internal capsule of the cerebrum. There is reason to hope, and possibly to be-

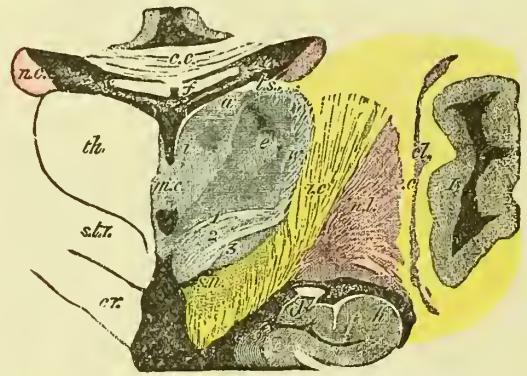


Fig. 10.—Section Across the Optic Thalamus and Corpus Striatum in the Region of the Middle Commissure. (Shäfer after a preparation by Mr. S. G. Shattuck.) Natural size. *th.*, thalamus; *a.*, *e.*, *i.*, its anterior, external, and internal nuclei respectively; *w*, its latticed layer; *m. c.*, middle commissure; above and below it is the cavity of the third ventricle; *c. c.*, corpus callosum; *f*, fornix, separated from the third ventricle and thalamus by the velum interpositum. In the middle of this are seen the two veins of Galen and the choroid plexuses of the third ventricle; and at its edges the choroid plexuses of the lateral ventricles; *t. s.*, tæniæ semicircularis; *cr.*, forward prolongation of the crusta passing laterally into *the internal capsule*, *i. c.*; *s. t. r.*, subthalmic prolongation of the tegmentum, consisting of (1) the dorsal layer, (2) the zona incerta, and (3) the corpus subthalamicum; *s. n.*, substantia nigra; *n. c.*, nucleus caudatus of the corpus striatum; *n. l.*, nucleus lenticularis; *e. c.*, *external capsule*; *cl.*, claustrum; *I.*, island of Riel.

lieve, that sooner or later isolated ganglia within the optic thalamus will be demonstrated to exist by normal and pathological anatomy, as well as by physiological experiment; but the conclusions even of so prominent an author should not be fully accepted without further testimony to substantiate their accuracy. Some of the later observations respecting the optic fibres, seems to disprove the view of Luys.

"A few interesting cases have, however, been brought forward, which certainly seem to sustain the views advanced. A case reported by Hun-

*If permanent symptoms remain after a lesion of the thalamus is suspected to exist, the *internal capsule* is probably indirectly involved. (See subsequent pages relating to the internal capsule.)

ter,* where a young woman successively lost the senses of smell, sight, sensation, and hearing, and who gradually sank, remaining a stranger to all external impressions, disclosed at the autopsy a fungus hæmatodes which had gradually destroyed the optic thalamus of each side, and the optic thalami alone, if the drawing given is reliable. Again, Fournié's experiments on living animals points strongly to the existence of localized centres in the thalamus. Three instances of unilateral destruction of smell, observed by Voisin and reported by Luys, have been found to be associated with a destruction of the anterior centre of the thalamus. A hemorrhagic effusion into the thalamus, on a level with the soft commissure (the situation of the optic centre of Luys), produced (in the experience of Serres) a sudden loss of sight in both eyes. Later observations seem, however, to point toward a relationship between the posterior extremity of the thalamus (the '*pulvinar*') and the optic fibres.

"Ritti's paper upon the effects of irritation of the thalamus upon the development of hallucinations, lends strength to the view that that ganglion in some way regulates the transmission of sensory impressions of all kinds to the cerebral cortex; and confirms the opinion that 'the optic thalami are to be regarded as intermediary regions which are interposed between the purely reflex phenomena of the spinal cord and the activities of psychial life.'

"The view taken by Lussana and Lemoigne, that the optic thalami contained motor centres in animals for the lateral movements of the forelimbs of the opposite side, seems to be completely overthrown by pathological statistics in the human race. The results obtained by these experimenters are also at variance with the belief, which has now become general among neurologists, that the thalami are intimately connected with the sensory tracts of the cerebrum and cord; since they concluded that no evidence of pain or any loss of sensibility resulted from injury to these bodies.

"The effects of all experiments on animals, however, agree entirely with the general experience of pathologists, that lesions of both the thalamus and corpus striatum produce results upon the opposite side of the body; whether the symptoms produced point to a disturbance of the kinesodic (motor) or æsthesodic (sensory) tracts. The view originally advanced by Carpenter and Todd, that the thalami are concerned in the upward transmission and elaboration of sensory impulses, in contradistinction to the corpora striata, which are concerned in the downward transmission and elaboration of motor impulses, seems to be gaining ground, and many facts may be urged in its favor."

The experiments of Monakow on rabbits lead him to views not entirely dissimilar to those advanced by Luys. He places the cortical

* *Medico-Chirurg. Trans.*, London, 1825, vol. xiii.

connections of centres in the thalamus, somewhat differently, however, from the conclusions already mentioned. According to this observer, the posterior tubercle (the *pulvinar*) is related to the visual tracts, as is also the external geniculate body; the internal geniculate body is related to the auditory fibres, and the cortical centres of hearing in the first temporal convolution; and the anterior tubercle and median nucleus are related to the frontal lobes.

The *pillars of the fornix* seem to unite the thalamus with the cortical centres of smell and taste; and, according to Ferrier, with the cortical centres of tactile sensibility.

Hemichorea and hemiathetosis have been observed in connection with lesions of the thalamus; but they must, to my mind, be regarded as an evidence of irritation of the motor fibres of the internal capsule (which lie closely adjacent to the thalamus). Fig. 9 will make this relationship clear to the mind of the reader.

THE CAPSULAR FIBRES OF THE CEREBRUM.*

Vertical and horizontal cuts made through the cerebrum exhibit a well-defined tract of fibres in each hemisphere which separates the *lenticular nucleus* from two other gray masses of the same hemisphere, viz., the *caudate nucleus* and the *thalamus*.

This tract of fibres (inclosed between these nodal masses of cells) is termed the "*internal capsule*," because it bounds the lenticular nucleus on its mesial aspect. A similar tract of fibres also separates the lenticular nucleus from the "claustrum" of the same hemisphere. This is known as the "*external capsule*" (Figs. 7 and 9).

The fibres, which form the "internal" and "external capsule" of each hemisphere, seem to pass through the substance of the cerebrum without any *structural relationship with the cells of the caudate-* or *lenticular-nuclei*, or *the thalamus* (Flechsig). In this respect they differ from all other fibres which serve to connect the cells of the cerebral cortex with collections of nerve cells outside of the cerebrum.

From a physiological and anatomical standpoint, the fibres of the internal capsule possess greater interest than many other bundles. Late researches have shown that it contains (1) the so-called "*pyramidal fibres*" (the "*will tract*" of Spitzka) which controls voluntary movements of the limbs; (2) the so-called "*sensory tract*," whose fibres convey sensations of all kinds from the surface of the body to the cells of the cerebral cortex where they can be appreciated by consciousness; (3) the so-called "*speech tract*," whose fibres allow of communication between the "speech area" of the cortex and the nuclei of origin of the seventh, tenth,

* This term is a misnomer. These fibres form a capsule to the "lentieular nueleus," properly speaking, and not to the eerebrum.

eleventh, and twelfth cranial nerves (within the medulla); (4) the motor fibres of the face; (5) bundles of fibres connected with the special senses (sight, smell, hearing, taste, and touch); (6) the so-called "*hypoglossal tract*," which connects the cortical centre for movements of the tongue with the nucleus of origin of the twelfth cranial nerve within the medulla; finally, many other bundles of fibres (whose functions are not yet determined) occupy the anterior part of the capsule in front of its "knee." (Fig. 9).

If a horizontal cut be made through the substance of the cerebrum at a level which shall include the basal ganglia (the caudate nucleus, the lenticular nucleus, and the thalamus of each hemisphere) in the plane of the section, we shall see that the outline of the internal capsule is marked by an angle, termed the "genu" or "knee" of this capsule. This is shown in the cut of Flechsig (Fig. 9), and also in another which is introduced later. A subsequent diagram offered as explanatory of some of the results of cerebral hemorrhage may be consulted in this connection with benefit to the reader.

That portion of the internal capsule which lies between the lenticular and caudate nuclei has been named the "*caudo-lenticular*" portion (Spitzka). It lies anteriorly to the "knee."

The part posterior to the "knee" has been termed by the same author the "*thalamo-lenticular*" portion.

In studying the cut of Flechsig (Fig. 9) it must be remembered (1) that the fibres which constitute the internal capsule pass vertically to the plane of the section; (2) that, above the upper limits of the basal ganglia, they radiate to different areas of the cerebral cortex; (3) that the component fibres of the capsule may be subdivided into groups. The functions of some have been quite positively determined of late. This has been done by a study of their apparent cortical distribution, and of secondary degeneration of special nerve tracts; by an analysis of the symptoms produced during life, when well-defined lesions of the internal capsule have existed; by the so-called "development method" of Flechsig; and by the effects of section of nerve tracts in newly-born animals (Gudden's method).

The following statements seem to be now quite well established, and to be of service as clinical guides to the localization of cerebral lesions which directly involve or create pressure upon these fibres.

(1) The "*caudo-lenticular*" *portion* is composed of fibres whose function is imperfectly understood. They seem to pass chiefly to the cortex of the frontal lobes.

(2) In the *region of the* "*knee*," the "thalamo-lenticular" portion contains the motor fibres of the face.

(3) Posterior to the facial tract lie the fibres of the so-called "*pyra-*

midal motor tract" or the "*will tract.*" Those of the arm probably lie anteriorly to those destined for the leg. Lesions affecting this part would create chiefly a hemiplegia of the opposed side. These fibres constitute the anterior pyramid of the corresponding half of the medulla oblongata; hence the name which is commonly applied to them.

(4) Posterior to the general motor fibres, we encounter the so-called "*sensory tract.*" A lesion of the entire bundle would induce *hemianæsthesia* of the opposed side.

(5) Next in order from before backward, the fibres of the "*speech tract*" are supposed to pass (Wernicke). A lesion confined to this bundle would produce aphasic symptoms.

(6) Finally, the *optic fibres* pass through the extreme posterior part of the capsule. Lesions of this bundle cause "homonymous hemianopsia" (the blindness being confined to the *right lateral half of each eye* if it affect the *right capsule*, or *vice versa*).

(7) The course of the fibres associated with the *special senses of taste and smell* is not yet as positively determined, as in the case of the optic and auditory fibres. The *auditory tract* probably passes through the lower and posterior part of the internal capsule.*

(8) The *hypoglossal cerebral tract* probably joins the lower third of the precentral gyrus (ascending frontal convolution) with the medullary nucleus of the twelfth cranial nerve by passing through the region of the "knee" of the internal capsule, anteriorly to the motor fibres which govern the limbs (Raymond and Artaud). Lesions of this tract produce symptoms closely allied to those of Duchenne's disease ("bulbar paralysis"—"glosso-labio-laryngeal paralysis").

The outlines of the surfaces of the thalamus and the lenticular nucleus of the corpus striatum, as seen in all vertical sections of the cerebrum, may be roughly compared to the form of a square, whose two halves are defined by a diagonal band (the "*internal capsule*,"†) running from the upper and outer corner to the lower and inner corner. These halves correspond to the respective ganglia. It may be worthy of remark, in this connection, that the surface of the thalamus, which lies in contact with the internal capsule of the cerebrum marks the central or receiving pole for the fibres, which join it with the cortex of the cerebral lobes. This is not the case with the lenticular nucleus.

*Spitzka, in a late article, states his conclusion that sound is transmitted from the cochlea through the following structures to reach the cortical centres of hearing. 1, the posterior division of the eighth pair; 2, the trapezium of the same side—where the auditory fibres cross to the opposite side; 3, a part of the lemniscus; 4, the posterior pair of the corpora quadrigemina; 5, the internal geniculate body; 6, the corona radiata; 7, the cortical centres in the superior temporal gyrus. (See also foot-note on page 42.)

† This bundle of fibres ceases with the posterior limits of the *lenticular nucleus* of the corpus striatum.

The external surface of the thalamus (which lies in contact with the *internal capsule* of the cerebrum) presents a peculiar appearance, which has given it the name of "lattice layer" (Kölliker). All along this surface, radiating fibres pass out of the thalamus to become intermingled with the fibres of the internal capsule, and to be distributed to the cerebral cortex. Those from the front of the ganglion pass to the frontal lobe; those from the middle are distributed to the posterior part of the frontal and to the parietal and temporo-sphenoidal lobes; those from the posterior part can be traced to the temporo-sphenoidal and occipital lobes. From the region of the pulvinar, or posterior tubercle, fibres can be traced into the optic tract.

The internal capsule will be considered in its clinical aspects in subsequent pages of this section, and also in connection with the symptomatology of cerebral apoplexy. Diagrams will then be given which will help to make the subject clear to the reader.

THE CRUS CEREBRI.

The stem of each cerebral hemisphere (Fig. 1) is composed of fibres that serve to connect the cells of some of the component parts of each cerebral hemisphere with other cells, situated either within the *crus* itself, the *pons Varolii*, the *cerebellum*, the *medulla oblongata*, or the *gray matter of the spinal cord.* These fibres are therefore of different lengths.

The shortest fibres of the crus probably terminate in the gray matter of the crus (substantia nigra of Sœmmering, and the red nucleus of Stilling, see Fig. 11). The longest fibres terminate in the lowest segments of the spinal cord.

The diagram of a cross-section of the crura cerebri, introduced at this point, will make some of the most essential points in their architecture apparent. It shows the gray masses of each crus; and also the situation of several different nerve tracts cut across.

The "*tegmentum cruris*" lies posterior to the substantia nigra. It contains the fibres designed for *sensory* conduction to the brain; and, possibly, a few motor filaments.

The "*crusta cruris*" (basis cruris) lies in front of the substantia nigra. It is chiefly composed of motor fibres.

The *fibres of the third cranial nerve* (motor oculi) traverse the crus (in the plane of the section), from the nucleus of that nerve in the gray matter around the *Sylvian aqueduct* (iter e tertio ad quarto ventriculo) to its point of exit.

The fibres of the *pyramidal tracts* (see Figs. 12 and 29) occupy but limited area in the crusta cruris (as shown in the diagram).

The *red nuclei* are closely related to the fibres of the *superior*

*peduncles of the cerebellum** (processus e cerebello ad cerebrum), and the fibres of the third cranial nerve. Their function is not positively determined. Lesions of these bodies seem to create symptoms of incoördination of movement and paralysis of the third cranial nerve.

This diagram (Fig. 11) may aid the reader in mastering the grounds for many clinical deductions respecting lesions of the crus, which will be mentioned later in this section.

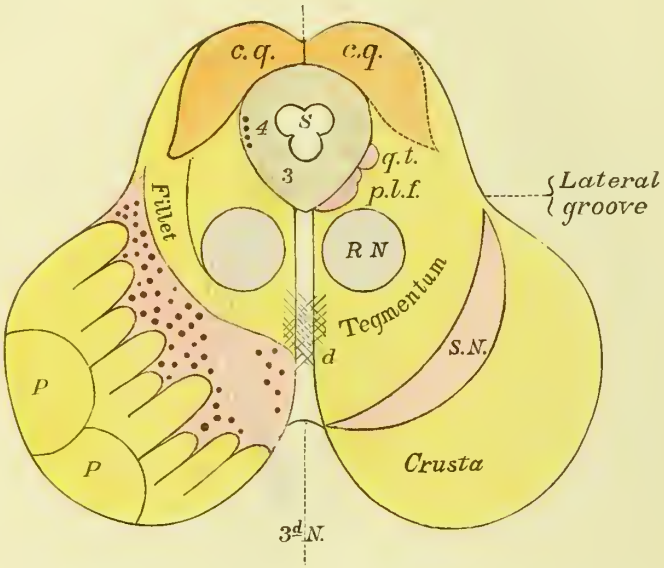


Fig. 11.—A Diagrammatic Representation of the Crura Cerebri in Cross-section. (After a blackboard drawing by the Author.) c. q., *corpora quadrigemina*; S, *aqueduct of Sylvius* (iter e tertio); *4, nucleus of the fourth cranial nerve* in the gray matter which surrounds the aqueduct; *3, nucleus of the third cranial nerve*, whose fibres are depicted; q. t., *root of fifth cranial nerve*; p. l. f., *posterior longitudinal bundle*; *R. N.*, the *red nuclei* of Stilling; *S. N.*, the *substantia nigra*; *P*, the portion of the "crusta" occupied by the *pyramidal fibres* (Fig. 29). The *tract of the fillet* or "lemniscus," as well as the relative situation of the "crusta" or "basis cruris," and the "tegmentum cruris" is also shown in the cut.

Respecting the formation of the *lemniscus tract*, Flechsig believes that the tract is composed of a very large bundle (which degenerates downward) and a smaller one (which degenerates upward).

This author thinks that the *larger bundle* arises from the outer body of the lenticular nucleus (Fig. 7), and he places its termination in the olivary body of the medulla. The *smaller bundle* is believed by this observer to start in the sensory decussation at the lower part of the medulla and to terminate in the corona radiata of the cerebrum.

*The termination of the fibres of the *superior cerebellar peduncle*, after their association with the red nuclei of the tegmentum (Fig. 14), is not yet determined. Flechsig believes that they go to the lenticular nucleus, or radiate in the corona radiata after passing through the thalamus. The cells of the "*substantia nigra*" are deeply pigmented, giving to it a blackish color.

Spitzka, who has written a very complete and lucid article upon this tract (see bibliography) differs from Flechsig in some of his conclusions. He traces the "olivary" bundle of Flechsig to a *spinal origin*, and the smaller bundle, described by Flechsig, beyond the sensory decussation to the *nuclei of the columns of Goll and Burdach of the opposite side.* These nuclei are shown in Fig. 37, which also illustrates Aeby's views respecting the "lemniscus tract."

The close proximity of the *corpora quadrigemina* to the tegmentum cruris, leads us to regard blindness, nystagmus, strabismus, and an absence of the pupillary reflex (which clinically mark a lesion of the *anterior pair*) as strongly diagnostic. From a similar train of reasoning, a marked disturbance in coördination would point to the fact that the *posterior pair* were involved, or that the *red nuclei* are diseased. A defective action of homologous branches of the third nerve of the two sides points strongly toward a lesion of the latter bodies.

THE PONS VAROLII.

This portion of the brain may be compared (as a homely illustration) to a collar around the crura, which helps to bind the cerebellar hemispheres together, and to tie them fast to adjacent parts.

As was the case with the crus, cross-sections made through the pons reveal (1) isolated gray masses (composed of nerve cells) in great abundance, and (2) distinct bundles of fibres. The direction of these fibres may be seen to be both horizontal and vertical.

The vertical bundles pass into the substance of the medulla and spinal cord, and are extended upward to the cerebrum.

The horizontal bundles probably serve two purposes: (1) to *connect the two cerebellar hemispheres;* and (2) to unite *each cerebellar hemisphere with the opposite cerebral hemisphere.*

The number of fibres which compose the crus is very largely in excess of that which exists within the medulla; hence we are justified in assuming (as Meynert first suggested) that many fibres derived from the cerebrum are deflected within the pons. A certain proportion only of the cerebral fibres is prolonged to the spinal cord. Some of those so prolonged are functionally associated with the transmission of *motor impulses* from the cerebrum to the muscles of the extremities,—the so-called "*pyramidal tracts*," because they form the "anterior pyramids" of the medulla. Others convey *sensory impressions* from the peripheral organs to the cells of the cerebrum,—the so-called "*sensory tracts.*"

The *gray matter of the pons* seems to be composed of cells that serve to interrupt the paths of communication between the cerebrum and cerebellum, and also between the two cerebellar hemispheres.

The association of the fibres which constitute the *middle peduncles of the cerebellar hemispheres* with the "cerebral tracts" (by means of the multipolar cells in the pons) is a crossed one—the left cerebellar hemisphere being joined to the right cerebral strand, and *vice versa*. This probably brings the cerebellar hemispheres into association with those motor fibres which act upon the corresponding limb; because the pyramidal fibres decussate to a great extent at the lower part of the medulla.

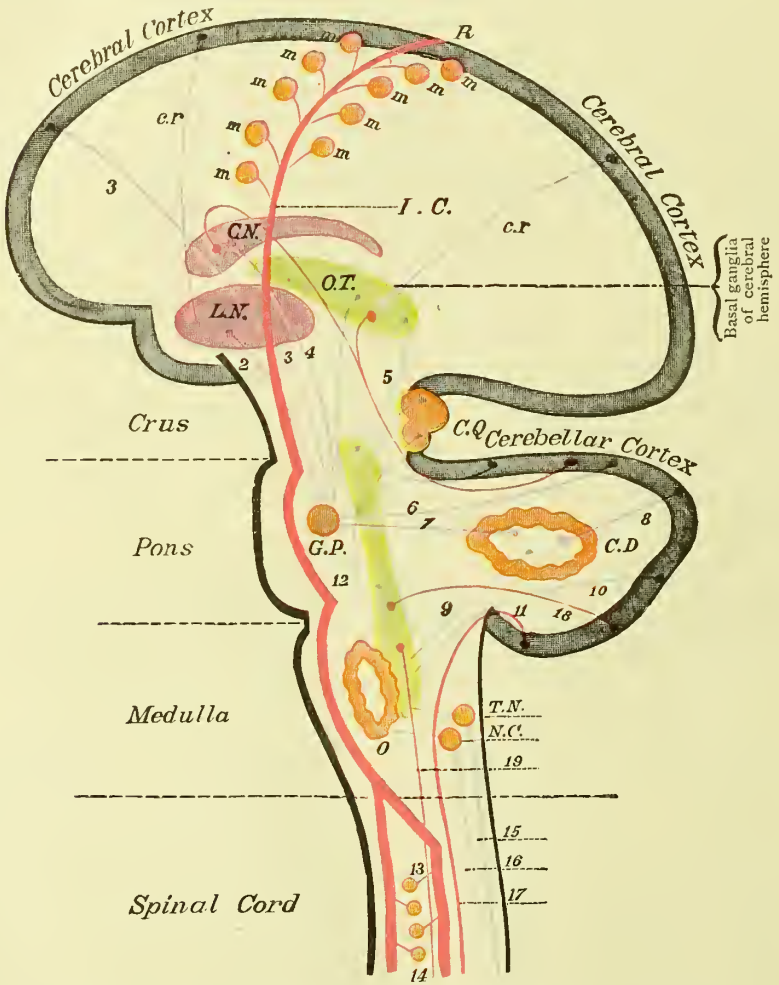


FIG. 12.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE COURSE OF CERTAIN NERVE-TRACTS WITHIN THE CEREBRUM, CRUS, PONS, MEDULLA, AND SPINAL CORD. (Modified from Flechsig.) *C. N.*, caudate nucleus; *L. N.*, lenticular nucleus; *O. T.*, optic thalamus; *G. P.*, gray matter of the pons; *F. R.*, formatio reticularis; *C. D.*, corpus dentatum; *O*, olivary body; *N. C.*, clavate nucleus; *T. N.*, triangular nucleus; *C. Q.*, corpora quadrigemina; *I. C.*, upper limit of the capsular fibres; *m*, *m*, *m*, motor centres around the fissure of Rolando; *c. r.*, fibres of the "corona radiata." 1, the "*pyramidal tract*,"

arising from the motor centres of the cerebrum and terminating in the cells of the anterior horns of the spinal gray substance (13 and 14); 2, 3, and 4, fibres connecting the cerebral cortex, the caudate nucleus and the lenticular nucleus with the *gray matter of the pons* after decussation (see Fig. 37), and then prolonged as 6 and 7 to the cerebellum; 5, fibres of the *superior cerebellar peduncle;* 6, 7, 8, 9, and 10, show by their colors the tracts which they are associated with, as well as their origin and termination; 11 and 17, the "*direct cerebellar tract*" of the spinal cord (whose probable termination is not correctly shown in the cut, as it probably ends in the vermiform process); 12, the *lemniscus* or "*fillet*" *tract*, connecting the olivary body with the optic thalamus and the corpora quadrigemina; 13, the cells of the cord connected with the *direct pyramidal tract;* 14, the cells of the cord connected with the *crossed pyramidal tract;* 15, fibres of the *column of Burdach*, terminating superiorly in the *triangular nucleus;* 16, fibres of the *column of Goll*, terminating superiorly in the *clavate nucleus;* 19, fibres of the cord which terminate in the so-called "*reticular formation*" directly; 18, fibres of the *ret. form.* going to the cerebellum. [The reader should compare this diagram with Figs. 36 and 37, and note the difference in the course of the sensory tracts during their passage through the medulla, there shown both in profile and in transverse section. The differences between the diagrams will make the various views held at the present time more apparent than a verbal description. In this diagram the crus is intentionally shown as distinct from and not related to the pons, in order to bring certain tracts of fibres into prominence. Some of the tracts shown in this diagram decussate.—See Fig. 37.]

This arrangement probably allows of an *automatic action of the cerebellum upon the skeletal muscles*, as exhibited in the maintenance of a fixed attitude (Spencer), the finer feats of rhythm (Spitzka), and difficult acts of equilibrium. Fig. 12 shows in a diagrammatic way the fascieuli that are defleeted in the pons to the cerebellum, as well as other important bundles of nerve fibres. It should be contrasted with Figs. 15, 36. and 37, since eaeh will aid in the comprehension of the other, and at the same time illustrate different views which are held in reference to the course of the sensory tracts in the medulla.

A magnified section, made through the pons shows, in addition to those points in its architecture already referred to, (1) the fibres of the sixth cranial nerve and its nucleus of origin; (2) a part of the trigeminal nucleus; (3) a part of the faeial nucleus; (4) the superior olivary body; (5) the posterior longitudinal bundle; (6) the round bundle (fasciculus teretes); and many other points whose functions cannot be described here in detail (Fig. 13).

The reticular formation (Fig. 15) is divided by the fibres of the hypoglossal nerve, into a *median area* (between the nerve root and the raphæ) and a *lateral area* (lying to the outer side of the nerve root).

The former area is chiefly composed of medullated fibres; while the latter contains numerous nerve cells. The fibres of the anterior root-zones of the spinal cord become lost chiefly in the median area, according to some observers; while those of the lateral column of the cord probably have an association of some kind with the cells of the lateral area.

The fibres of the reticular formation appear to end, in part, in the substantia nigra of the crus and the medullary laminæ of the thalamus; while some appear to join with a bundle of fibres from the red nucleus of the tegmentum, and to pass through the posterior part of the internal capsule, and to radiate toward the eortex.

The posterior longitudinal bundle (Fig. 11) is believed by Spitzka to arise in the deep gray of the corpora quadrigemina, and to unite the eells of these bodies with the nuclei of the fourth and sixth nerves, and the nuclei of the muscles of the neek. He is led to the conclusion that it

presides over the automatic relationship between the movements of the head and the visual apparatus.

Within the substance of the pons, those fibres of the *facial nerve* which are prolonged cephalad, decussate. The level of this decussation may be designated by an *imaginary line*, which shall connect the apparent origins of the fifth cranial nerves (Gubler).

Lesions of the pons above the line of Gubler, which affect the facial fibres, produce facial paralysis on the side opposed to the lesion, and, when below that level, upon the same side as the lesion.

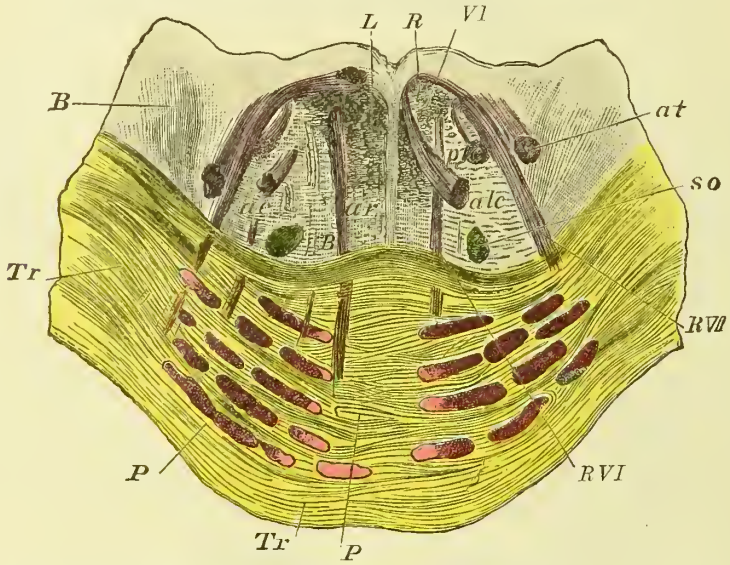


FIG. 13.—A TRANSVERSE SECTION THROUGH THE PONS, ON A LEVEL WITH THE ROOTS OF THE SIXTH AND SEVENTH CRANIAL NERVES FROM A NINE MONTHS' EMBRYO. (Modified from Erb and Ross.) The right half represents a section made a little lower than the left. *Tr.*, transverse fibres of the pons; *P*, pyramidal fibres (see Figs. 12, 36, and 37); *so*, superior olivary body; *L*, posterior longitudinal fasciculus; *t*, fasciculus teretes (round bundle); *Rvi*, root of abducens; *R VII*, root of facial; *at*, ascending root of trigeminus. (This figure shows well the interlacing of the vertical pyramidal fibres with the horizontal [transverse] fibres of the pons.) *R*, round bundle; *B*, peduncle of cerebellum; *a, r.*, upward prolongation of the anterior root-zone of the spinal cord; *a. l. c.*, anterior nucleus of the facial nerve; *p. l. c.*, posterior nucleus of the facial nerve.

The pyramidal fibres, the fibres of the sensory tracts, the fibres of the so-called "speech tract," some of the facial fibres, and also of the trigeminal and hypoglossal nerves, run cephalad through both the pons and the crus to reach cortical centres of the cerebrum. For this reason, lesions of the crus or pons may produce symptoms which indicate destruction of one or more of these tracts. Among these symptoms may be mentioned the following: Hemiplegia, hemianæsthesia, facial palsy, facial anæsthesia, disturbances of speech, paralysis of the third and sixth cranial nerves. These conditions will be discussed separately in subsequent pages of this section.

THE CEREBELLUM.

Although the cerebrum usually overlaps the cerebellum in animals of the higher types, the fact that it does not do so is not necessarily an indication of a lower grade in the scale of development. The construction of both the cerebrum and cerebellum becomes more intricate as development progresses. Benedict has advanced the view that in criminals brains the cerebrum did not overlap the cerebellum. Statements of this kind have been shown by Wilder to be open to suspicion, from the defective methods employed in the examination of the brain. The contour of the brain (when hardened *in situ* by Wilder's method) presents a marked contrast to the outlines commonly accepted as normal.

This ganglion is connected with many other component parts of the brain by fibres which compose three pairs of processes, called the *inferior*, *middle*, and *superior peduncles* of the cerebellum.

1. The fibres which compose the INFERIOR PEDUNCLE (*restiform body —processus e cerebello ad medullam*) joins the cerebellum to the medulla. Although authorities differ respecting its formation, it probably comprises three distinct sets of fibres. These are as follows: (1) those constituting the *direct cerebellar column* of the corresponding lateral half of the spinal cord; (2) a set derived from the *olivary body* of the opposite side of the medulla; (3) a set derived from the *nucleus of Burdach's column* of the same side of the cord.

These three sets carry impressions of different forms of sensation to the cells of the *cerebellar cortex* and the *corpus dentatum* (a collection of cells within the substance of the cerebellum).

The cerebellum receives, therefore, through its inferior peduncle, two centripetal tracts at least, one derived from the lateral columns, and the other from the posterior columns of the cord. Spitzka believes that the *muscular sense* is conveyed to the cerebellum by means of Clarke's columns, acting in conjunction with the direct cerebellar columns, and the *tactile sense* by means of Burdach's columns and the olivary nuclei of the medulla.

The fibres of the *direct cerebellar column* are supposed by Starr to connect the cerebellum (indirectly through the cells of Clarke's column) with the *thoracic and abdominal viscera*. The other fibres, according to this observer, transmit to it certain impressions of the *tactile muscular-sense* from the lower and upper extremities.

2. The fibres which compose the MIDDLE PEDUNCLE (*processus e cerebello ad pontem*) assist to form the pons. Its fibres are probably associated by a direct communication with the gray masses found within the pons. Some of these act as commissural tracts between the cerebellar hemispheres. Others are probably a part of the motor and auditory apparatuses.

The fibres of the SUPERIOR PEDUNCLE (*processus e cerebello ad cerebrum*) connect the cerebellum with the higher centres. They pass into the posterior part of the corpora quadrigemina (*optic lobes*), converge beneath these bodies, decussate into the red nuclei of Stilling (Fig. 14), and then pass to the cerebral hemisphere. Their termination within the cerebral hemisphere is, as yet, a matter of doubt. Luys believes that they pass to the caudate nucleus, and assist in charging its cells when exhausted. Meynert considers that they help to form a part of the motor apparatus.

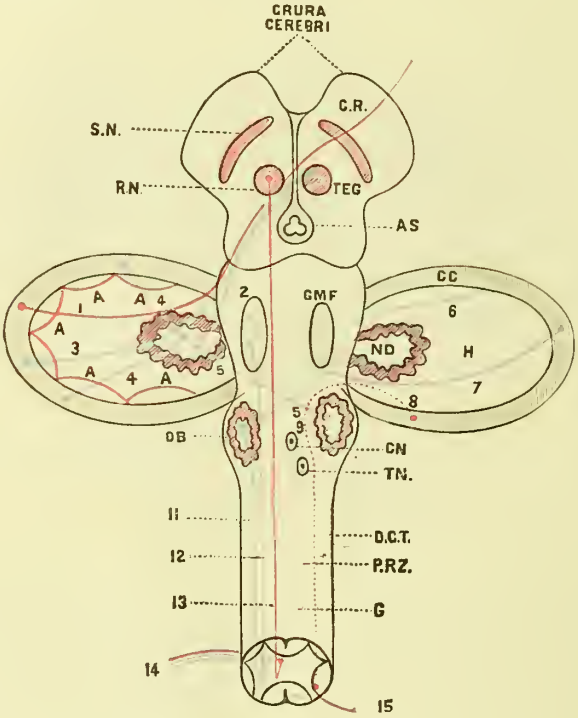


FIG. 14.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE VARIOUS SETS OF FIBRES COMPRISED WITHIN THE CEREBELLO-SPINAL SYSTEM. (Modified from Ross.) C. R., crusta cruris; T. E. G., tegmentum cruris; A. S., aqueduct of Sylvius, surrounded by the tubular gray matter; S. N., substantia nigra; R. N., red nucleus of the tegmentum; G. M. P., anterior gray matter of the pons; C. C., cerebellar cortex; N. D., nucleus dentatum; O. B., olivary body; C. N., clavate nucleus; T. N., triangular nucleus; D. C. T., fibres of the "direct cerebellar tract" of the spinal cord; P. R. Z., fibres of the "posterior root zone" of the same; G., fibres of the "column of Goll;" 1, cerebro-cerebellar fibres; 2, fibres from the red nucleus of the tegmentum to the dentate nucleus of the cerebellum; 3, fibres from the red nucleus to the cerebellar cortex; 4, fibres from the cerebellar cortex to the dentate nucleus; 5, fibres from the dentate nucleus to the olivary body of the opposite side; 6, fibres from the cerebellar cortex to the olivary body of the opposite side; 7, fibres from the cerebellar cortex to the anterior gray nucleus of the pons of the opposite side; 8, fibres of the direct cerebellar tract; 9, fibres connecting the clavate nucleus and the olivary body of the same side; 10, fibres connecting the triangular nucleus and the olivary body of the same side; 11, fibres passing from the olivary body to the horns of spinal gray matter; 12, fibres passing from the anterior gray matter of the pons to the horns of spinal gray matter; 13, fibres passing from the red nucleus of the tegmentum to the anterior horns of the spinal gray matter; 14, fibres escaping from the spinal cord through the anterior root of a spinal nerve; 15, fibres of the posterior root of a spinal nerve, entering at the posterior horn of the spinal gray matter. The dots in the cut end of the spinal cord, near to 15, indicate the relative position of the different tracts with which they are connected. A, A, A, represent fibres which are destined to connect different convolutions of the cerebellar cortex (fibræ propriæ).

That the *red nucleus* has no direct connection with the sensory tracts of the central nervous system, seems to be proven by the fact that lesions of that nucleus tend rather to disturb coördination of movement (probably by disturbing the relationship between the nucleus and the cerebellum) than the conduction of sensory impulses. Atrophy of one cerebellar hemisphere is always accompanied by a similar change in the opposed red nucleus.

Spitzka regards the cerebellum as an "informing depot," by which the cerebrum is made cognizant of "the relations which the body bears to time and space." This author believes that all rhythmic movements (such as dancing, etc.) and skillful feats of equilibrium are presided over by this ganglion. Mitchell is led to think that the cerebellum is simply a storage reservoir for nerve force.

It is stated by different observers that some filaments of the nerves of hearing (eighth pair), and of the trigeminal (fifth pair), the motor oculi (third pair), the abducens (fourth pair), and the pneumogastric (10th pair), can be traced (directly or indirectly) to the cerebellum.

The masses of gray matter comprised within the cerebellum (which have been specially named) comprise (1) the *cerebellar cortex;* (2) the "*nucleus of the ventricular roof*" (Spitzka) or the *nucleus fastigii;* (3) the *nucleus emboliformis;* (4) the *nucleus globosus;* and (5) the *corpus dentatum.*

I quote from a lecture of mine, published by the *Medical Record* some years ago, the following paragraphs, subject to some corrections:—

"From a standpoint of our present knowledge, the cerebellum must be considered as the 'terra incognita' of the brain. The clinical evidence is discordant. The anatomical connections of the cerebellum with other parts of the nervous system are remarkable, and their minute structure is, as yet, imperfectly understood. The region overlapped by the cerebellum is interspersed with important collections of gray matter, which act as nuclei of origin for important nerve tracts; so that all experiments made upon the cerebellum itself or its peduncles are liable to cause injury to some of the neighboring parts, and thus to yield results which are puzzling and unreliable. Conjecture inevitably forms an important element in all of the theories advanced respecting the functions of the ganglion itself, or of certain of its parts. Nothnagel claims to have demonstrated that mechanical stimulation of the surface of the cerebellum will give rise to muscular movement without signs of pain being perceived. He found that these movements developed slowly, appearing first on the side operated upon and then ceasing, only to appear upon the opposite side. He states that he has demonstrated that the fifth, facial, and hypoglossal nerves, as well as nerves distributed to the trunk and extremities, can be thus called into action. The same observer concludes that destruction

of the *commissural fibres* and the *vermis* produces incoördination of movement. Hitzig and Ferrier believe that injuries to the lateral lobe produce the same varieties of 'forced movements' as are noticed after section of the middle peduncle. Flourens observed that injuries to the anterior or posterior parts of the vermis caused animals to fall forward or backward respectively, and his views have been confirmed by others. Ferrier found that stimulation of the cerebellar cortex, by the interrupted electric current, produced in monkeys, cats, and dogs movements of the eyeballs, with associated movements of the head, limbs, and pupils. Adamuck produced the same effects, however, by stimulating the corpora quadrigemina. Hitzig refutes the view that Ferrier's results were due to an escape of the current by claiming to have produced similar effects by mechanical irritation of the cortex. Eckhard has brought forward facts which tend to show that in certain parts of the cerebellum lesions tend to produce diabetes or simple hydruria, thus resembling the effects of irritation of the medulla in the region of the floor of the fourth ventricle.

"In the face of this conflicting mass of experimental evidence, I mention now one of the most plausible and attractive theories respecting the relation of the cerebellum and cerebrum to muscular contraction, which has been advocated by Spencer and sustained by Hughlings-Jackson, Ross, and others. It is believed by these authors that all *continuous tonic muscular contraction* is governed by the cerebellum, and the *alternate* or *clonic muscular contractions* by the cerebrum, in so far as they are required to maintain a posture or produce a change in attitude. In all efforts to maintain an attitude (once assumed as the result of some cerebral impression received) the cerebellum holds the muscular apparatus in its proper state of tonicity; but when the attitude is to be changed, for any possible reason of which the cerebrum is conscious, the proper muscles are relaxed and others thrown into a state of contraction by means of the higher ganglion. The body is then intrusted to the influence of the cerebellum if the attitude is to be again maintained. Thus it is suggested that the cerebellum be considered as capable of automatic action, but still as a subordinate to the cerebrum, which possesses the power of overcoming it in one of two ways · First, by increasing the supply of nerve force to certain sets of cells, then under the influence of the cerebellum, and thus altering their action upon muscles; or second, by inhibiting or totally arresting the cerebellar influx to the antagonistic sets of muscles. Both are designed, according to this view, to act either automatically or in unison, but the cerebellum is the servant of the cerebrum to do its bidding when required.

"It will be at once perceived that this theory applies to the complex physiological acts of walking; the prolonged maintenance of any given posture; the transfer of the centre of gravity; the passive state of groups

of muscles; and many of the morbid phenomena observed in muscles, as the result of impairment of the higher nerve centres. It will be impossible to discuss all of these conditions in this connection. Hughlings-Jackson and Ross have covered the more important points in their works. If we form our views of the physiological functions of the cerebellum purely from the standpoint of the anatomical connections which that ganglion is known to possess, we cannot but agree with Bechterew in some of the conclusion which he has lately advanced. This author believes that the cerebellum is intimately connected with three organs which tend to exert an influence upon equilibrium, as follows: First, the *semicircular canals*, connected with the organ of hearing; second, the *organ of sight*, since the movements of the globe of the eye and possibly the sense of vision may be traced to a relation with the gray matter in the floor of the third ventricle and subsequently with the cerebellum; third, the *olivary gray matter*, which this author thinks is probably connected with the organs of tactile sensibility.

"The views of this author have been in part anticipated and sustained by Spitzka, who, in an article published some years ago, considers the cerebellum as the centre where 'impressions of touch and position are associated with those of time and space,' and hence the seat of coördination of the most delicate forms of movements; such as are necessary, for instance, 'to the proper adjustment of the drum-membrane of the ear for the correct appreciation of sounds, the appreciation of time and rhythm, and the finer acts of equilibrium.' In filling this position, the latter author believes that the cerebellum is subordinate to the cerebrum, to which it acts as an 'informing depot' for coördination, rather than as a distinct centre."

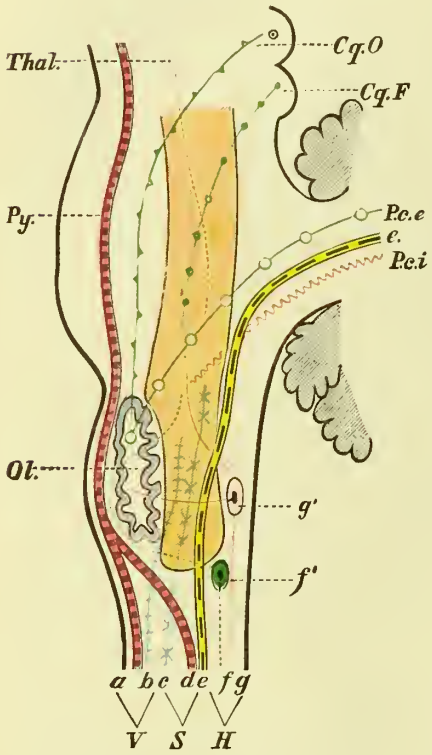
THE MEDULLA OBLONGATA.

Space will not allow of a description of the architectural details of this very intricate structure. Its component parts may, however, be thus classified: 1. Certain fibres which are known to pass without interruption through its substance; thus joining the cerebrum or cerebellum with the spinal cord.* 2. Other fibres which arise within the medulla from the cells of its different nuclei. 3. Collections of gray matter, which are analogous to well defined parts of the spinal gray matter. 4. Collections of nerve cells which are destined for special cranial nerve roots; these have no analogue in the spinal gray substance.

*The "direct cerebellar fibres" first appear in the second or third lumbar segment, and are continued upward to the inferior peduncle of the cerebellum. The cells of Clarke's column are multipolar and much smaller than those of the anterior horns. According to Ross and Gaskell, the homologues of these cells are observed in the nucleus of the vagus nerve (Fig. 15).

Under heading (1) may be enumerated: the motor or "pyramidal tracts" (direct and crossed); the so-called "sensory tracts" to the cerebrum; and the "direct cerebellar tracts" formed indirectly through the agency of the cells of Clarke's column within the spinal cord.

FIG. 15. FIG. 16.



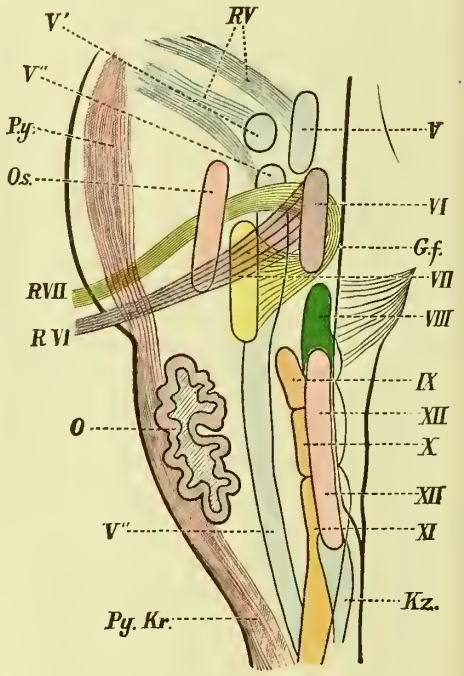


FIG. 15.—DIAGRAM OF THE CHIEF TRACTS IN THE MEDULLA. (After Erb.) (The formatio reticularis is represented by shading.) *Ol.*, olivary body; *V*, anterior; *S*, lateral, and *H*, posterior spinal funiculi; *a*, pyramido-anterior tract; *d*, pyramido-lateral tract; *Py*, pyramidal tract; *b*, remainder of anterior column; *c*, remainder of the lateral column; *e*, *e*, cerebello-lateral tract; *f*, funiculis gracilis; and *f'*, nucleus of the same; *g*, funiculus cuneatus, and *g'*, nucleus of the same; *P.c.i.*, internal fasciculus of the pedunc. cerebelli; *P.c.e.*, external fasciculus of the same; *Cq.F.*, tract from corp. quadr. to format. retic.; *Cq.O* the same to the olivary body; *Thal.*, tract from the thalamus opticus.

FIG. 16.—TRANSPARENT LATERAL VIEW OF THE MEDULLA, SHOWING THE RELATIVE POSITIONS OF THE MOST IMPORTANT NUCLEI; RIGHT HALF OF THE MEDULLA, SEEN FROM THE SURFACE OF SECTION; THE PARTS THAT LIE CLOSER TO THIS SURFACE ARE DEEPER SHADED. (After Erb.) *Py*, pyramidal tract; *Py. Kr*, decussation of pyramids; *O*, olivary body; *O.s*, superior olivary body; *V*, motor, *V'*, middle sensory, *V''*, inferior sensory nucleus of trigeminus; *VI*, nucleus of abducens; *G.f*, genu facialis; *VII*, nucleus facialis; *VIII*, posterior median acoustic nucleus;* *IX*, glosso-pharyngeal nucleus; *X*, nucleus of vagus; *XI*, accessorius nucleus; *XII*, hypoglossal nucleus; *Kz*, nucleus of the funiculus gracilis; *R V*, trigeminus roots; those of the *R VI*, abducens, and *R VII*, facialis.

*Respecting the auditory nerve roots, Spitzka believes that the posterior auditory root is the direct path for the transmission of sound impulses, and that the anterior auditory root is a path for the transmission of impressions which assist in the determination of equilibrium. Edinger rather inclines also to a somewhat similar view.

Under (2) the following bundles might be classed: The fibres which form the "fillet" or "lemniscus tracts;" those which join the nuclei of Goll's and Burdach's columns with the olivary bodies; those which pass into the inferior cerebellar peduncles from the olivary bodies; some fibres of the formatio reticularis; the hypoglossal, facial, and trigeminal cerebral tracts; the so-called "speech tract," etc.

Regarding the ultimate distribution of the fibres of the *lemniscus tract*, a wide diversity of opinion between authors of note exists. Flechsig believes that its sensory fibres pass lateral of the red nucleus, then into the posterior third of the internal capsule, then to the centrum ovale, and that they end in the cortex. Others, among whom may be mentioned Wernicke, Forel, and Roller, believe that they end in the medullary laminæ of the thalamus. Others again trace fibres from this tract to the corpora quadrigemina. (See also paragraphs on page 32.)

Under (3) and (4) come the nuclei of the cranial nerves; the olivary bodies; the cells of the "formatio reticularis;" the nuclei developed at the upper end of Goll's and Burdach's columns; and the accessory olivary bodies. The two diagrams of Erb which are introduced here will aid the reader in gaining a conception of the situation and extent of the more important nuclei, and the course of some of the tracts of fibres mentioned. It may be well, for one not familiar with the subject, to compare them with Fig. 13, and also with a section of the spinal cord which shows the situation of the columns spoken of (Fig. 32).

Note (particularly in Fig. 16) the situation of each of the nuclei, the peculiar course of the facial fibres, the situation of the olivary body and the superior olive, the enormous length of the inferior sensory nucleus of the fifth cranial nerve,* and the situation of the decussation of the "pyramidal" or motor tracts.

The looping of the fibres of origin of the facial nerve around the nucleus of the sixth cranial nerve in the medulla, has caused some observers to favor the view that these fibres are associated in some imperfectly understood way with the cells of that nucleus.

In Fig. 15 the reader should trace separately the course of the pyramidal tract (from above downward); that of the direct cerebellar tract (from below upward); the course of the fibres derived from the nuclei of the columns of Goll and Burdach; the various tracts that terminate in the reticular formation, as well as those that pass through it; and, finally, the fibres of the cerebellar peduncle.

* The trigeminus nerve is known to possess motor, sensory, and vaso-motor or secretory fibres. Spitzka concludes that the vaso-motor fibres spring from sub-ependymal nuclei; that the motor root arises in part from a continuation of the lower facial nucleus; and that the sensory root can be shown to arise from cells within the medulla, the cervical segments of the spinal cord, and the cerebellum.

Spitzka compares the medulla to "a hypertrophied segment of the cord, in which the longitudinal associating fibres outweigh in number and length those of any other spinal segment." He considers it as a ganglion which presides over all *reflex acts in which rhythm is an essential factor.*

Regarding the course, termination, and function of some of the sensory nerve tracts depicted in this diagram, it may be well to state that differences of opinion exist among authors of note. Whether some are first deflected to the cerebellum (being compelled to pass through the substance of that ganglion in order to reach their termination in the cortical centres of the cerebrum) is still an open question (see Figs. 12, 14, 15, 36, and 37).

Respecting the effects of the formation of the fourth ventricle upon the central gray masses, Spitzka remarks, that if we start with the comparison of the gray substance of the cord to a capital letter H, and imagine the vertical branches of the H to be separated posteriorly till they become almost horizontal, the anterior horn becomes the most internal, the lateral cornua intermediate between the anterior and the posterior, and the posterior horns the most external. Thus the "motor system" is to be sought for nearest the median line; the "mixed system" to its outer side; and the "sensory system" becomes the outermost.

The same author remarks, in this connection, that "as the median line of the cord becomes changed by the horizontal expansion of the fourth ventricle in the medulla, we may surmise that the more axial muscles will be represented by nuclei situated near the ventricular floor, the more appendicular muscles further away from it."

Concerning the trigeminal nerve roots and their nuclei, he also makes the following suggestions: "On the strength of the law of segmental harmony, we can theoretically infer that the part of the trigeminus origin situated in the level of the cervical spinal cord corresponds to the temporal cutaneous branches, which, with the upper cervical nerves that originate at the same level, share the distribution to the occipito-temporal region. That part which is in the level of the hypoglossal nucleus, will presumably correspond to the distribution of the lingual branch of the fifth. Further forwards (cephalad) in the level of the facial and motor trigeminal nuclei, we will have the dental, mental, and infra-orbital distribution projected, and in such a way that the nerves of the upper jaw will be above that of the lower. Still further cephalad, in the altitude of the oculo-motor muscles, will be the centre of the ophthalmic distribution area."

The statement is now generally accepted by neurologists as proven, that both the motor and sensory tracts, which unite the cerebral cortex with the cells of the spinal gray matter, *are functionally associated with both sides of the body.*

The main distribution is to that lateral half of the body which is opposed to the cerebral hemisphere to which the fibres can be ultimately traced. The decussation of both the motor and sensory nerve tracts is, therefore, not complete. The motor fibres decussate at the lower part of the medulla. Some of the sensory fibres (probably those which preside over the so-called "muscular sense") decussate also in the medulla (called by Spitzka the "piniform decussation," because it occupies a cone-shaped area in cross-sections of the medulla made at that level). Those tracts, which convey sensations of pain, touch, and temperature, probably decussate within the substance of the spinal cord.

FIG. 17.

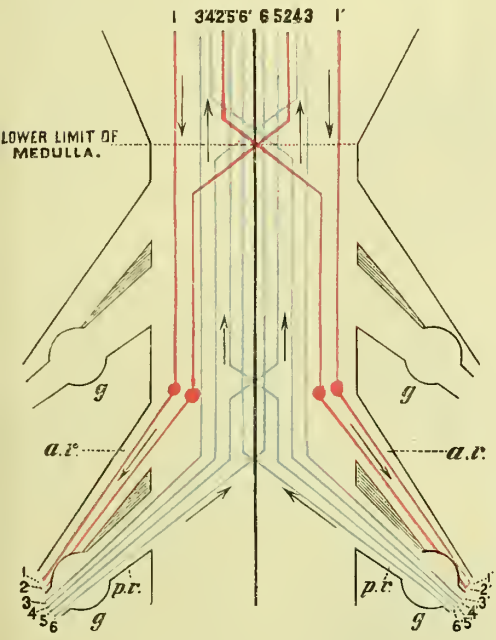


FIG. 18.

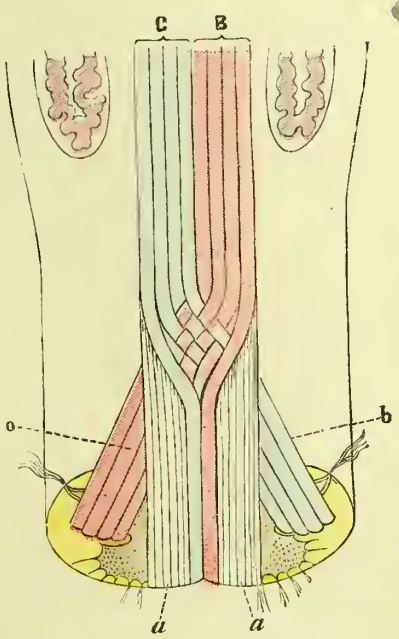


FIG. 17.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE COURSE OF THE FIBRES WHICH COMPOSE THE SPINAL CORD. 1,1′, direct pyramidal bundles; 2, 2′, crossed pyramidal bundles, decussating in medulla; 3, 3′, direct cerebellar fibres; 4, 4′, fibres related to "muscular sense," decussating in medulla; 5, 5′, and 6, 6′, fibres related to the appreciation of touch, pain, and temperature. The motor bundles (red) have a dot upon them to represent the motor cells of the cord (ant. horn). Note that the red fibres escape from the anterior nerve root (*a. r.*) and that the sensory bundles enter at the posterior nerve root (*p. r.*), which have a ganglion (*g*) upon them.

FIG. 18.—A DIAGRAM OF THE LOWER PART OF THE MEDULLA (as if transparent) TO SHOW THE DECUSSATION OF THE MOTOR BUNDLES. (After Erb). *a*, the non-decussation bundle (direct pyramidal fibres); *b*, the decussating bundle (crossed pyramidal fibres). The former occupy the column of Türck (Figs. 19 and 29) and the latter a portion of the lateral columns of the spinal cord (Figs. 19 and 29).

The "*reticular formation*" acts probably as a conductor of sensory impulses of pain and temperature which pass cephalad to reach the cells of the cerebral cortex where they become transformed into conscious realities (Starr).

The *lemniscus tract* or the "*fillet*," probably conveys impressions of muscular sense to the cerebral cortex, after the fibres related to that sense have decussated in the medulla (Starr). (See also page 32.)

The *pyramidal fibres* in the medulla tend to displace in a backward and outward direction, the fibres which probably assist to form the anterior root zone (Fig. 18).

Spitzka advances the view that the *nuclei* of origin of the *spinal accessory nerve* have different functions; that within the spinal cord is probably designed to innervate the trapezius and the sterno-mastoid muscles; the inner accessory nucleus is associated with the fibres distributed to the larynx, and may, therefore, according to this observer, be called the "nucleus laryngeus;" finally, the outer accessory nucleus is termed by him the "deglutitory nucleus," because it apparently shares in the motor supply of the muscles of deglutition.

The *posterior longitudinal bundle* becomes closely intermingled with the interolivary tract in the medulla; hence it is impossible to distinguish these fibres below the level of the pons.

THE DIAGNOSTIC VALUE OF SOME OF THE SPECIAL SYMPTOMS OF NERVOUS DERANGEMENT.

Some three years since, I published in the *Medical Record*, as an abstract of lectures delivered by me, a series of articles which discussed the various tests that have to be made at times by a neurologist to detect the existence of organic disease in the brain, the spinal cord, and the cerebro-spinal nerves. These articles will be reproduced in this and the following section, with such modifications and additions as subsequent reflection and experience on my part have suggested to me. A portion of the matter included under this heading constitutes a portion of this course of lectures. They were delivered in the New York Post-Graduate Medical School and Hospital, and also before the classes of the Medical Department of the University of Vermont. I quote from them as follows:—

"Before the various tests which are employed by the specialist in neurology to determine the existence of diseased states of the nerves and muscles are separately discussed (as they will be in Section II. of this work), it may be necessary to hastily summarize a few of the more important facts in nervous symptomatology.

"Not only are some of the tests, described later, complex in themselves, and therefore difficult of comprehension, but they would be absolutely useless in practice if the clinical bearing of each were not clearly comprehended. For example, a physician who has acquired a smattering of nervous symptomatology may be called upon to examine a patient who gives evidences of impairment of motor power in some part of his

body. This paralysis may be due to some trouble either in the brain of his patient. his spinal cord, or in some special nerve. If in the brain, the physician is called upon to decide (for himself at least) whether it is sit-

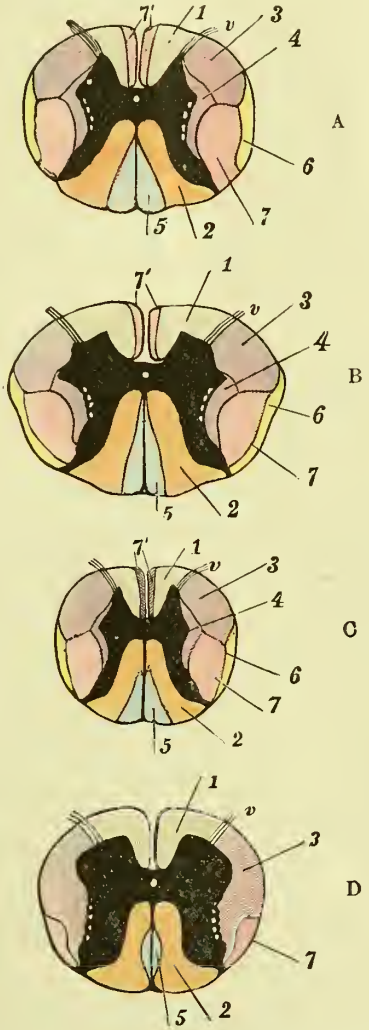


FIG. 19.—A DIAGRAM ILLUSTRATING THE DEVELOPMENT OF THE DIFFERENT SYSTEMS OF FIBRES IN THE SPINAL CORD. (After Flechsig.) A, section at level of 3d cervical nerves; B, at level of 5th cervical; C, at level of 6th dorsal; D, at the level of 4th lumbar nerves. 1, principal mass of anterior columns; 2, Burdach's columns; 3, lateral columns; 4, lateral boundary of gray substance; 5, columns of Goll; 6, direct cerebellar columns; 7, crossed pyramidal columns; 7', Türck's columns; *v*, anterior roots. Note that Türck's columns disappear in D; that Goll's columns increase in size from below upward; that the direct cerebellar columns appear in C, and increase in size in B and A; that the crossed pyramidal columns reach the surface in D; and that the shape of the gray substance differs in all the sections. The numerals employed in the cuts indicate the *order of development* of the various parts designated. It will be seen that the motor-tracts of the cord are the last to attain their complete development.

uated in (1) the coverings of the brain, (2) the external gray matter that invests it like a cap (the cerebral cortex), or (3) in parts more or less distant from its exterior. It is important, from a standpoint of prognosis and treatment, that he comes to some definite conclusion also regarding the character of the trouble. If the disease be confined to the spinal cord of the patient, it becomes necessary for the physician to discriminate again between affections that follow separate bundles of nerve fibres (systematic lesions of the cord) and those that spread transversely from column to column (focal lesions of the cord); and to decide also as to the height of the lesion, its pathological character, and the special regions that are affected by it. Finally, if the paralysis be due to some spinal nerve, the possibility either of brain or spinal disease must be excluded, and the cause must be sought for along the course of the nerve whose function is impaired.

Before I discuss the clinical tests of nervous diseases in detail, I direct the attention of the reader to some extracts from the chapters on the Diseases of the Brain and Spinal Cord that are embodied in the third edition of my work on 'Surgical Diagnosis.'

MOTOR PARALYSIS.

Anything which tends to impair the generating power of the nerve centres or the conducting power of nerve fibres may produce paralysis of motion or sensation.

"Motor paralysis (when due to a lesion affecting the cerebro-spinal axis) can result, therefore, from any condition which interferes with the motor convolutions of the brain, or the nerve fibres which start from them and are continued as the so-called 'motor tract.' The latter aid in all *voluntary movements of the extremities.* They pass through the following parts successively: (1) The white substance of the cerebral hemispheres; (2) the corpora striata; (3) the crura cerebri; (4) the pons Varolii; (5) the medulla oblongata; and (6) down the motor columns of the spinal cord.

"The disturbing lesions may be therefore classified as: (1) Those of the gray matter of the convolutions of the brain (cortical lesions); (2) those of the central mass of the cerebral hemispheres, including lesions of the "internal capsule;" (3) those of the corpora striata; (4) those of the crura cerebri; (5) those of the pons Varolii; (6) those of the medulla oblongata; (7) those of the spinal cord.

"The various tests which are employed to determine the existence and extent of a loss of muscular power will be given later."

CORTICAL PARALYSIS OR SPASM.

These may be dependent upon some lesion of the gray matter of the cerebral convolutions (the cerebral cortex). They may occur in connection with abscesses, blood-clots, spots of softening, tumors, depressed bone, meningeal thickenings and exudations, embolism, thrombosis, etc

The researches of Ferrier, Luciani, Exner, Horsley, Beevor, and others have lately taught us the situation of special motor centres scattered over the convolutions of the so-called "motor area" of the cerebrum. From this standpoint we are often enabled to judge of the seat of the lesion by the aid of the groups of muscles which exhibit the paralytic state (monoplegia). Hughlings-Jackson and Brown-Séquard have added to our knowledge of the relative effects of destructive and irritative lesions of the cerebral cortex.

"*Irritative lesions* of the cerebral cortex are usually ushered in by convulsive attacks, which leave the subject paralyzed in some special group of muscles (monoplegia); or, if hemiplegia ensues, some parts of the body are more affected than others. The paralysis is usually transient, and returns again after subsequent convulsive attacks. These irritative lesions are particularly liable to be of syphilitic origin.

"*Destructive lesions* of the cerebral cortex are characterized by paralysis of special groups of muscles (monoplegia, or mono-anæsthesia), as was the case with the irritative lesions. This is in marked contrast to the hemiplegia, or hemianæsthesia which follows lesions of the central portions of the brain. If the lesion be very extensive coma may be produced, but consciousness is not usually lost unless the attack be accompanied by convulsions. Pain of a local character within the head is often complained of, and percussion over the seat of the lesion frequently elicits it, if it should be absent. The sensibility of the paralyzed parts is not impaired unless more or less sensory paralysis exists as a complication. The paralyzed muscles exhibit the normal electro-contractility. As is the case with all cerebral lesions, the paralysis is developed on the side opposite to the exciting cause (except in very rare instances). In cortical lesions of the motor area, the muscles frequently exhibit a state of post-paralytic rigidity in the early stages of the disease. The various types of monoplegia and the surgical guides for trephining over special motor centres have been discussed in the Author's work upon the anatomy of the nervous system.

Horsley has lately added a valuable contribution to the subject of cortical localization, based upon experimentation on monkeys, and also on observations in ten cases where the diseased area was successfully determined in the human subject prior to operative procedure. His conclusions are therefore worthy of note. They may be summarized as follows:—

1. *Sulci, or fissures, are not to be regarded as accurate boundaries to cortical areas*, although they constitute valuable landmarks for operative procedures upon the cortex.

2. The *motor centres*, according to this observer, are capable of *further subdivision* than those described by Ferrier, and they overlap each other at their borders.

3. The *face area*, taken as a whole, embraces the lower third of both central convolutions (Fig. 4). This is subdivided into (*a*) an upper and anterior portion, which controls the upper part of the face and the angle of the mouth; (*b*) the anterior half of the lower portion, which governs the movements of the vocal cords; and (*c*) the posterior half of the lower portion, which governs the lower part of the face and the floor of the mouth.

4. The *area for the upper limb* occupies the middle third of both central convolutions, and also the base of the superior and middle frontal convolutions. It joins, and also merges with, the area for movements of the head and neck in the middle frontal gyrus, and with that of the leg in the superior frontal gyrus.

In the area described as pertaining to the upper limb, the uppermost part is thought to control the muscles of the shoulder; below, and posteriorly, the elbow is represented; still further below and somewhat anteriorly, the wrist; next in order, anteriorly, the finger-movements are placed, and lowest of all, and posteriorly, the thumb-movements are located. These views he substantiates by observations made in cases of cortical tumors, where spasm was developed and appeared first in an isolated region of the upper limb.

5. The *area for the lower limb* is described by this observer as embracing the upper portions of the two central convolutions; also the whole of the superior parietal, the base of the superior frontal convolutions, and the para-central lobule. This description is not materially different from that of Ferrier (Fig. 3).

The subdivisions of this area are as yet incompleted, but the points given are of interest to the surgeon. The movements of the big toe are referred to the para-central lobule; those of the leg alone to the middle part; those of the leg and arm combined to the most anterior portion. Most of these conclusions agree in the main with those of Ferrier (Fig. 3).

6. The *area for movements of the head and neck*, and also for conjugate deviation of the eyes, is placed by this observer (in common with Ferrier and Munk) in the bases of the three frontal gyri (see 12, in Fig. 3).

7. Respecting the steps required to locate the fissures of Rolando and Sylvius upon the human subject during life (as a basis for surgical procedures) the following conclusions are reached :—

(*a*). The method first described by Thane for *locating Rolando's fissure* is adopted. A careful measurement is first made along the mesial line of the skull, starting from the root of the nose and extending to the occipital protuberance. This distance is then halved. The fissure of Rolando at its upper part lies one-half inch posteriorly to its central point. A strip of flexible iron (with a movable arm placed at an angle of sixty-seven degrees to it) is now laid upon the middle line of the head; the point of junction of the movable arm with the mesial strip being carefully located at the point previously determined as overlying the upper end of Rolando's fissure. When this is accurately done, the movable arm marks the course of the upper two-thirds of the fissure of Rolando, but, as the lower third tends to bend slighty backward, it does not as clearly define the lower third of that fissure.

(*b*). To accurately locate the *fissure of Sylvius* upon the skull no little precision is required. A few points in the bones of the skull have first to be accurately determined. These are as follows: (1) The point where the temporal ridge crosses the coronal suture (the "*stephanion*"). This can usually be felt with the finger, the coronal suture appearing to the touch either as a depression or as a ridge lying between two grooves. (2) Exactly midway between the stephanion and the upper border of the zygoma, on a line drawn vertical to the zygoma toward the stephanion, lies another point known as the "*pterion.*" (3) To determine the highest point of the suture which exists between the squamous portion of the temporal bone and the inferior border of the parietal bone (the "*squamo-parietal*" *suture*) a measurement has to be made, because that suture cannot be felt beneath the temporal muscle.

In front of the temporo-maxillary articulation, an upright upon the line C–D in Fig. 20, would cross the zygoma. The junction of the upper and middle thirds of the measurement made upon such a vertical line between the upper border of the zygoma and the ridge formed by the temporal muscle, indicates the situation of the highest point of the squamo-parietal suture.

The anterior limb of the Sylvian fissure starts from a point which lies from one-half to one line (one-twenty-fourth to one-twelfth of an inch) in front of the "pterion." It runs anteriorly and upward from that point. The posterior limb passes backward and slightly upward from the same point.

8. The *sulci of the frontal lobe*, and also the *inter-parietal sulcus* (which limits the so-called "motor area" of the cortex posteriorly), are next to be located upon the exterior of the skull, in order to map out the convolutions. The guides to the sulci are as follows:—

The *precentral sulcus* lies somewhat behind the coronal suture and parallel to it. It extends to about the middle of Rolando's fissure.

The *inferior frontal sulcus* diverges from the preeentral at about the level of the temporal ridge.

The *superior frontal sulcus* starts at a point in the preeentral gyrus somewhat posterior to the line of the preeentral sulcus if continued upward. The exaet point is about midway between the fissure of Rolando, and an upward eontinuation of a line in the direetion of the precentral suleus. Its altitude in the cerebrum is slightly above the level of a point (midway between the mesial line of the skull, and the eentre of the parietal eminenee) which designates the lower limit of the superior parietal convolution.

The *inter-parietal sulcus* in its ascending course starts from a point on a level with the junetion of the middle and lower thirds of Rolando's fissure. It turns backward on a level situated midway between the mesial line of the skull (marked by the longitudinal fissure) and the centre of the parietal eminenee.

HEMIPLEGIA.

This condition is charaeterized by a paralysis of motion in *one lateral half* of the body. It is often associated with more or less anæsthesia, but it may exist independently of it. I quote from a previous article of my own, as follows:—

"Hemiplegia may be produeed by any lesion which interferes with the free aetion of the 'motor tract' of fibres during their passage from the motor convolutions of the eerebrum to the eolumns of the spinal cord; and lesions of the spinal cord itself (if sufficiently high up and restrieted to a lateral half of the cord on the side which corresponds to the paralysis) may also induce it.

"If the lesion be within the cavity of the cranium the hemiplegia will be on the opposite side of the body; if it be spinal the hemiplegia will be upon the same side.*

"Hemiplegia from intraeranial lesions may be the result of embolism, thrombosis, apoplexy, softening, abscess, tumors, compression of the brain from traumatie eauses, destruction of limited portions by injury, general pressure from inflammatory exudations, etc.

"Conseiousness is generally lost when eerebral hemiplegia is developed. Convulsive attacks are not usually present at the onset of the paralysis. The paralysis is more profound, as a rule, than that of cortieal lesions, and of longer duration. The special senses are not infrequently involved to a greater or less degree. Other eranial nerves, which are not assoeiated with the special senses, may also give evidenee of being implieated by the lesion. The faeial nerve is most frequently affected.

*This rule is not absolutely true, but the exceptions to it are so rare that it is a safe one to follow in clinical deductions.

"By means of anatomical guides the seat and extent of an intracranial lesion may often be determined with positiveness. The co-existence of impairment of sensation with motor paralysis is a valuable diagnostic sign that the exciting lesion is within the substance of the brain and not upon its surface. The exceptions to this rule are extremely rare.

"The localization of non-cortical lesions is more difficult and somewhat less certain than those which are confined to the cortex. A careful study of all the symptoms presented (when combined with a knowledge of modern cerebral and spinal anatomy) will often, however, lead to the most positive deductions. It should be remembered that accuracy of diagnosis often leads to success in treatment of disease, and in no case is it better exemplified than in the nerve centres."

CROSSED PARALYSIS.

A condition in which the face or some organ of special sense gives evidence of an *impairment of a cranial nerve, while the body is simultaneously rendered hemiplegic on the opposite side*, is termed "crossed paralysis"—the "paralysie alterne" of the French authors. We owe much of our knowledge of this subject to Professor Romberg, of Berlin, who has written extensively upon it.

"The more common forms of crossed paralysis are named from the cranial nerve which exhibits an impairment of its functions. They are as follows: First cranial nerve (olfactory) and body type; third cranial nerve (motor oculi) and body type; fifth cranial nerve (trigeminus) and body type; seventh cranial nerve (facial) and body type. They will be discussed later.

"It may be well to remark in this connection that 'crossed paralysis' is of special clinical importance, because it often imparts the most positive information to the surgeon in regard to the seat of the intracranial lesion which has produced it."

COMPLETE PARALYSIS.

When a lesion is situated at the base of the brain, and is sufficiently large to involve the motor fibres of both hemispheres, the body may be completely paralyzed below the head.

"Various cranial nerves—chiefly the third, fifth, sixth, and seventh—are liable to then exhibit the effects of simultaneous pressure upon them; hence the general paralysis of the body is apt to be associated with paralytic symptoms confined to the face.

"*Bilateral spinal lesions* when situated high up in the cervical region, may also cause a form of complete paralysis of the body—the so-called 'cervical paraplegia.'"

SENSORY PARALYSIS.

The sensation of special parts of the body may be so modified by lesions of the nerve centres as to constitute a type of paralysis. The various forms of this condition may exist independently of motor paralysis, or may co-exist with it. The tests commonly employed to detect the limits and degree of sensory paralysis will be given later.

"Sensory paralysis may be classified as follows: (1) Paralysis of those cranial nerves which are not endowed with motor attributes; (2) paralysis of sensory nerves below the head. The latter subdivision comprises hemianæsthesia, general anæsthesia, and local anæsthesia.

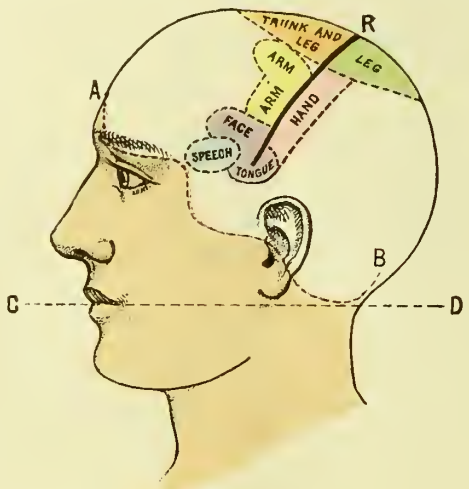


FIG. 20.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE REGIONS OF THE CORTEX OF THE BRAIN ASSOCIATED WITH SPECIAL PARTS OF THE BODY, AS A GUIDE TO THE SEAT OF DESTRUCTIVE PROCESSES IN CONNECTION WITH MOTOR PARALYSIS OR SPASM.—*A*, *B*, dotted line to indicate the relative depth of the brain in the anterior, middle, and posterior fossæ of the skull; *C*, *D*, a line running from the cusps of the teeth of the upper jaw to the tip of the mastoid process of the temporal bone. This is useful as a base line from which to erect vertical lines, by careful measurement during life, which shall intersect the different centres of the brain. Trephining for the relief of *monoplegia* and *aphasia* can thus be performed with scientific accuracy. The circle designated in the diagram as the speech area is related only to the *motor acts* required in speech. It has no relationship to the various forms of sensory aphasia.

The views lately advanced by Horsley (p. 49), seem to suggest that this diagram might be modified somewhat.

"Among the various clinical evidences of lesions which affect the sensory nerve tracts of the brain and spinal cord, the following may be mentioned: (1) *hyperæsthesia*, or an excitation of sensibility; (2) *numbness;* (3) *formication*, or a sensation like the creeping of ants; (4) *abolition of sensation*, or complete anæsthesia—this condition may be general or local; (5) *anosmia* and *hemianopsia;* (6) *delayed sensation*, as is evidenced by a perceptible interval of time between the contact of a foreign body with the skin and its conscious appreciation by the patient

when the eyes are closed. The pricking of the skin with a needle is a test commonly employed to determine the latter condition.

"Some of these conditions will be now considered in their more important aspects. Others will not be separately described, as they would require too much space, provided such a *résumé* was attempted."

HEMIANÆSTHESIA.

This condition is characterized by a loss only of sensation (not of motion) in *one lateral half* of the body. It is often associated with more or less marked hemiplegia. When hemiplegia and hemianæsthesia exist upon the *same side* a cerebral lesion may be strongly suspected; when upon *opposed sides*, a spinal lesion probably exists.

The tests employed to determine the existence of this state and its degrees of intensity are the same as those employed in any form of sensory paralysis. They will be described later.

"Hemianæsthesia (when not due to hysteria or spinal lesions) indicates that the exciting lesion has impaired the conducting power of the fibres associated with the so-called 'sensory area' of the cerebral convolutions (Fig. 5). There is strong clinical evidence to sustain the opinion that these fibres run in the posterior third of the 'internal capsule.' Lesions of this latter region are not infrequently the cause also of more or less impairment of sight, smell, hearing, and taste, in addition to their effects upon general sensation. Charcot, Ferrier, Rendu, Raymond, and others who have studied the effects of lesions of the posterior third of the internal capsule of the cerebrum concur in this statement.

"Hemianæsthesia is frequently accompanied by the development of choreiform movements after the paralysis has developed. These may assume the type of athetosis, true ataxia, or tremor. The same may also be said of that type of hemiplegia which occurs as the result of lesions of the internal capsule of the cerebrum. Finally, in cerebral hemianæsthesia there is usually more or less insensibility to touch, pain, and temperature, and also abolition of muscular sensibility with complete retention of electro-motor contractility. The mucous membranes of the eye, nose, and mouth, are also frequently rendered anæsthetic. Aphasic symptoms have been observed to co-exist with hemianæsthesia (see page 68)."

NUMBNESS AND FORMICATION.

In connection with sensory paralysis, a condition of numbness, which the patient describes as feeling as if some special part was "fast asleep" is often experienced. In others, a sensation which has been compared to the "creeping of ants" over some special region is complained of. The latter has been termed "formication."

"These abnormal sensations are confined exclusively to those parts in which the sensory nerves are more or less impaired. This impairment may result from some lesion of the nerves after their escape from the brain or spinal cord, or from lesions of the nerve centres which involve their fibres of origin.

"By a careful study of the symptoms, a skilled anatomist is often enabled to decide whether the lesion is cerebral, spinal, or confined to special nerve trunks. This field is too extensive, however, to be considered in detail here."

HYPERÆSTHESIA.

In connection with lesions of the brain and spinal cord, a condition of excessive sensibility is sometimes encountered. It is termed "hyperæsthesia."

"It may exist independently of motor or sensory paralysis; or, again, it may co-exist with them. Its clinical significance depends upon its seat and extent and the other evidences of disturbed nervous functions which co-exist. It will be discussed from a clinical point of view in subsequent pages."

HEMIANOPSIA.

A loss of vision in one lateral half of each retina is termed "hemianopsia" and "hemianopia." It is called "hemiopia" by some authors; although incorrectly so, as that term means "half-vision," while the two others mean what they are intended to express.

The following steps are commonly employed to detect the existence of this symptom: Request the patient to close one eye by pressing the lid down with the finger, and to so direct the open eye as to concentrate its gaze upon some fixed object near to it. [I usually hold up the forefinger of my own hand within a foot of the patient's open eye, and tell him to look steadily at it.] Having done this, take some object which is easily seen (such as a piece of white paper) in the unemployed hand, and move it to the right and left of the object upon which the patient is gazing, and also above and below the object, asking the patient, in each case, if the two objects are seen simultaneously and with distinctness, and notice upon which side of the fixed object the patient cannot perceive the moving object. It is self-evident that the retina is blind upon the side opposite to that upon which the moving object is lost to sight.

The most common form of hemianopsia is that in which the nasal half of one eye and the temporal half of the other is blind. This condition is termed *homonymous hemianopsia*. It is the result of pressure upon, or actual destruction of one of the optic tracts, the pulvinar of the thalamus, the cortex of the occipital lobe (probably the *cuneus*), or the fibres that connect it with the optic tract. (This seems to be proven by the late researches of Munk, Wernicke, Starr, Seguin, and others.)

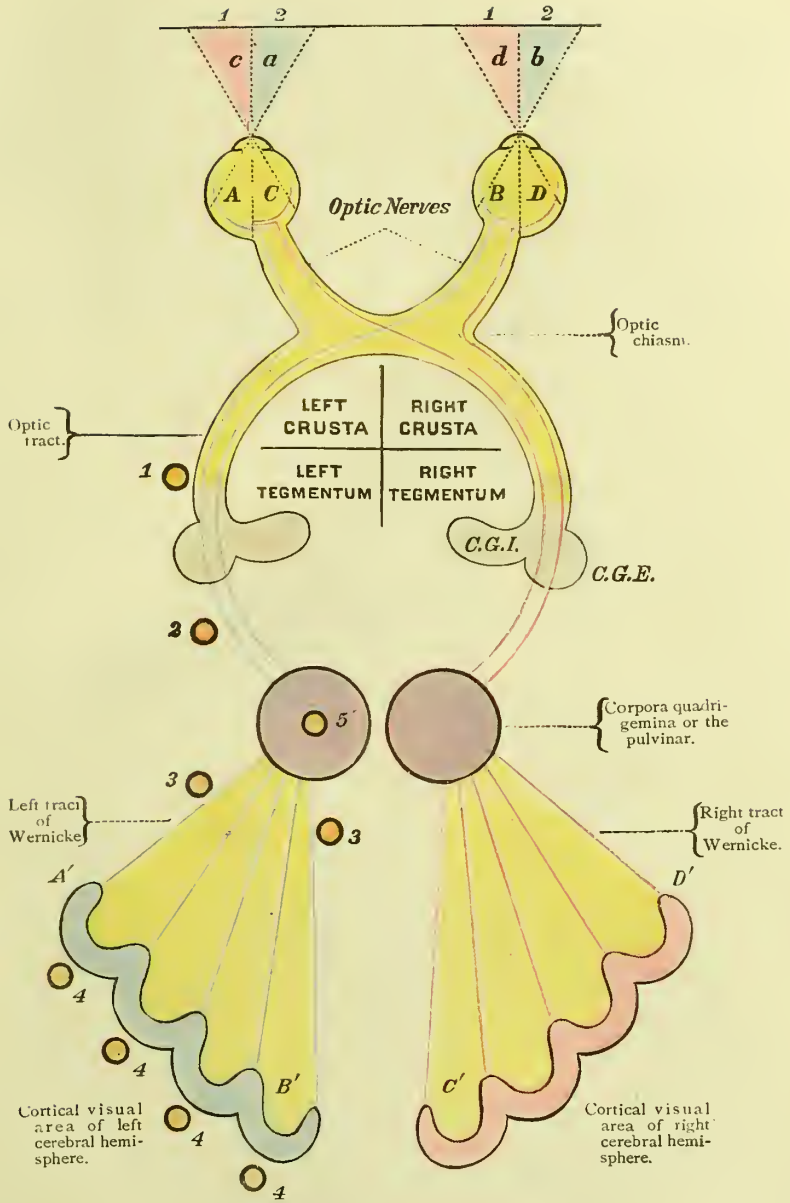


FIG. 21.—A DIAGRAM BY THE AUTHOR EXPLICATIVE OF HEMIANOPSIA. The lines (*A* and *B*) indicate the fibres associated with the left cerebral hemisphere. Those of the right hemisphere (*C* and *D*) appear as separate lines. Both will be seen in the diagram to pass from the retina through the following parts: The optic nerves; the crossing fibres through the optic chiasm; the optic tracts; the external geniculate body; the corpora quadrigemina or the "pulvinar" of the optic thalamus; and the internal capsule. The fibres are shown to end in the cortex of the occipital lobes.

A lesion situated at the points designated as 1, 2, 3, 4, and 5, will cause homonymous hemianopsia. Lesions of the *right hemisphere* of the cerebrum produce blindness of the *right half* of each eye, and *vice versa*

Lesions at the base of the skull frequently produce this variety of hemianopsia, if they lie posteriorly to the optic chiasm. In this situation, the lesion usually produces the symptoms which are referable to pressure upon, or destruction of the bundles of motor or sensory fibres found in the crus and below it, or some of the nerves which escape from the base of the skull. Fig. 21 will aid the reader in appreciating the clinical value of this suggestion.

Whenever the chiasm is affected, we meet the binasal type. Regions *c* and *b*, in Fig. 21, would then be deprived of visual perceptions.

There is still one more form of hemianopsia which is occasionally encountered, viz., the bitemporal type. This has been interpreted by

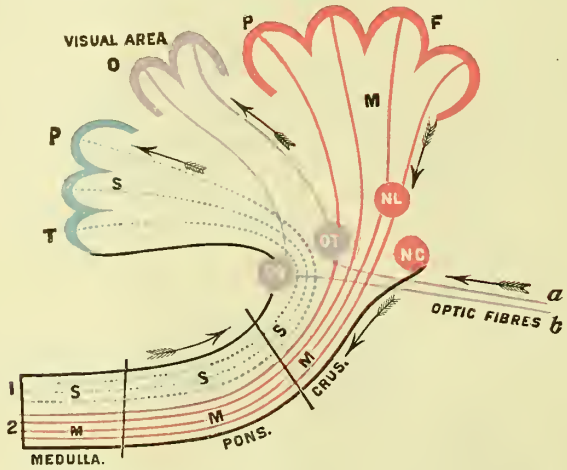


FIG. 22.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE GENERAL COURSE OF FIBRES OF THE "SENSORY" AND "MOTOR TRACTS," AND THEIR RELATION TO CERTAIN FASCICULI OF THE OPTIC NERVE TRACTS. (Modified from Seguin.) S, Sensory tract in posterior region of mesocephalon, extending to O P and T, occipital, parietal, and temporal lobes of hemispheres; M, motor tract in basis cruris, extending to P and F, parietal and (part of) frontal lobes of hemispheres; C Q, corpus quadrigeminum; O T, optic thalamus; N L, nucleus lenticularis; N C, nucleus caudatus; 1, the fibres forming the "tegmentum cruris" (Meynert); 2, the fibres forming the "basis cruris" (Meynert); *a*, fibres of the optic nerve which become associated with the "optic centre" in the optic thalamus, and are subsequently prolonged to the "visual area" of the occipital convolutions of the cerebrum; *b*, optic fibres which join the cells of the "corpora quadrigemina," and are then prolonged to the visual area of the cerebral cortex.

an autopsy made upon a case intrusted to the care of Professor H. Knapp, of this city. It must be evident that the chances would, of necessity, be extremely small of ever encountering a bilateral lesion which would affect only those fibres of the optic chiasm or optic tract which supply the temporal half of each retina, and at the same time leave the decussating fibres intact. How, then, are we to account for the fact that this form is sometimes met with? I would call attention to a peculiar arrangement of the arteries in the region of the optic chiasm as a factor in causing this condition. It has been shown that atheromatous degeneration of the "circle of Willis" (a peculiar arrange-

ment of blood-vessels at the base of the brain) so impairs the elasticity of the arteries as to create through their pulsation a type of injury to the chiasm, so limited in its extent as to impair only the fibres distributed to the temporal halves of the retinæ, and thus to create bitemporal hemianopsia.

Hemianopsia will be more fully discussed in connection with the effects of lesions of the optic nerve. The diagrams introduced will, I trust, make the facts stated clear to the mind of the reader.

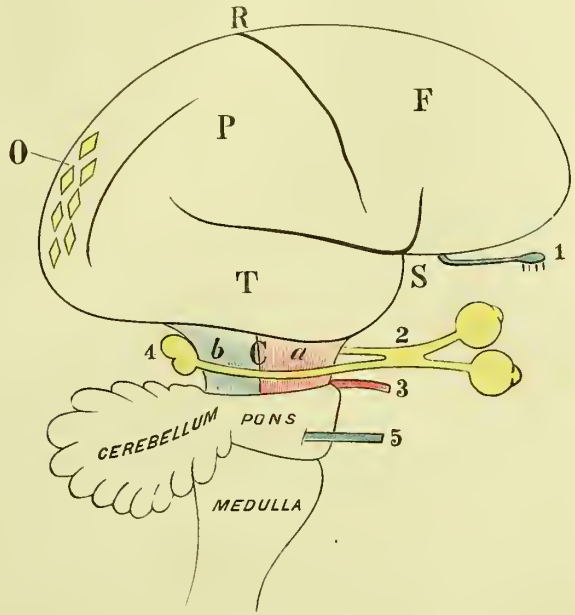


Fig. 23.—A Diagram Designed by the Author to Show some of the Relations of the Optic and Olfactory Nerve Fibres to Surrounding Parts. F, Frontal lobes of cerebrum; P, parietal lobe; T, temporo-sphenoidal lobe; S, fissure of Sylvius; R, fissure of Rolando; O, occipital lobe; C, cerebellum; M, medulla oblongata; 1, olfactory nerve; 2, optic chiasm; 3, motor-oculi nerve; 4, corpora quadrigemina; 5, trigeminus nerve; *a*, basis cruris; *b*, tegmentum cruris. The diamonds in the occipital lobe, the *cortical visual centres of Munk*. The cerebellum and pons Varolii are shown as if separated from the cerebrum, in order to made the relations of the crus to the optic tracts apparent. This diagram should be compared with the preceding ones (Figs. 21 and 22) to make its bearings upon cerebral localization apparent.

We may, therefore, summarize the clinical significance of this peculiar form of blindness as follows: (*a*) The homonymous variety indicates lesions affecting the optic tract or its continuation backward; or, possibly a lesion of the cortex of the occipital lobe of the same side. (*b*) The binasal variety indicates a lesion pressing upon the central portion of the chiasm. (*c*) The bitemporal variety indicates atheromatous degeneration of the circle of Willis. Symmetrical lesions of the outer part of the chiasm might possibly (?) also cause it.

APHASIA.

An impairment of the idea of language or its expression (independent of paralysis of the tongue) constitutes this condition.

It is commonly described as of two varieties—the sensory or "amnesic form," in which the memory of words or of symbols is more or less effaced, and the motor or "ataxic" variety, in which the memory is perfect, but the subject cannot properly pronounce words, from an inability to perfectly coördinate the muscles concerned in articulation.

The symptoms of this malady in either of its forms are always of great clinical interest, because some peculiarity in each case causes it to differ from others which may have been previously encountered.

I quote from the third edition of my work on "Surgical Diagnosis," some selected paragraphs relating to this symptom, with occasional changes in their phraseology:—

"In the *amnesic variety* the most familiar objects are commonly misnamed; the subject being oftentimes aware that the error has been committed, and yet is not able to correct it. The form which this loss of memory takes is liable to vary with each case. As an illustration of this, some forget only names; others only numbers. In certain reported cases, the names of things only in dead or foreign languages were retained; in others, the reverse had been observed, the patient losing all memory of acquired tongues. Again, the sound of words often will not be recognized when the letters which form them will; and the reverse of this condition is not infrequently met with in aphasic subjects.

"We owe to Broca the credit of the discovery that the centre for the *coördination of the movements* of the tongue, lips, and palate, necessary to articulate speech, could be located in the posterior portion or base of the third frontal convolution; and to many of the later pathologists the debt of overthrowing what once was the popular view, viz., that this centre is not confined exclusively to the left cerebral hemisphere. Subsequent pathological observation seems to have added strength to the view that lesions of the 'island of Reil,' as well as the medullary substance which intervenes between it and the centre of Broca, must be included in the so-called 'motor speech area.' The amnesic form may be dependent likewise upon lesions of the so-called 'sensory areas' of the cortex.

"The 'centre of Broca' is supplied with blood by the middle cerebral artery. An embolus within that vessel will tend, therefore, to arrest the circulation, of that important area, and, at the same time, it will interfere more or less with the nutrition of the motor area of the cortex and the corpus striatum—the ganglion which probably modifies all motor impulses sent out from the brain to the muscles of the opposite side of

the body. Now we know clinically that embolism is a frequent cause of aphasia, and that hemiplegia almost always accompanies it. We also know that the middle cerebral artery of the left side is the most frequent seat of embolic obstruction. This fact helps us to interpret the development of right hemiplegia in connection with aphasia, as is found to exist in the large proportion of such cases. Seguin found two hundred and forty-three cases in which right hemiplegia existed out of a total of two hundred and sixty—left hemiplegia being present in but seventeen cases.

"In the *ataxic variety* of aphasia, the patient can usually write what cannot be spoken, thus proving that the memory of words seen or heard is not effaced, but rather the ability to so coördinate the muscles of speech as to properly pronounce them. This condition must not be confounded with aphonia (loss of voice). Several cases have been reported where the amnesic form has given place to the ataxic, and the lesion has been found over the centre of Broca. It would seem, therefore, that the third frontal convolution (although placed in close relationship with the oral and lingual centres of Ferrier) has some imperfectly understood connection with the memory of words, as well as with the coördinated movements of the apparatus of speech.

"If irritative or destructive lesions of the cerebral cortex exist as the exciting cause of the aphasia, *convulsions* may be associated with its development.

"If numbness or anæsthesia co-exist with hemiplegia and aphasia, it indicates that the 'motor and sensory tracts' which connect the cerebrum with the extremities are involved, as well as the centre of speech, or the 'speech tract.'"

We have reason to believe that the cortical cells of the so-called "sensory area" of the cerebrum not only enable us to appreciate the many facts telegraphed to them by the organs of smell, sight, hearing, taste, and touch; but that each cell is able furthermore to store up such impressions as it is specially designed to take cognizance of, and to recall them at the command of the will as *memories of past events.*

Munk has lately demonstrated that the cortical cells of the occipital lobes preside over vision; and that a permanent loss of sight follows the total destruction of these lobes. The same observer has shown, also, when a circumferential ring of cells in the occipital lobes were not included in the experiment (the central portions only of the lobes being removed), that an animal will slowly regain its familiarity with surrounding objects through the sense of sight. A dog, for example, will learn to again recognize faces, can be taught anew to fear the lash, to recognize food by sight, etc. The only explanation of such facts is that the new sight-memories are formed in place of those that were obliterated by the operation.

Experimental investigation and pathological facts lead us to the conclusion that the *various forms of memories* recognized are stored up in those cortical cells which were originally thrown into activity by the fact remembered. Thus, for example, the cells of the area of hearing give to us our memories of sound; those of the sight area our memories of visual impressions; and those of the smell area our memories of odor. Some remarkable clinical facts sustain the view that the cells of the motor area even are capable of giving to us memories of muscular efforts. These are totally distinct from other forms of memory. Professor Charcot lately reported a case where a gentleman could read by tracing the lines with his finger, when a lesion of the brain had deprived him of his ability to recognize written or printed characters by sight. He could write with ease, but could read what he had written only by retracing the lines, or going through the motions necessary to reproduce the letters.

Dr. M. A. Starr has lately written two popular articles,* in which the physiology of speech is discussed at some length. It is illustrated with some admirable diagrams. This author cites many interesting cases which illustrate the various types of aphasia, and he supports the view that the parietal convolutions, which are not related to muscular movements, are the seat of our conscious appreciation of tactile impressions and of touch-memories. Ross, Hughlings-Jackson, Bastian, Broadbent, Kussmaul, and others, have written extensively upon this subject.

Much light has been shed by recent investigations upon those cases of aphasia where the ability to respond to spoken questions has been destroyed by focal lesions of the brain, and the patient has still been able to appreciate written interrogations and to reply to the same. Cases also where the reverse has been observed, are now understood.

The condition known as "*word-deafness*" is to be clinically regarded as a symptom of a lesion affecting the superior temporal convolution, in which the centres of hearing are situated. The condition known as "*word-blindness*" indicates a lesion of the occipital lobes.

The centre of Broca must, therefore, be regarded as related exclusively to *motor speech memories*, which can be called into activity by the different parts of the cortex in case any impression received by them demands a verbal response.

We are in possession of facts to-day that render it certain that the nuclei of origin within the medulla oblongata of the nerves which preside over the tongue, lips, and palate, are connected with those cortical centres that are functionally related to speech by the fibres of the so-called "*speech tract.*" Wernicke has lately traced the course of these fibres by a study of reported cases which bear upon this field. He places them in the *posterior part of the internal capsule* (Fig. 24), and states that they

* *Popular Science Monthly*, Sept., 1884; *Princeton Review*, May, 1886.

pass also through the *external capsule* to reach the third frontal convolution. This discovery enables us to explain the co-existence of aphasia with hemianæsthesia and hemianopsia, which has been observed. It also clears up those cases where lesions of the crus, pons, and medulla have produced aphasic symptoms. The "speech tract" must not be confounded with the cerebral extension of fibres of the hypoglossal nerve, which have a different course.

Clinically, we may be called upon to recognize the following varieties of defective speech produced by brain lesions:—

(1) Motor or "Ataxic" Aphasia.	
(2) Sensory or "Amnesic" Aphasia. .	Paraphasia. Word-deafness. Word-blindness. Agraphia.

PARAPHASIA.

This is a condition where the *substitution of wrong words or symbols* occurs in conversation or during attempts at writing. The patient is conscious of this error, but is unable to correct it. Nouns are more frequently lost than verbs. Patients of this type often exercise great ingenuity in avoiding, during conversation or writing, the words which they are liable to fail in properly recalling. By means of oddly-constructed sentences they will often hide this defect in speech from strangers. A good test often for such cases is to request the patient to say his alphabet, and to count until requested to cease. These patients will probably substitute wrong letters or figures for the proper ones.

Paraphasia is due to a lesion of the island of Reil. On the left side of the brain in right-handed subjects, or *vice versa.*

TRUE MOTOR APHASIA.

This condition is due to a lesion in the centre of Broca (third frontal convolution).

These patients cannot articulate correctly. They are painfully conscious of this defect; hence they frequently become mute rather than to expose themselves to criticism or ridicule. I have known such patients to be deemed a melancholiac because they could not be induced to talk.

In case certain words are retained or regained after the attack, these words or expressions are used in a peculiarly automatic way by the patient in reply to any question asked. It is not uncommon for a patient with motor aphasia to use some absurdly irrelevant phrase as an answer to any question which may be propounded. Occasionally, this phrase may be traced to some peculiar expression which existed in the

mind of the patient at the time when the attack occurred. As examples, a case is reported by Hammond where a patient would reply "hell to pay" under all circumstances; and another by Hughlings-Jackson where the unvarying reply was, "I want protection."

AGRAPHIA.

This term is applied to a condition where, from cerebral disease or other causes, the *power of writing* is suddenly or gradually lost.

The explanation of this condition rests in the fact that the patient has *lost certain memories* which previously enabled him to make the necessary finger movements for placing upon paper results obtained by his mental processes.

Such a person might be able to perform any or all movements of the fingers (that are not connected with the writing of letters or figures) with his accustomed delicacy. He cannot write from dictation, or copy from a printed or written slip. He is not paralyzed, nor is he affected with "writers' cramp." The memory is alone at fault; hence this condition is a variety of "amnesic aphasia."

In some instances, delicate finger movements required in the mechanical trades, the use of musical instruments, etc., have been known to be suddenly taken away from a similar loss of motor-memories. Such cases are not included under the term "agraphia."

WORD-DEAFNESS.

This is a form of sensory aphasia which is due to a lesion of the first temporal convolution. These patients cannot be made to *understand spoken language*, because their centres of hearing have been impaired. They are not deaf to sound, but they fail to appreciate the meaning of certain sounds. Their own tongue is as unintelligible to them as a foreign language.

This condition prevents the patient from speaking correctly, because of an inability on his part to recall the proper sound of many words previously employed by him. Their efforts to talk or to read aloud, result in an "unintelligible jargon" which the patient does not recognize as in any respect unnatural or inexpressive of ideas he desires to communicate to you, because his ear does not properly interpret his own utterances.

You may test such a patient, therefore, by asking him to read aloud some printed selection, or to write at your dictation. With neither of these tests will he be able to fully comply.

Starr quotes from Broadbent the following illustrative case:—

"One such person was asked to read the sentence, 'You may receive a report from other sources of a supposed attack on a British consul-

general. The affair is, however, unworthy of consideration.' He read it slowly, and in a jerky manner, as nearly as could be taken down, thus: 'So sur wisjee coz wenement apripsy fro freuz fenement wiz a seconce coz foz no Sophias a the freckled pothy conollied. This affair eh oh cont oh curly of consequences.' It was evidently an effort to read aloud, requiring close attention, and he read seriously and steadily, apparently unconscious of the absurdity of his utterances, till interrupted by laughter, which it was impossible to restrain. He was never able to write at dictation, but he signed his name quite well, and could copy accurately, though as he wrote each letter he would attempt to name it aloud, but always pronounced a wrong letter."

WORD-BLINDNESS.

This is another form of sensory aphasia. It is due to a lesion of the visual centres in the occipital lobes. It is accompanied by a loss of memory of the meaning of *printed* or *written symbols*. Such patients can generally recognize familiar objects or faces, but they cannot read correctly.

When asked to read a printed selection or a written slip, their interpretation is an incorrect one They can often write from dictation, but they cannot read what they have written. Their conversational powers are not impaired unless "word-deafness" is also present.

The following illustrative case is quoted by Starr, from a contribution of Ross upon this condition:—

"One man who had suffered from this affection seemed at first unconscious of his actual condition. When asked to read he would make very elaborate preparations, putting on his spectacles and moving the paper or book backward and forward until he seemed to get it into a position where he could see well. He would then read aloud, uttering a few sentences which had not the remotest connection with anything that was before him on the printed page. He was handed a note which read as follows: 'Dear Sir, I shall be much obliged if you will let me know whether or not you consider it likely that A. B. will recover.' He looked at it carefully, and seemed to glance it through, and then read slowly and deliberately, and without much hesitation: 'Dear Sir, You are requested to bring this note with you the next time you come to the infirmary;' and then he added, 'that is what I make of it; I don't know whether it is right or not.' He often tried to read a newspaper aloud, and his wife said that he 'read a lot of stuff all made up out of his own head.' On one occasion she took the paper and read it to him. He was very quiet for a time, and then asked, 'Is that what it says in that paper?' and when she assured him that it was, he said, 'Well, then, I must be an idiot.' At that time he would remark, 'I don't know what is the matter

with the newspapers nowadays, they are filled with such silly stuff.' Soon, however, he began to realize that the trouble lay in himself rather than in the papers, and then he gave up attempting to read."

It is not uncommon to encounter this form of aphasia in conjunction with word-deafness, a fact which is easily explained by the close proximity of the visual and auditory centres (see Fig. 5).

GENERAL DEDUCTIONS RELATIVE TO APHASIA.

In summary, the following deductions relative to disorders of speech may be given :—

1. The cortex of the posterior part of the *third frontal convolution*, and possibly the *island of Reil* also, presides over the coördination of such muscular acts as are necessary to speech. It also stores the memories of such acts, so that any combination of articulate sounds can be voluntarily reproduced when the proper form of excitation is furnished (chiefly in response to sight or sound-impressions).

This centre is connected by "associating fibres" with the centres of hearing (first temporal convolution) and those of sight (the occipital convolutions). It is also put in communication with the nuclei of the facial, hypoglossal, pneumogastric, and glosso-pharyngeal nerves (within the medulla) by means of two distinct tracts of fibres, viz., the "hypoglossal cerebral tract," and the so-called "speech tract."

Thus, this cortical centre of coördinated speech-movements is capable of receiving excitation from the centres of hearing, when replies to spoken language are demanded; and from the centres of sight, when written or printed language calls for a verbal response. It is also put in direct communication with the nerves which preside over the apparatus of speech (whose nuclei of origin are situated within the medulla).

2. The form of amnesic aphasia, known as "*word-deafness*" (Kussmaul) indicates the existence of a lesion of the first temporal convolution* of the left side, which has impaired the memories of spoken language. Hearing may not be impaired, in spite of the fact that the appreciation of words, music, etc., may be totally absent.

3. The condition known as "*word-blindness*" (Kussmaul) indicates the existence of a lesion of the left occipital convolutions, which has impaired the memories of written or printed symbols of language, numerals, familiar objects, etc.

4. The condition termed "*paraphasia*" by Kussmaul (in which the amnesic and ataxic varieties of aphasia seem to be peculiarly combined) may be excited by a lesion which interferes with the action of the asso-

* In right-handed subjects the left hemisphere, and in left-handed subjects the right hemisphere, seems to monopolize the function of sound-interpretation to the speech centre.

ciating tracts of fibres between the areas of hearing or sight and the motor speech centre of Broca (Wernicke).

5. The condition of imperfect speech, termed "*anarthria*," is produced by a lesion of the medulla, which interferes with the functions of the nuclei of the cranial nerves associated with speech. It is occasionally observed in connection with focal lesions of the floor of the fourth ventricle. These cases are to be differentiated from aphasia of cortical origin by the co-existence of other symptoms produced by the medullary lesion (see subsequent page).

6. In order to properly pronounce any word, it is essential that both the *cortical centre* of speech, and also the *nuclei of the medulla*, which are associated with it, must be called into action.

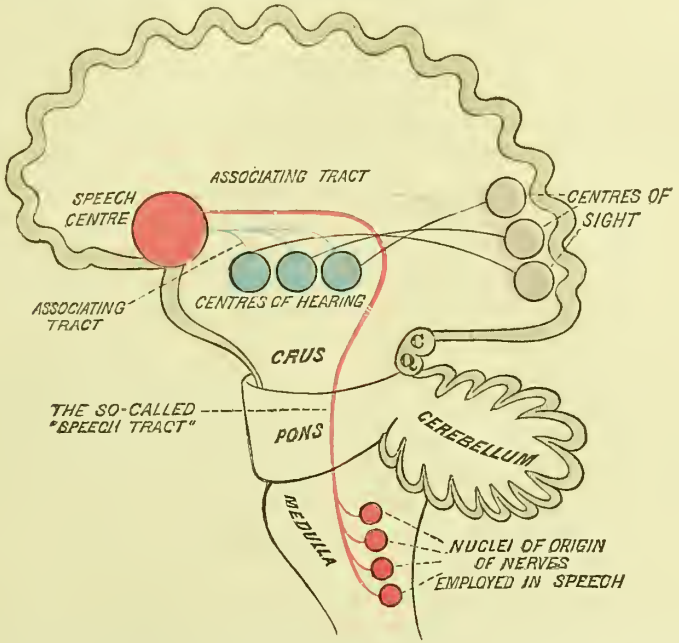


FIG. 24.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE MECHANISM OF THE APPARATUS REQUIRED IN SPEECH.—The reader must not regard this diagram as intended to accurately portray the anatomical relations of the various centres and tracts to each other.

7. The peculiar course which the fibres of the "*speech tract*" take within the cerebral hemisphere, sheds light upon those reported cases of aphasia where the lesion was situated posterior to the centre of Broca, These fibres run from the third frontal gyrus close to the surface of the hemisphere, and in an antero-posterior direction (passing in the external capsule) to reach the posterior part of the lenticular nucleus. They dip at this point into the posterior part of the internal capsule. They then pass through the middle part of the crus and pons to the medulla

(Wernieke). Within the internal capsule, the fibres of the "speech-tract" lie (according to this observer) between the optic fibres and those of the sensory tract.

8. Should aphasia be developed, as a result of a *lesion of the internal capsule*, hemianopsia, or hemianæsthesia, would be liable to coexist, on account of the relationship of the optic and sensory fibres of the capsule to the speech tract.

9. It is possible to have aphasic symptoms develop as a result of a *lesion within the crus or pons*. This is because the speech tract passes through them to reach the medulla.

10. The cortical centres of hearing, smell, and taste, are probably associated (wholly or in part) with the corresponding organ of the opposite side. Hence, we may clinically refer an abolition of the function of hearing (in case it be due to a cortical lesion) to the hemisphere opposed to the deaf ear. "Word-deafness" may ensue, however, when the centres of hearing of only one cerebral hemisphere are involved. In right-handed subjects, the left superior temporal convolution appears to govern this function; while, in left-handed subjects, the right superior temporal convolution assumes it. This is probably due to the fact that the hemisphere which is the most exercised, becomes more rapidly developed.

11. When the *third frontal convolution* is alone diseased, the patient will be able to understand spoken or written questions perfectly, but will not be able to properly regulate the movements of the speech apparatus that are requisite to a reply.

12. When the *superior temporal convolution* is alone diseased, the patient cannot recognize or properly interpret spoken language. He may, however, be able to repeat *single words* when propounded, but not sentences. Exclamations of various kinds may be uttered by these subjects when irritated or distressed; but they are more or less involuntary, and often irrelevant. The efforts of these patients to talk or read aloud are peculiarly unintelligible.

13. When the *associating fibres* between the centre of hearing and the centre of Broca are alone diseased, the patient can comprehend written or spoken language perfectly; but, in talking, such a subject is apt to interpolate, from time to time, some irrelevant and unexpected word in a sentence in place of the one desired. The effects of destruction of the associating tracts of the cerebrum will be discussed later.

A SUMMARY OF THE DIAGNOSTIC SYMPTOMS BY WHICH LESIONS OF THE BRAIN MAY BE LOCALIZED DURING LIFE.

The contents of the preceding pages will probably enable the reader to appreciate the grounds which justify the following conclusions respecting the diagnosis of focal brain lesions.

Frequent references will be made in subsequent sections of this volume, to these clinical deductions; hence the importance of a thorough mastery of the closing pages of this section cannot be too strongly impressed upon the reader.

CORTICAL LESIONS OF THE CEREBRUM.

Lesions of the motor convolutions, when of small size, produce some form of *monoplegia*, or *mono-anæsthesia*; when of large size, a *hemiplegia* may be produced.

Consciousness is not necessarily lost at the time of the attack. As a rule, the patient is not rendered totally unconscious.

Early rigidity of the paralyzed muscles is often present. This is probably due to irritation of the cortex.

Cortical hemianæsthesia will be produced when the entire parietal cortex is involved by a cortical lesion, and, in addition, the balance also of the motor area, which lies outside of the parietal lobe. Such an extensive cortical lesion is rarely, if ever, encountered. We, therefore, do not observe co-existing hemiplegia and complete hemianæsthesia in cortical disease.*

Localized pain in the head is a symptom which is often present in connection with cortical lesions. If it be absent, percussion over the lesion will generally tend to excite. This step will also tend to increase the pain, in many cases, where it exists prior to this test.

* The experiments of Munk, made with a view of determining the area of common sensation in the cerebral cortex, lead to the conclusion that the entire parietal cortex must be destroyed, and the ascending frontal convolution as well, before complete and permanent anæsthesia is produced on the opposite side of the body below the head. These results make the motor area overlap the sensory area to some extent, and tend to refute the deductions of Ferrier, who places the centre of tactile sensations in the temporal lobe, and to confirm the views held by Luciani and Exner. If a partial destruction of the sensory area of Munk be produced in animals, the anæsthesia persists only for a few weeks, because the adjacent regions learn to perform vicariously the functions of the part destroyed.

Tripier, of Montpellier, France, has lately affirmed the conclusions of Munk, respecting the existence of sensory centres in the central convolutions, as has, also, Moeli, of Berlin. These three observers support the view that *the motor and sensory centres of any one limb coincide*. This view was advanced theoretically by Luys some years ago.

Exner has collected from European journals all cases of cortical disease that have been associated with disturbances of sensation, and M. Allen Starr has lately performed the same labor in American literature. An analysis of the cases so collected seems to justify the conclusions of Munk and his followers, and to add some clinical suggestions of value. These cases demonstrate (1) that the cerebral cortex of each hemisphere appreciates *sensory impressions from both sides* of the body, but are chiefly associated with the sensory tracts of the opposite lateral half of the body; (2) that the sensory area includes the central convolutions (Fig. 4) and the posterior part of the parietal lobe; (3) that the sensory centres coincide to some extent with the motor centres of similar parts; (4) that no disturbances of general sensation have been known to result from lesions confined to the frontal, temporo-sphenoidal, or occipital lobes.

Convulsions, when followed by *transient attacks of paralysis* (Jacksonian epilepsy), indicate an irritative lesion of the cortex. They are frequently encountered in connection with syphilitic disease of the brain. Subjective sensations (paræsthesiæ) may also be excited in limited portions of the limbs.

Blindness of that half of each retina, which corresponds to the cerebral hemisphere affected, occurs when extensive cortical disease of the cuneus in the occipital lobe is present. "*Word-blindness*" may also be produced by lesions of these lobes (especially if upon the left side).

Abolition of hearing, and also the condition known as "*word-deafness*" occur from lesions of the first temporal convolution (chiefly upon the left side).

Abolition of the sense of smell, or of taste, may result from lesions of the tip of the temporal lobe. The memories of taste-and-smell-perceptions may also be impaired or lost.

Ataxic aphasia and *paraphasia* may be developed as a result of cortical lesions, which involve respectively the speech centre of Broca and the island of Reil.

The *face is never rendered totally hemiplegic* by cortical lesions; as far as my clinical observation and research among reported cases goes to show.

The conditions known as "*mono-anæsthesia*," by which we mean an impairment, or total arrest of sensation in some distinctly localized part, as, for example, the hand, arm, leg, etc., and, also, the condition known as "*mono-paræsthesia*," which signifies the existence of subjective sensations of a definitely localized character, are particularly diagnostic of cortical lesions lying *posterior to the fissure of Rolando*. The former indicates a destructive lesion, the latter an irritative lesion.

The *muscular sense* is liable to be impaired (when a *cortical lesion of the motor area* exists) in the parts functionally associated with the limits of the part diseased.

Monoplegia and *monospasm* are peculiarly diagnostic of a cortical disease *anterior to the fissure of Rolando*.

The *memories of sensory impressions* are more frequently impaired by cortical lesions of the left hemisphere than of the right (as shown, for example, in ataxic aphasia, word-blindness, word-deafness, paraphasia, etc).

Motor memories may be impaired by cortical disease affecting the motor area. Subjects may thus lose a dexterity with the fingers, arm, hand, leg, etc., which they had acquired previous to the development of the lesion. A knowledge of this fact may sometimes aid in the localization of a lesion.

Irritative lesions of the cortex of the cuneus (a part of the occipital

lobes) may cause *hallucinations of vision.* If one hemisphere only is affected, the objects seen will appear to lie on the side opposed to the lesion, and to move with the eyes as they are turned from side to side.

Lesions of the "*island of Reil*," or "*insula*" of the left side (Fig. 9), seem to create (in some instances) symptoms of *ataxic aphasia*, and also *paraphasia* (the substitution of wrong words). The *motility of the face and arm of the opposed* side may occasionally be impaired from cortical lesions of this region.

Lesions of the cortex confined to the *apex of the temporal lobe* (Fig. 3) are liable to cause an impairment of the *sense of smell or of taste* (if destructive in character); or subjective odors and tastes (if irritative in character).

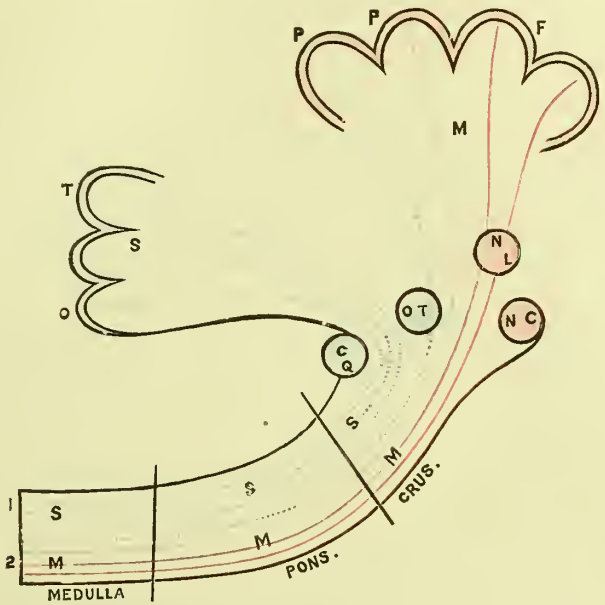


FIG. 25.—A DIAGRAM DESIGNED TO ILLUSTRATE THE GENERAL COURSE AND DISTRIBUTION OF THE MOTOR AND SENSORY TRACTS OF THE CEREBRUM. (Modified slightly from Seguin.) P, Parietal lobes, F, frontal lobes, T, temporal lobes; O, occipital lobes; M, motor bundles, S, sensory bundles, N C, nucleus caudatus, N L, nucleus lenticularis; O T, optic thalamus, C Q, corpora quadrigemina, 1, sensory (posterior) bundles of the medulla, pons, and crus, 2, motor (anterior) bundles of the same. Note that the motor fibres are associated with the frontal and parietal lobes; and the sensory fibres with the parietal, temporal, and occipital lobes

Destructive lesions of the cortex of the *motor convolutions* (Fig. 5) are followed by a *descending degeneration* of the fibres which arise from these gyri. This may account (?) for the late rigidity of the muscles paralyzed, which is occasionally observed after such lesions.

Cortical lesions of the *base of the brain* are especially liable to produce vomiting, choked disc, bilateral paralysis, and symptoms of impair-

ment of some of the cranial nerve trunks. The crus, pons, and island of Reil may also be involved and give additional symptoms.

Cortical disease of those frontal gyri *which lie anteriorly to the motor centres* (Fig. 5) is often attended with no marked symptoms of a diagnostic character. The higher mental faculties may occasionally give signs of more or less impairment. Connected thought, the control of the emotions, accurate reasoning, and concentration of the attention are particularly difficult under such circumstances.

The *memories* of sound- or sight-impressions, as well as those of smell, taste, muscular movements, etc., may be separately annihilated by cortical disease (see Fig. 5).

NON-CORTICAL LESIONS OF THE CEREBRUM.

Many of the clinical facts pertaining to non-cortical cerebral lesions may be thus summarized:—

Profound coma is more often encountered in non-cortical lesions than in cortical; possibly because the cerebro-spinal fluid is more liable to be displaced from the ventricles (Duret).

Hemiplegia commonly exists in *combination with more or less hemi-anæsthesia*, and *paresis of the lower part of the face.* These symptoms are observed, as a rule, upon the side of the body opposed to the cerebral lesion.

Pain, when present in the head, is less circumscribed than in cortical disease; and is not increased by percussion; or, when absent, elicited by that step.

Muscular rigidity in the paralyzed muscles develops late. Typical *monoplegia* is probably never observed.

Tremor, *hemichorea*, and *athetosis* are not uncommon sequelæ of non-cortical cerebral lesions.

The *senses of sight*, *smell*, *hearing*, *and tactile sensibility* are occasionally impaired to a greater or less extent by non-cortical lesions. The seat of the lesion will modify the evidences of such impairment, because the fibres of some of the cranial nerves may be involved by the lesion, while others may escape injury.

Typical attacks of Jacksonian epilepsy do not occur; although *general convulsions* may be excited.

LESIONS OF THE EXTERNAL CAPSULE.

These may cause (if within the left cerebral hemisphere) the condition of *paraphasia*, which has been previously described. This is due to the fact that the "speech tract" probably passes through it before it enters the internal capsule.

LESIONS OF THE INTERNAL CAPSULE.

These often result in the development of hemiplegia, hemianæsthesia, or a combination of the two. Hemiparalysis of the lower half of the face may be produced. The nerve fibres of sight, hearing, and smell, and the so-called "speech tract" may be implicated. Conjugate deviation of the head and eyes is not infrequent. "Choked disc" may accompany this condition, because it is a clinical evidence of an excess of intracranial pressure. The different forms of tremor already mentioned are most common when the internal capsule is implicated. Paraphasia may be induced, if the "speech tract" is involved.

LESIONS OF THE CAUDATE NUCLEUS.

These are seldom, if ever, associated with hemianæsthesia. Hemiplegia, if developed, is probably due to pressure upon the motor fibres of the internal capsule. The face may develop paralysis in its lower part upon the opposed side for the same reason.

Many of the symptoms enumerated above (as indicative of a capsular lesion) may exist also when the caudate nucleus, the lenticular nucleus, or the thalamus, are individually attacked by any lesion which markedly increases their size, and thus creates pressure upon the fibres of the internal capsule (see page 22).

LESIONS OF THE LENTICULAR NUCLEUS.

These chiefly affect motility. Hemianæsthesia may occur if the posterior capsular fibres be pressed upon.

Hallucinations are very common in connection with disease of the thalamus (Ritti). The senses of sight, hearing, smell, and tactile sensibility are perhaps more liable to be affected than motility.

LESIONS OF THE CRUS CEREBRI.

The symptoms which point to a lesion of the crus (Fig. 11) may be summarized as follows :—

Crossed paralysis of the "third nerve and body type" never occurs except from a lesion of the crus.

If the lesion be confined to the *tegmentum cruris* (the sensory portion), *hemianæsthesia* of the opposite side will ensue, and the third and fifth cranial nerves of the same side may possibly be paralyzed. Incoördination may be developed; provided that the fillet (lemniscus) is involved.

If the *crusta cruris* (the motor portion) be alone involved, paralysis of the third nerve will generally co-exist with hemiplegia on the opposed side. The lower part of the face may be rendered paretic, in some instances, showing that filaments of origin of the facial nerve are impaired.

Symptoms referable to lesions of the *corpora quadrigemina* may be developed in connection *with lesions of the tegmentum cruris* (Fig. 11). Among these, the following may be prominently mentioned: Incoördination of movement; abolition of the pupillary reflex; nystagmus; and strabismus. Blindness may be found to exist independent of the presence of a choked dise, atrophy of the optic nerve, or an optie neuritis.

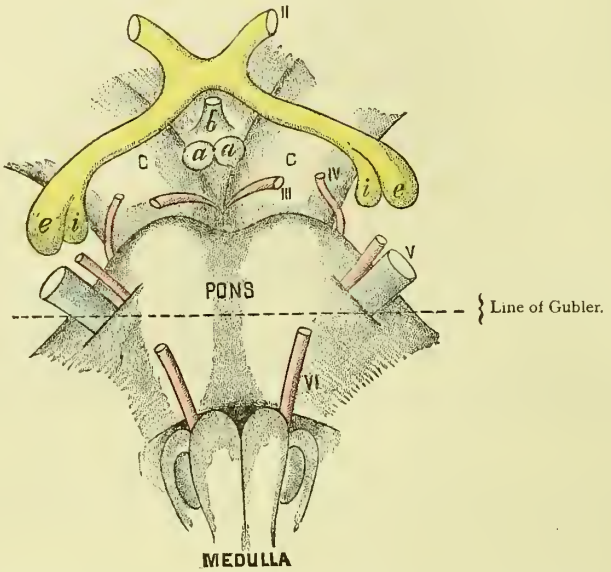


Fig. 26.—A Diagram of the Base of the Brain, Designed to Show the Parts Adjacent to the Optic Nerve Tracts and Chiasm.—The nerves are represented by their respective numbers. II., optic; III., motor oculi; IV., trochlearis; V., trigeminus, VI., abducens; C, crus cerebri of each hemisphere; *b*, infundibulum, the pituitary body being cut off to show the optic chiasm; *a*, the corpus albicans (mamillary tubercle); *e*, external geniculate body; *i*, internal geniculate body. The dotted line which crosses the pons Varolii, connecting the roots of the fifth nerves, is Gubler's line, an important guide, since lesions of the *pons* in front of it cause "crossed facial paralysis." Lesions in the region of the *crus* may involve the third and second nerves simultaneously. Lesions about the chiasm may press upon the corpus striatum within the mass of the cerebrum. The crus comprises both the motor and sensory tracts of the cerebrum.

LESIONS OF THE PONS VAROLII.

Apoplectie elots and foci of softening are not infrequently met with in this region (Fig. 13), and tumors are sometimes encountered. Certain clinical deductions of value can be drawn from a study of reported lesions of the pons, as follows:—

The imaginary line that conneets the apparent origin of the trigeminal roots (line of Gubler) marks the level of decussation of the fibres of the facial nerves that pass cephalad.

Lesions above the line of Gubler are liable to produce facial palsy and hemiplegia upon the same side of the body (the one opposed to the lesion).

Lesions below the line of Gubler produce "crossed paralysis of the seventh nerve and body type," the face being paralyzed upon the same side as the lesion, while a hemiplegia is developed upon the opposed side of the body.

The *trigeminus nerve* may be paralyzed by lesions of the pons, if it lies within the inner two-thirds of the reticular formation (according to the researches of Starr).

If such a lesion be situated high up in the pons, trigeminal paralysis will co-exist with a hemianæsthesia of the opposed half of the body; if situated low in the pons, the trigeminal paralysis and the hemianæsthesia will be upon the same side. The point of union of the ascending and descending roots of the fifth nerve is nearly at the level at which the fifth nerve escapes from the pons (line of Gubler).

Difficulties of articulation may often be considered as diagnostic of lesions of the pons or medulla, provided the presence of aphasia of cerebral origin can be excluded by the history of the case. There is unquestionably a tract of fibres (the motor speech tract) that serves to connect the centres in the medulla with the cortical centres for the movements of the face and tongue.

Conjugate deviation of the eyes may accompany a lesion of the pons. This symptom is not pathognomonic, however, because it may occur also with cortical lesions of the cerebrum and lesions of the internal capsule.

The *motor*, *sensory*, *and vaso-motor effects* of lesions within the pons are manifested in the extremities, chiefly, but not exclusively, upon the side opposed to the lesion. This is not the case with those cranial nerves whose fibres of origin probably traverse the pons (the fifth, sixth, seventh, eighth [?], eleventh [?], and twelfth). The effects of intrapontine disease upon some of these nerves, at least, are modified by the seat of the lesion, as has been shown in preceding paragraphs.

Contraction of the pupils during an apoplectic attack is to be regarded as strongly diagnostic of a clot within the pons.

Hemorrhage into the pons is usually followed by coma and sudden death, if the clot be large or if the blood escape into the fourth ventricle. The diagnostic points mentioned above apply, therefore, more particularly to foci of softening and destructive lesions of small size and slow development. When blood escapes into the fourth ventricle, convulsions are observed, and death is liable to follow rapidly.

Disturbances of the circulatory and respiratory functions may occur in connection with lesions of the pons; but they are to be regarded rather as evidences that the medulla oblongata is directly implicated or subjected to pressure.

LESIONS OF THE CEREBELLUM.

The functional attributes of this ganglion are as yet imperfectly determined, and the effects of lesions (tumors, hemorrhage, softening, atrophy, and sclerosis) which involve its different regions vary with their seat. The following deductions are chiefly those of Nothnagel, who has devoted special attention to diseases of this ganglion. Seguin has also lately contributed to this field a valuable article.

Lesions of one of the *cerebellar hemispheres* are often incapable of diagnosis, especially if only one hemisphere be involved.

Lesions of the *vermiform process* are generally attended with symptoms of a more decided character.

Incoördination of movement, an *intense vertigo* (identical with that of Ménière's disease), and a "*titubating gait*," are the more common effects of cerebellar lesions; but these are not in themselves pathognomonic of cerebellar disease, because they may be produced by lesions of other parts of the brain. The consideration of all the morbid phenomena of each case (both of a positive and negative character) is required to render the diagnosis certain.

A staggering gait is especially liable to be developed in case the "worm" of the cerebellum is directly involved, or is pressed upon by lesions of adjacent parts. It only exists when the subject is in the upright posture, and the ataxic symptoms rarely affects the delicate movements of the fingers.

Gastric crises (chiefly exhibited by *persistent vomiting*) are a diagnostic feature of lesions of the cerebellum, in many cases. When destructive lesions of the cerebellum exist, vomiting is less frequently observed than when that ganglion is encroached upon by lesions of other parts.

Atrophy of the cerebellum has been observed to produce *imperfections of speech* (ANARTHRIA). The difficulty seems to be confined exclusively to the motor apparatus. The memory of words is not disturbed. It is probably to be attributed to interference with the "speech tract" (Fig. 24).

Pain in the occipital region is often present in cerebellar disease. It may exist also in the frontal region, or be entirely wanting.

The *organ of vision* may be affected. Occasionally, the eyes may exhibit incoördination of movement and nystagmus; and also the evidences of choked disc, amblyopia, and amaurosis.

Hemorrhage into the cerebellum is sometimes associated with a loss of facial expression, due to a slight paresis. The patient may also exhibit a tendency to assume one position, and to return to it when moved by the attendants. Should hemiplegia occur, in such a case, it indicates

that the lesion exerts pressure-effects upon the pyramidal tracts, either in the crus, pons, or medulla.

Irregularity of the heart's action, which is sometimes observed in connection with a cerebellar lesion, indicates a pressure upon the cardio-inhibitory centre of the medulla.

Abnormal mental symptoms are generally absent in connection with cerebellar lesions. When atrophy of the organ is present, or when other parts of the brain are diseased simultaneously with the cerebellum, mental derangements may be observed.

When the *middle crura of the cerebellum* (going to the pons) are affected by lesions which create irritation, *rotary movements* of the body and a *lateral deflection of the head and eyes* may be developed. As a rule, these rotary movements are toward the healthy side; but this is not invariably the case, as they sometimes are toward the side upon which the lesion is situated. It is a curious fact that most of the effects of cerebellar lesions are attributable to a greater or less extent to irritation of the crura.

Lesions of the *superior peduncle of the cerebellum* are liable to induce paralysis of the motor oculi nerve, as shown by the development of ptosis, external strabismus, and dilatation of the pupil. Hemianæsthesia and more or less ataxia may be induced by pressure upon the tegmentum and the fillet tract (lemniscus) respectively.

"*Bulbar symptoms*" may develop late in the course of a cerebellar lesion. Such phenomena are usually attributable to obliteration of the vertebral and basilar arteries and their branches, as a result of arteritis obliterans (Seguin).

LESIONS OF THE MEDULLA OBLONGATA.

The size of this ganglion almost precludes the existence of lesions, even if small, which do not influence to a greater or less extent the nerve nuclei contained within it.

An implication of the cranial nerve roots (Figs. 16 and 26) may cause disturbances of respiration, circulation, phonation, deglutition, and articulation.

The sensory and motor tracts to the extremities may be simultaneously involved; and thus anæsthesia (?) and paralysis of motion may occur upon the side of the body opposed to the lesion. The fillet tract (Fig. 11) may be also affected by the lesion, in which case evidences of ataxia will be developed in the extremities. Finally, the lower part of the face may be rendered paretic.

Of the above-mentioned symptoms, *aphonia* and the *impairment of the respiratory and circulatory symptoms* are particularly diagnostic of medullary lesions.

The symptoms of *Duchenne's disease* are present only when chronic progressive degeneration of the nuclei of the medulla exists.

Suddenly developed lesions of the medulla are liable to cause instantaneous death.

Diabetes and *albuminuria* may be excited by lesions of the medulla.

When the *pneumogastric nerves* are implicated, dyspnœa, irregularity of the action of the heart, and gastric or intestinal derangements are encountered.

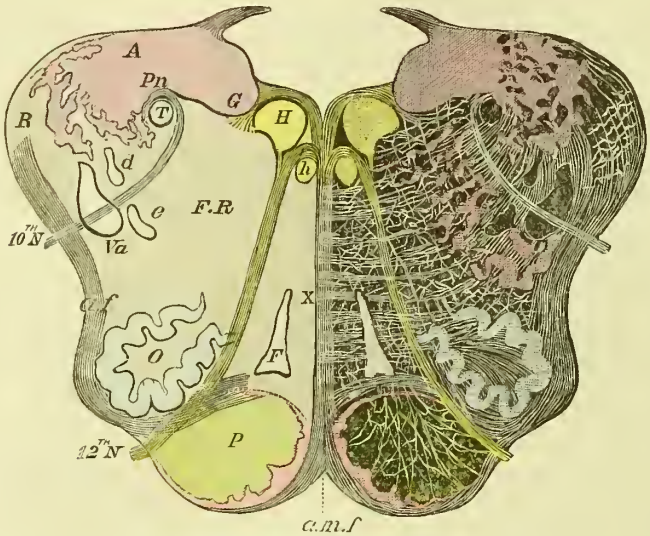


FIG 27.—A TRANSVERSE SECTION OF THE MEDULLA (PARTLY SCHEMATIC) MADE THROUGH THE MIDDLE OF THE OLIVARY BODY. (Modified from Spitzka.) *H*, and *h*, nuclei of origin of the hypoglossal nerve (twelfth cranial); *F. R.*, reticular formation, with its cell masses; *O*, olivary body; *P*, pyramid; *a. m. f.*, antero-median fissure; *G* and *Pn*, masses of cells probably associated respectively with the glosso-pharyngeal and pneumogastric nerves; *Va*, ascending root of fifth cranial nerve; *B*, restiform column; *a. f.*, arcuate fibres; *F*, fibres passing through the inter-olivary tract; *e* and *d*, bundles of fibres from the posterior spinal tracts, cut across on their way to the inferior cerebellar peduncle after decussation; *T*, the "trineural fasciculus" of Spitzka; "solitary" or "round" bundle of other authors. Note that the solid masses represented in the cut in red and yellow are composed of cells; the black areas are designed to represent conducting fibres running vertical to the plane of the section; the white lines represent fibres which run in the plane of the section; and, finally, that some of the conducting strands are left uncolored (as, for example, *T*, *Va*, *F*, *e* and *d*).

In a few instances, tumors and foci of softening in the medulla have been known to exist and create no symptoms of a diagnostic character.

Dysphasia, and the *loss of the power of protrusion of the tongue*, points to an implication of the hypoglossal and glosso-pharyngeal nuclei.

FOCAL LESIONS INVOLVING CRANIAL NERVES.

In the third edition of my work upon "Surgical Diagnosis,"* I have incorporated some axioms which bear upon the diagnosis of focal lesions of the brain that affect cranial nerves.

* William Wood & Co., New York, 1883.

Some of the axioms there given require modification, when viewed from the standpoint of our present knowledge. Many of the suggestions referred to had been selected by me from some of my *earlier writings* on this field, and, if taken literally, would now be in conflict with later observations of an anatomical and clinical character published since that date by others. Some other points given by me in that work are more or less imperfect, although perhaps technically accurate. I have, therefore, seen fit to alter the wording of certain parts of this work, which I shall now repeat in substance :—

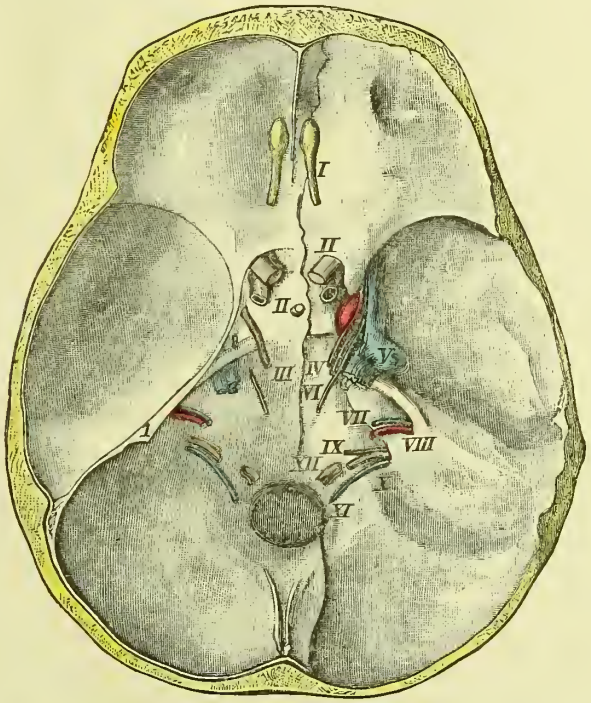


FIG. 28.—THE BASE OF THE SKULL WITH THE NERVES WHICH ESCAPE FROM ITS FORAMINA. The cranial nerves are numbered in their customary order.

LESIONS AFFECTING THE OLFACTORY NERVE.

Anosmia (loss of smell) may occur from any lesion which involves the first cranial nerve. It is usually unilateral.

Whenever it occurs in connection with hemiplegia, the body paralysis is on the side opposite to, and the anosmia on the same side as the lesion. This condition is known as crossed paralysis of the "first cranial nerve and body" type. Anosmia indicates the existence of a lesion *situated in the anterior fossa of the cranium*, or a destructive lesion of the cortex of the temporal lobe near to its apex.

Crossed paralysis of the "olfactory nerve and body type" may occur whenever a localized pressure is exerted chiefly upon parts *within the anterior fossa of the skull*. The fibres of the so-called "motor tract" (Fig. 12) are involved by an upward pressure upon the caudate or lenticular nucleus; or the fibres of the internal capsule are directly affected by the lesion. This accounts for the hemiplegia of the opposite half of the body. The olfactory nerve (which lies near the optic chiasm) is affected by pressure in the downward direction, and the optic chiasm or tract may be simultaneously involved; hence, a loss of smell in the nostril on the same side as the lesion may co-exist with some form of hemianopsia, as well as with a crossed hemiplegia.

LESIONS AFFECTING THE OPTIC NERVE.

Hemianopsia may occur when the *optic chiasm*, the *optic tracts*, the *thalamus*, the *posterior part of the internal capsule*, or the *cortex of the occipital lobes* (chiefly the cuneus) are pressed upon or destroyed. It is evident, therefore, that the trephine cannot afford relief of this symptom in most cases, because the lesion is commonly situated at the base of the cerebrum. When syphilitic gummata may be suspected, the prognosis is extremely favorable if active treatment be employed.

The variety of hemianopsia may indicate the seat of the lesion with great exactness. Fig. 21 will make this apparent.

If *paralysis* (in any of its forms) co-exists with hemianopsia, a valuable guide may often be afforded in determining the extent of the lesion.

The *binasal*, and also the *bitemporal varieties* of hemianopsia are due (as a rule, at least) to lesions confined to the *anterior fossa* of the cranium; hence we sometimes find the *olfactory nerve* (of the side corresponding to the seat of the lesion) simultaneously affected, and creating anosmia (loss of smell) with or without subjective odors.

If the lesion be situated within the *middle fossa* of the cranium, the *optic tracts* may be affected, thus causing homonymous hemianopsia (Fig. 21). The *motor nerves of the eye* may be simultaneously pressed upon, as they pass through that fossa on the way to their foramen of exit from the cranium (the sphenoidal fissure), and thus more or less impairment of the movements of the eyeball of the same side may be created.

The value of these complications cannot be over-estimated, when they exist, because they may be of the greatest aid in diagnosis. They may often enable the skilled anatomist to positively determine the seat of the lesion.

Hemiplegia may occur in *connection with hemianopsia*, provided that the lesion is of sufficient size to affect any part of the so-called "*motor tract*" of fibres simultaneously with the optic nerve fibres (Fig. 23).

Motor paralysis is, under such circumstances, developed chiefly if not exclusively on the side opposite to the lesion, because the fibres of the motor tract decussate, to a greater or less extent, at the lower part of the medulla. Flechsig has shown that, in rare cases, *exceptions* to this rule are to be explained by an abnormality in the decussation of the motor fibres.

Hemiplegia is seldom observed in connection with hemianopsia alone.

The olfactory, motor oculi, trigeminus, or facial nerve roots are especially liable to be simultaneously involved. This explains the mechanism of the four varieties of "crossed paralysis" which are clinically encountered. The hemiplegia being developed on the side opposite to the lesion as a rule, while the symptoms produced by paralysis of the cranial nerve are confined to the side corresponding to the lesion.

Homonymous hemianopsia, when it occurs without any impairment of mobility or sensibility, points strongly toward a lesion of the cuneus.

Ataxic manifestations, occurring in connection with evidences of impairment of the sense of sight, open a wide field for speculation. The proximity and intimate structural relations of the cerebellum with the optic lobes, basal ganglia, crus, and medulla, suggest the possibility of cerebellar lesions when these two symptoms are present to a marked degree, and the patient can stand with the eyes closed.

Hemianæsthesia may occur in connection with hemianopsia and other disturbances of vision. It indicates some disturbance of the nerve fibres of the so-called "sensory tract;" the loss of sensation being confined to the lateral half of the body opposite to the lesion which causes it, because the sensory fibres decussate in the spinal cord.

In *cerebral hemianæsthesia*, there is more or less insensibility to touch, pain, and temperature, and also an abolition of muscular sensibility with complete retention of electro-motor contractility. The mucous membranes of the eye, nose, and mouth are also anæsthetic. If it be due to hysteria, the *special senses* are either abolished or rendered deficient, and *hyperæsthesia over the ovaries* exist (Ferrier). These facts will often enable the diagnosis to be made between hysterical and cerebral hemianæsthesia of organic origin.

Choked disc is a common symptom of lesions situated at the base of the cerebrum, and also of any intra-cranial disease which produces a gradually increasing pressure. *It is especially diagnostic of cerebral tumors.*

It is not associated with impairment of vision until late, so that it is often unsuspected when present. The ophthalmoscope is necessary for its detection. It may co-exist with hemianopsia, and is always bilateral. It is a positive contra-indication to trephining.

Lesions at the base of the skull may *cross the mesial line*, and still involve only one optic tract. If this occurs, the hemianopsia will be accompanied by other symptoms of diagnostic importance, no longer confined to one side. Double anosmia, general paresis or complete paralysis, general anæsthesia, and paralytic symptoms referable to both eyeballs might be thus produced. Lesions of this character are more liable to affect the chiasm of the optic nerves than the optic tracts; in either case, however, hemianopsia would result, and its type would be a reliable guide to the seat of pressure.

Motor aphasia sometimes co-exists with hemianopsia. I have met with two instances of this kind. In one there was slight paresis of the left side, tending to prove that aphasia can occur with lesions involving the right hemisphere. Both were cured with specific treatment. We must attribute the development of this complication to pressure upon parts in the neighborhood of Broca's centre.

LESIONS AFFECTING THE MOTOR OCULI NERVE.

The *nucleus of origin* (Fig. 12) of the third cranial nerve of each side seems to be capable of subdivision into groups of cells which preside over movements of special muscles of the orbit.

Thus we may clinically recognize the existence of a special nucleus for visual "accommodation," for pupillary movements, and for the internal rectus, the superior rectus, the levator palpebræ, the inferior oblique, and the superior oblique muscles.

This fact probably explains how the existence of "*external ophthalmoplegia*" and other distinct forms of orbital paralysis may occur from organic lesions in the region of the tegmentum.

Paralysis of this nerve is indicated by the following symptoms: (1) a falling of the upper eyelid (*ptosis*); (2) external strabismus; (3) dilatation of the pupil; (4) a slight bulging of the eye forward, on account of muscular relaxation; and (5) a loss of accommodation of vision.

When the third cranial nerve is paralyzed from cerebral lesions the *lower part of the face* is often paretic on the same side as the lesion. This is not the case when a lesion involves the nerve after it escapes from the crus cerebri, viz., within the middle fossa of the cranium or the orbital cavity.

Crossed paralysis of the "motor oculi nerve and body" type, indicates a *lesion situated within the crus cerebri.* We find that the eye on the same side as the lesion can no longer be turned toward the nose, or made to act in parallelism with the opposite eye; that the pupil is dilated; and that the upper eyelid droops over the eyeball, giving it a sleepy appearance. On the side opposite to the lesion the body is hemiplegic. There are few conditions which are of greater clinical importance

than this type of crossed paralysis, because the seat of the lesion is positively indicated.

If the optic tract, which lies in close relation with the crus be simultaneously affected by the lesion, the evidences of "homonymous hemianopsia," will be superadded, viz., the eye on the same side as the lesion will be blind in its temporal half, and that of the opposite side in its nasal half.

One half of the pupil may fail to react to light when hemianopsia exists. This is known as the "*hemiopic pupillary reaction.*"

LESIONS AFFECTING THE FOURTH AND SIXTH CRANIAL NERVES.

The nerves which are associated with the movements of the eyeball —the third, fourth, and sixth cranial—pass through the middle fossa of the cranium in company with the fifth cranial nerve. For this reason, lesions situated at the *base of the brain* are liable to involve any of these nerves separately, or all simultaneously, according as its pressure-effects are felt in one direction or another.

In addition to cranial causes, lesions of the *orbit* may also create impairment of the third, fourth, ophthalmic branch of the fifth, or sixth cranial nerves—all of which pass through the sphenoidal fissure into the orbit.

Impairment of the sixth cranial nerve is indicated by the development of *internal strabismus;* the extent of which varies with the degree of the paralysis.

If this nerve be affected by lesions within the cranium, *other nerves are liable to be simultaneously involved;* and an impairment of the cerebral motor tract may also be evidenced by a co-existing *hemiplegia or paresis* of the side of the body opposite to the seat of the lesion.

LESIONS AFFECTING THE FIFTH CRANIAL NERVE.

The following propositions will cover the diagnostic points which relate to lesions of the trigeminal nerve (after it escapes from the pons).

Peripheral lesions cause *anæsthesia of special parts* supplied by small branches or single filaments of the nerve.

The *co-existence of paralysis of other cranial nerves* with anæsthesia of the face, indicates a lesion in the vicinity of the base of the cerebrum.

If *a part of the face* and the *corresponding facial cavity* (orbital, nasal, or buccal) are simultaneously affected with a loss of sensation, the lesion is within the cranium, and so situated as to involve one of the three main divisions of the nerve.

If the anæsthesia extends over the *entire area supplied* by *all of the branches* of the nerve, and evidences of disturbance in the *nutrition*

of the parts are also present, the lesion affects the ganglion of Gasser or its immediate neighborhood.

If the *muscles of mastication* are paralyzed, and no anæsthesia exists, the lesion is outside of the cranium and involves only the motor root of the inferior maxillary branch of the nerve.

The anterior two-thirds of the tongue, the mucous lining of the floor of the mouth, and the integument of the chin will be rendered anæsthetic simultaneously if the *sensory trunk* of the inferior maxillary nerve is involved; and taste may be affected also on the same side as the sensory paralysis.

Neuralgia of the various branches of the fifth nerve may exist in place of anæsthesia, whenever the lesion simply *irritates* the nerve trunks, but does not impair their power of conduction of sensory impulses.

All late authorities agree in the statement that the deep trigeminal fibres may be traced as two roots: the so-called descending root (which comes from the cerebrum), and the ascending root, which is apparent in cross-sections at different levels of the medulla. The view of Meynert, that the fibres of the descending root cross within the substance of the pons, is sustained by clinical facts, as shown by Starr. This author draws the following deductions, respecting the clinical significance of facial anæsthesia:—

1. Lesions affecting the ascending root of the trigeminus produce anæsthesia of the face upon the *same side as the lesion.*

2. Lesions affecting the descending root of the trigeminus produce anæsthesia of the face upon the *side opposed to the lesion.*

3. Disturbances of sensibility in the face indicate a lesion situated within the medulla or pons, and in the *external lateral part of the formatio reticularis* (provided it be not due to neuritis of the trigeminus or a cerebral lesion).

4. If the *face be rendered anæsthetic upon one side, and the body upon the opposite side* (the condition known as "crossed sensory paralysis"), the lesion affects the *entire extent of the formatio reticularis*, and lies, in the medulla or pons, below the point of union of the ascending and descending roots of the trigeminus.

5. *If the face and limbs be rendered anæsthetic upon the same side.* the lesion lies in the brain at a point higher than the junction of the two roots of the trigeminus. It may, therefore, be found within the *formatio reticularis* of the *upper part of the pons* and *crus*, or, if cephalad of the crus, it may affect the *posterior third of the internal capsule* of the corresponding cerebral hemisphere, the *centrum ovale* of that hemisphere, or the *sensory area of the cerebral cortex*, in which all the sensory tracts terminate.

LESIONS OF THE SEVENTH CRANIAL NERVE.

The following propositions will cover the diagnostic points of lesions which induce facial paralysis (*Bell's palsy*):—

If the paralysis be limited to *distinct parts of one lateral half of the face*, the lesion affects only individual branches of the nerve, and is outside of the cranium. An apparent exception to this rule is sometimes met with in connection with lesions of the internal capsule and of the crus cerebri—paralysis of the lower half of one side of the face being clinically observed to occasionally accompany hemiplegia, and also paralysis of the motor oculi nerve on the same side as the lesion.

If the *fauces* and *palate* exhibit paralytic changes the lesion is within the cranium or in the temporal bone.

If the *sense of taste* be lost in the anterior two-thirds of the lateral half of the tongue (on the same side as the general facial paralysis), the lesion is either within the cranium, or in the temporal bone above the origin of the chorda tympani branch.

If the *sense of hearing* is rendered *very acute upon the same side as the facial paralysis*, the lesion is probably within the temporal bone and involves the ganglionic enlargement found upon the nerve in the aqueduct of Fallopius.

Facial paralysis (when dependent upon *cerebral lesions*, or those of the *crus cerebri* or the *pons*) is commonly associated with *hemiplegia*, which may be upon the same side as the lesion or on the opposite side.

Crossed paralysis of the "facial nerve and body type" indicates a *lesion of the pons Varolii* posterior to the line which connects the trigeminus nerve with its fellow at their escape from the pons. (Gubler.) The reader is referred to Fig. 27.

If the lesion be situated in front of Gubler's line, the facial paralysis and the hemiplegia will be on the *same side*.

LESIONS AFFECTING THE CRANIAL NERVES ARISING FROM THE MEDULLA OBLONGATA.

The facial, auditory, glosso-pharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves have their apparent origin from the medulla, and are more or less imperfectly understood in regard to their connection with different parts of the encephalon.

Labio-glosso-pharyngeal paralysis ("Duchenne's disease" or "bulbar paralysis") is associated with successive destruction of the nerve nuclei in the floor of the fourth ventricle and a secondary degeneration of the nerve trunks connected with them.

The nerve which exhibits the first evidences of paralysis will often afford clinical data from which some deductions respecting the original

seat of the lesion may be drawn. The more common lesions of the medulla include arteritis, thrombosis, traumatism, softening, hemorrhage, sclerosis, and tumors.

The development of "bulbar paralysis" is associated, as a rule, with neuralgic pains, muscular spasms, anæsthesia, and disorders of special senses.

Compression of the medulla oblongata has been shown to cause the respiratory phenomena termed "Cheyne-Stokes respiration," and also albuminous and diabetic urine. In the former, the frequency and character of respiration constantly changes in some regular order—gradually increasing to a certain maximum, and then gradually decreasing in frequency till they cease, when they begin again to increase in frequency and in depth.

The *vaso-motor centres*, which are situated within the medulla, help to explain many other visceral phenomena which are observed when it is diseased. These are too numerous and complex in their nature to be discussed here.

The differential diagnosis of suddenly-developed lesions of the medulla, which are not immediately fatal, must rest upon the co-existence of certain functional disturbances. Among these may be chiefly mentioned: 1, epileptiform attacks, occurring at the onset or later; 2, hemiplegia or paraplegia; 3, loss of consciousness; 4, hyperæsthesia or circumscribed anæsthesia; 5, dysphagia, vomiting or hiccough, and Cheyne Stokes respiration, from interference with the pneumogastric nerve; 6, embarrassment of speech, from interference with the hypoglossal nerve; 7, deflection of the velum palati and uvula, from interference with the facial nerve; 8, hydruria, from interference with the centre of renal circulation; 9, diabetes, probably from interference with the centre of the vaso motor nerves of the liver; 10, normal electro-muscular contractility in the paralyzed parts.

If the lesion be *very extensive* and of *sudden advent*, death may occur without the bulbar symptoms being well defined.

A SUMMARY OF THE MORE IMPORTANT PHYSIOLOGICAL AND PATHOLOGICAL FACTS PERTAINING TO THE SPINAL CORD.*

Much of an anatomical nature relating to the spinal cord might be introduced at this point with profit to the reader if space would permit. It is hoped, however, that by the aid of the diagrams incorporated, the various "tracts" or bundles of fibres which compose the cord will be comprehended. Additional information of an anatomical kind may be

* Some parts of this section have already appeared in the chapter upon the Diseases of the Spinal Cord in the last edition of the Author's work, "A Practical Treatise on Surgical Diagnosis." William Wood & Co., N. Y., 1884.

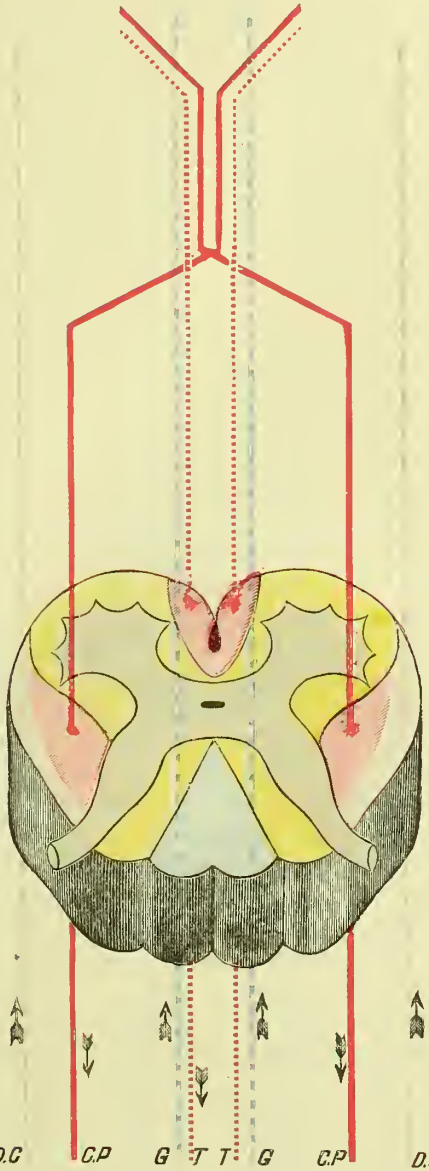


FIG. 29.—A DIAGRAMMATIC REPRESENTATION OF THE CONDUCTING TRACTS OF THE SPINAL CORD. (Modified from Bramwell.) *T*, fibres of Türck's column (direct pyramidal bundle); *C. P.*, "crossed pyramidal fibres;" *G*, fibres of the column of Goll (postero-median column); *D. C.*, fibres of the "direct cerebellar column." Note that the arrows show the direction of the impulses carried by each tract of fibres. Also that the motor fibres of the lateral column decussate at the *lower part of the medulla*. Each of the "anterior pyramids" of the medulla is composed of the motor fibres (direct and decussating) above the lower limits of the medulla.

gained by the reader (in case the diagrams prove insufficient) by referring to the introductory pages of a subsequent section which treats of diseases of the spinal cord.

1. The *anterior* and *lateral pyramidal columns* of each side contain only *motor fibres*. Those in the former (Türck's columns) are connected with the *corresponding cerebral hemisphere*, while those of the latter (the "crossed pyramidal tracts") are connected with the *opposite cerebral hemisphere*. (Fig. 29.)

2. The *posterior column* of each side (comprising two portions—that of Goll and Burdach) serves to convey *sensory nerve fibres* apparently connected with the tactile sense, and also *commissural fibres* (?) which connect different segments of the cord; hence they are physiologically associated both with *tactile sensation* and the *coördination* of *muscular movement*. (Fig. 29.)

3. The *lateral column* of each side (exclusive of the crossed pyramidal fibres and those of the direct cerebellar column) probably conveys *vaso-motor fibres* and possibly those of *sensation* also. It has been proven also to convey fibres directly to the cerebellum (the "*direct cerebellar column*"). The *crossed pyramidal tract* occupies a distinct area of this column.

4. The *multipolar nerve cells* in the *anterior horn* of the spinal gray matter possess a "*trophic function*." When they are destroyed, the nerve fibres arising from them, and the muscles also which are supplied by those fibres, undergo atrophy. (Fig. 33.)

5. The fibres of the anterior and lateral pyramidal columns have their "trophic centre" in the *motor area of the cerebral cortex*. Any lesion which tends to sever these fibres from this centre creates a *descending degeneration* of all the nerve fibres so disconnected, as far as their ultimate distribution, viz., to segments of cord below the lesion.

6. The *spinal nerves* may be regarded as guides to the *various segments* of the spinal cord; each segment consisting of a disc of the cord of sufficient thickness to include a separate pair of spinal nerves which are attached to it. (Fig. 31.)

7. Each *spinal segment*, with its attached nerves, may be figuratively regarded as a distinct spinal cord for that limited portion of the body to which its nerves are distributed, viz., the *muscles* to which the anterior roots of the spinal nerves proceed, and the *parts supplied with sensation* by means of the posterior roots of the same.

8. The *superimposed segments* of the cord are bound together by *tracts of nerve fibres*. Some of these are *continued into the brain*, while others are *purely commissural in type*. We can attribute to the former group (the "motor" and "sensory tracts") the conduction of motor impulses from the brain to the various spinal segments, and of impressions

of a sensory character from the periphery of the body to the brain itself The other group mentioned (the "associating fibres") serves to assist the different spinal segments in the performance of all acts where a *harmonious* and *simultaneous action* of *several segments* of the spinal cord is demanded.

9. The two *lateral halves* of each segment of the spinal cord are not totally distinct from each other, because a connecting band of the gray substance of the cord (the gray commissure), and also one of white substance (the white commissure), bind them together. The white commissure lies at the bottom of the anterior median fissure; the gray commissure fills the remaining space between the anterior and posterior median fissures (Fig. 30).

10. The *anterior horns* of the spinal gray matter contain *cells of large size* which are connected (1) with motor nerve fibres, joining each spinal segment with the brain (somewhat indirectly), and (2) with the fibres of the anterior root of the spinal nerve (associated with the muscles controlled by each segment). Thus these nerve cells are interposed be-

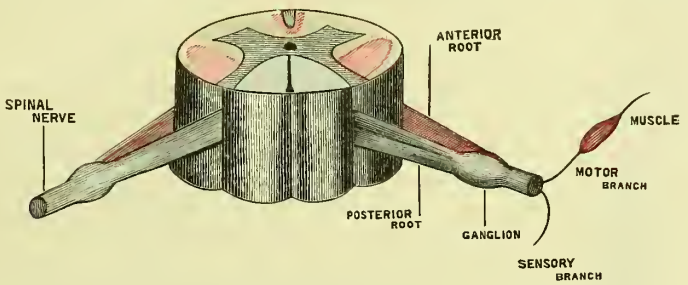


FIG. 30.—A SPINAL SEGMENT. The two roots of the spinal nerve are shown; also the sensory and motor fibres of which each is composed.

tween the fibres which pass from the brain to the cord and those distributed to the muscles, an arrangement which permits of an automatic action of the cord, irrespective of cerebral influences.

The *cells of the anterior horns* appear to control also the *nutrition of the muscles*, connected with them by means of the nerve fibres.

11. The *cells of the posterior horns and central portion* of the spinal gray matter are probably connected more or less intimately with the fibres of the *posterior or sensory roots of each spinal segment*, and possibly also with the *paths of conduction of painful impressions* to the brain. They do not exert any apparent influence upon the nutrition of the parts associated with them by means of the spinal nerve fibres.

12. The *spinal reflexes* are probably performed by means of an anastomosis of the processes of the cells of the anterior and posterior horns of each lateral half of a spinal segment. This enables a *sensory*

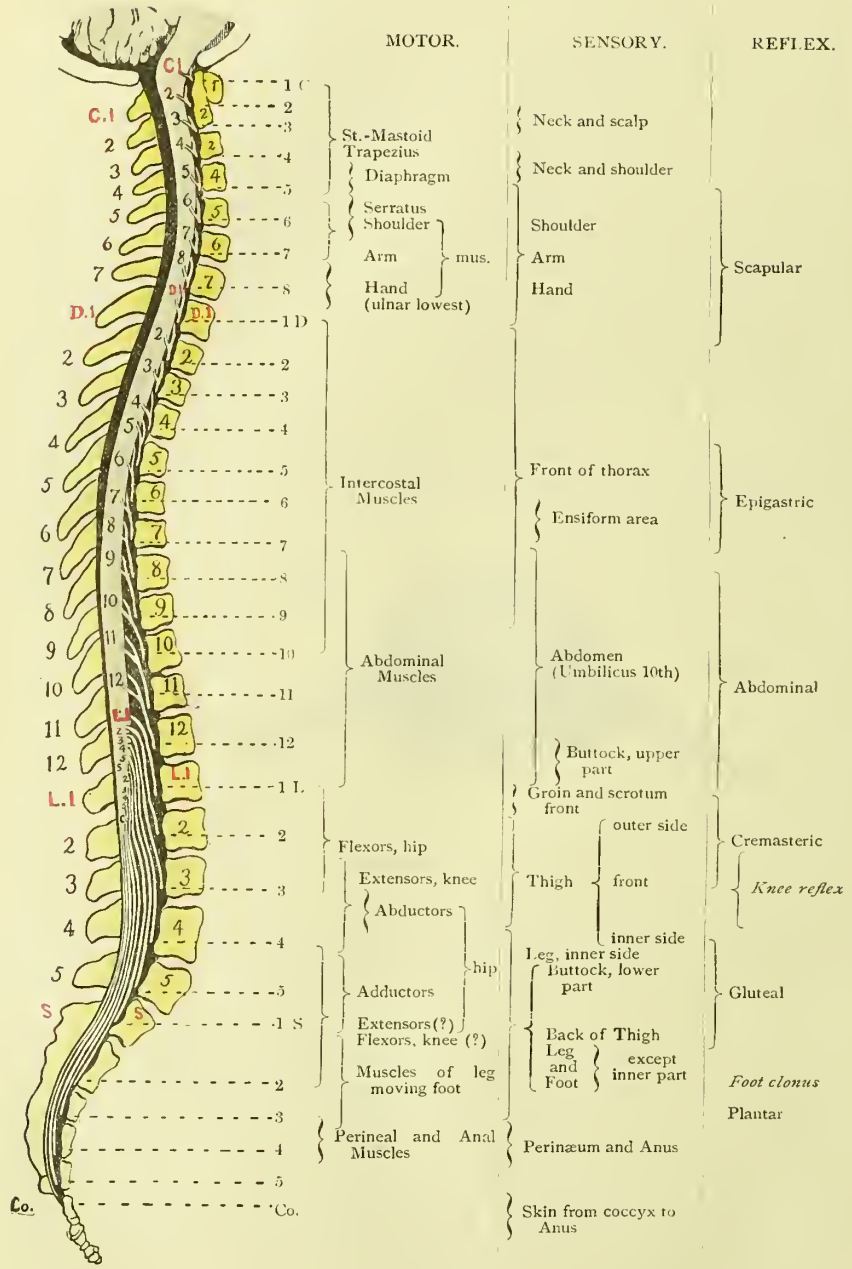


FIG. 31.—A DIAGRAM DESIGNED TO SHOW THE RELATIONS OF THE VERTEBRÆ TO THE SPINAL SEGMENTS, AND OF THE SPINAL NERVES TO THE MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE SPINAL CORD. (GOWERS.)

impression, which is conveyed to the spinal segment by means of the fibres of the posterior root of the spinal nerve, to become transformed into *motor impulses* in the cells of the anterior horn. These are then transmitted to the muscles by the fibres of the anterior root of the spinal nerve.

13. *The sense of touch* may be destroyed, in parts below the point of injury, by section or disease of the *posterior columns*. The sense of pain and the appreciation of temperature are apparently still conducted, provided the gray matter escapes injury.

14. *The sense of pain* is destroyed when the *gray matter* is rendered incapable of transmitting such sensations. The sense of touch apparently remains unaffected, if the posterior columns escape.

15. *The sensation of pain and of touch may be independently retarded* by lesions of the cord that impair, but do not totally destroy the conductivity of the paths for such sensations. The amount of such retardation depends upon the extent of the destructive process within the cord. Cases have been reported where thirty or more seconds would elapse between each painful contact on the periphery of the body and its appreciable sensation.

16. *Destruction of a posterior root* of a spinal nerve, or the network of fibres formed by it within the substance of the cord, must affect the transmission of all *sensations of touch, pain, and temperature* from the peripheral area of distribution of the nerve so affected (Fig. 31).

17. *Destruction of a posterior nerve root* causes anæsthesia to pain, temperature, and touch. *Trophic disturbances* of the skin are also liable to follow, particularly if the nerve-root is injured outside of the ganglionic enlargement developed upon it (Fig. 30).

18. *Destruction of the columns of Burdach and Goll* is followed by tactile anæsthesia of definite areas, that correspond to the spinal segments affected, and sometimes in parts below the injury. Anæsthesia of the arms is especially characteristic of a lesion in Burdach's column; when in the legs, of Goll's column.

19. *When the posterior columns* of the cord are affected with diseased conditions that create *irritation* of the parts, the so-called "*girdle pain*," or "*cincture-feeling*" is developed in those nerves that traverse the disease area of the cord. Below the level of the spinal lesion, subjective sensations of touch (such as formication, numbness, abnormal sensations of heat or cold, etc.), and more or less hyperæsthesia are usually created.

20. *Lesions of the sensory tracts* (the so-called "æsthesodic system") cannot induce paresis, paralysis, spasm, or muscular atrophy. They can only create sensory manifestations (such as anæsthesia, hyperæsthesia, numbness, formication, abnormal sensations of heat and cold,

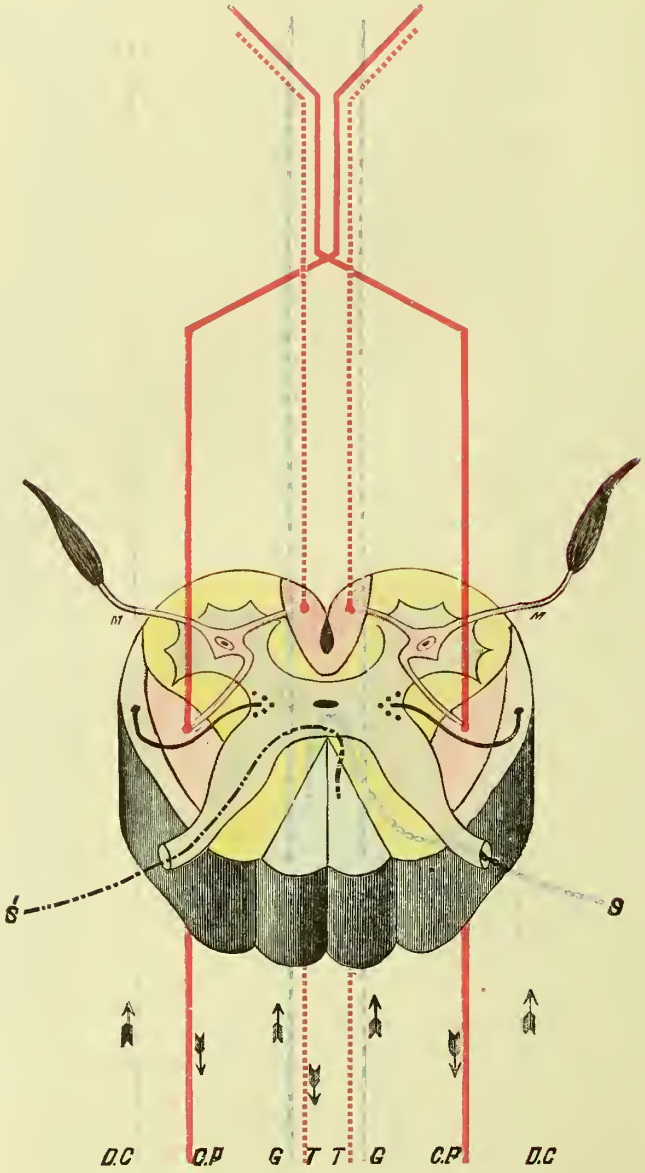


Fig. 32.—A Diagram Designed to Illustrate the Connections of the Motor and Sensory Conducting Tracts of the Cord with the Spinal Nerves. (Modified from Bramwell.) *M*, motor fibres of the anterior root of a spinal nerve; *S*, *S'*, sensory fibres of the posterior root. Note that the course of *S* and *S'* are not the same. Some sensory fibres pass directly through the posterior horn of the spinal gray substance, and others through Burdach's column to read the gray substance. The direct cerebellar column is composed of fibres which start in Clarke's column of cells (Fig. 33). The fibres of the two pyramidal tracts become united to the motor cells in the anterior horns of the spinal gray substance.

and pain), and, in addition, an inability to properly coördinate muscular movements (ataxic symptoms).

21. *Sensory phenomena* are manifested, as a rule, upon the side of the body opposed to the seat of the lesion. If they occur upon the same side, either the posterior nerve roots are directly involved, or the sensory tracts are affected during their ascent in the posterior columns before their decussation.

22. *The so-called "cincture feeling"* or "*girdle pain*" may be taken as a valuable guide in deciding as to the probable limits of a focal lesion of the cord.

23. *Pain in the region of the spine* is a rare symptom of disease of the spinal cord. When it exists, it commonly indicates a disease process that is confined to the vertebræ or the spinal meninges.

24. *Destructive lesions of the posterior columns* (if bilateral) are commonly associated with a *band of complete anæsthesia* that corresponds to the area of distribution of nerve roots affected by the lesion. Below this girdle of anæsthesia, sensations of touch are usually impaired or absent, and sensations of pain are localized with difficulty, but are still transmitted by the gray matter of the cord.

25. *Lesions that create irritation of the cervical gray substance* are accompanied by dilatation of the pupil. If destructive processes are subsequently developed, the pupil becomes contracted (Argyll Robertson's pupil).

26. *Lesions of the posterior columns* that irritate primarily and subsequently destroy the spinal substance tend, as a rule, to progress upward. As they advance, the girdle of pain travels upward and leaves behind it a girdle of anæsthesia that steadily increases in width. In parts situated below the level of the lesion, the sense of touch is usually lost, although subjective sensations of touch, such as formication, tingling, numbness, etc., may exist.

27. *The existence of a girdle pain*, without any impairment of the sense of touch in parts below it, or the presence of motor paralysis, points strongly to some lesion that involves the posterior nerve roots only.

28. *Lesions that affect only the motor tracts* (the so-called "kinesodic system") may induce paresis or paralysis, spasm, and atrophy of muscles. They never cause sensory symptoms (such as marked and permanent pain, hyperæsthesia, anæsthesia, numbness, formication, etc).

29. *Paralysis of motion and muscular atrophy*, when due to spinal lesions, develop upon the same side of the body as the lesion. The same is generally true of the symptoms referable to *incoördination of movement*,—the so-called "ataxic" symptoms. The fibres that convey the "muscular sense" do not decussate until they reach the medulla.

30. *Atrophic changes in muscles* points strongly toward a degenerative change in the *motor nerve-cells* of the anterior horns of the spinal gray matter. These cells are the trophic centres for the fibres of the anterior nerve roots. (Fig. 33.)

31. *Contracture*, or a *permanent shortening of paralyzed muscles*, is strongly diagnostic of a lesion that involved the "crossed pyramidal tract" within the lateral column of the cord. (Fig. 29.)

32. *Symptoms referable to special organs* (when dependent upon a spinal lesion) indicate that some of the special centres of the cord are involved. Such symptoms may be of value in determining the extent and situation of the lesion.

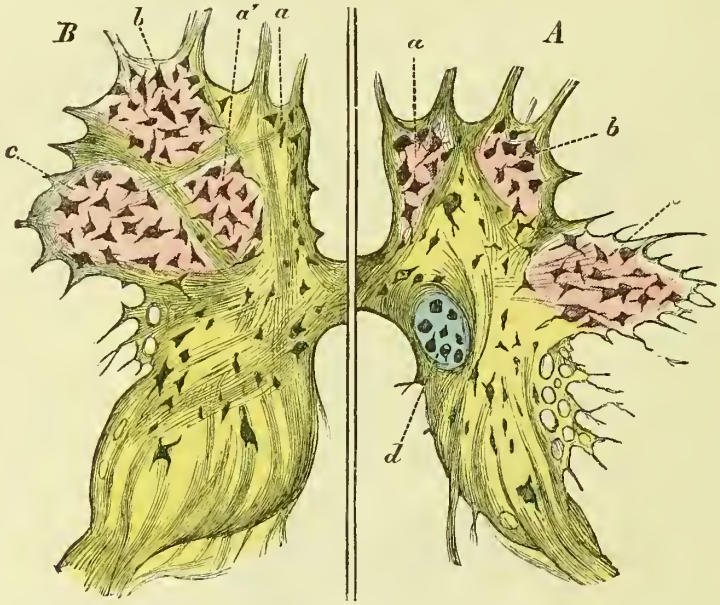


FIG. 33.—SEMI-DIAGRAMMATIC TRANSVERSE SECTION OF THE GRAY SUBSTANCE OF THE CERVICAL (*A*) AND LUMBAR ENLARGEMENT (*B*) OF THE SPINAL CORD. (Erb.) *A*. *a*, median group of cells; *b*, antero-lateral group; *c*, postero-lateral group; *d*, vesicular column of Clarke. *B*. *a*, median group; *a'*, group that appears first in the lumbar region, possibly, belonging to *a*; *b*, antero-lateral group; *c*, postero-lateral group. Note that the cells are few and scattered in the posterior horns, and also that the shape of both horns differs markedly in *A* and *B*.

33. *Lesions of the so-called "motor tracts"* of the cord (the "kinesodic system"), if destructive in character and sufficiently large to sever the connection of the motor fibres from their connection with the cells of the cerebral cortex, produce complete paralysis of motion below the level of the lesion on the corresponding side of the body. The paralyzed muscles will probably undergo subsequent contracture, and the deep or tendon-reflexes will become exaggerated.

34. *Lesions of the anterior horn of the spinal gray matter* are liable to produce paralysis in the areas of distribution of the related nerves, without disturbance of sensibility, but with marked trophic disturbances.

35. *Slight pressure upon the cord* may induce moderate paralysis (paresis) of the extensor muscles and secondary contracture of the flexors.

36. *Lesions of one lateral half of the cord* produce complete motor paralysis, vaso-motor disturbances, incoördination of movement, and hyperæsthesia on the corresponding side below the level of the lesion, and a loss of sensibility on the opposite side with more or less paresis in some cases. A zone of anæsthesia, surmounted by one of hyperæsthesia, may exist at the level of the lesion on the corresponding side.

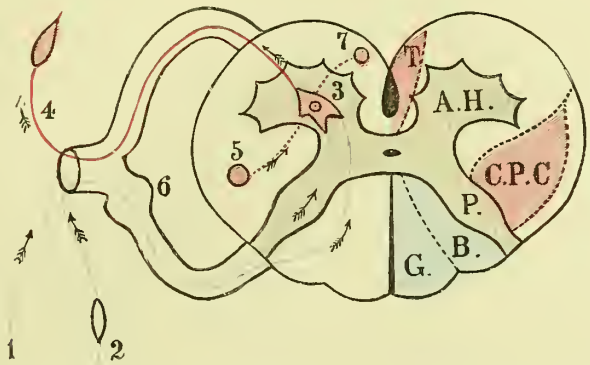


FIG. 34.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE VARIOUS CHANNELS THROUGH WHICH A MOTOR CELL OF THE CORD MAY BE CALLED INTO ACTION.—A. H., anterior horn; C. P. C., crossed pyramidal column; P., posterior horn; B., column of Burdach; G., column of Goll; 1, fibre for pain sensations; 2, fibre for touch, tendon, etc.; 3, motor cell; 4, motor fibres; 5, fibre from opposite cerebral hemisphere going to cell (3); 6, ganglion, or posterior nerve root; 7, fibre from cerebral hemisphere of same side, going to cell (3).

37. *Complete compression or division of the spinal cord* causes an exaggeration of the reflexes of the spinal segments below the seat of injury on both sides; in addition to serious disturbances of motility and sensibility.

38. *Localized destruction of the gray matter of the dorsal region* of the cord seems to arrest the control of the will over the reflex acts of micturition and defecation, which are governed by the lumbar region of the cord. These functions are still performed with nearly their normal regularity, however, by reflex action, if the centres that govern those acts are not included in the diseased area.

39. Scratching or stroking the skin over certain regions of the body causes a reflex contraction of special muscles when the cord is healthy. These are the so-called "*superficial spinal reflexes.*" They are of value

oftentimes in deciding as to the upper limits of a lesion. Among the more important of these superficial reflexes may be enumerated the plantar, cremasteric, abdominal, epigastric, and scapular. Both cerebral and spinal lesions create modifications of them, which possess clinical value.

40. The so-called "*deep spinal reflexes*" are called into action by first putting a muscle in a state of moderate tension, and then exciting it to contraction by some artificial stimulus, as a slight tap or blow for example. Among the more important of these may be mentioned: (1) The knee-jerk or patella-reflex; (2) the foot- or ankle-reflex; (3) the peroneal or lateral foot-reflex. These tests are employed, like the preceding ones, to determine the state of the spinal cord when the existence of a lesion is suspected; they may be increased, diminished, or abolished, according to the character of the lesion.

41. A *persistent foot-clonus* never occurs in health. It indicates that the lateral columns of the cord are probably involved by some spinal lesion. In supposed hysterical affections, this symptom will often decide the question of the existence of organic disease. It must not be mistaken for the involuntary foot-clonus which sometimes occurs when an unnatural posture is long maintained, even in health. It is usually associated with exaggeration of all the other deep reflexes.

42. All *reflex tests become abolished* when the muscles are separated from their connection with the spinal cord; hence, severing of a nerve, posterior sclerosis, compression of the spinal nerve roots, destruction of the gray matter of the cord, poisons, etc., are often associated with their complete abolition.

43. Disease of the *lateral columns* usually *decreases the skin reflexes*, especially those of the trunk. This is particularly true of the so-called descending degeneration of these columns, which follows cerebral lesions.

44. When marked *incoördination of movements* is present and the deep reflexes are not abolished, it indicates that sclerosis of the lateral columns probably co-exists with similar changes in Burdach's or Goll's columns.

45. *Spasm* is a marked symptom in many diseases of the spinal cord. It commonly indicates an excessive action of the reflex motor centres. It is particularly common as an acute symptom in spinal meningitis. In chronic organic diseases of the cord, it assumes the form of *contracture of muscles*, especially if the lateral columns are attacked. This condition becomes transformed into that of genuine spasm when the slightest forms of peripheral impressions are experienced, as in delicately manipulating the muscles for example.

A DIAGRAMMATIC SUMMARY OF SOME OF THE PRINCIPAL FEATURES OF CEREBRAL AND SPINAL ARCHITECTURE.

I have endeavored, as a sequel to my previous remarks, to represent in a schematic way the mutual relations of the encephalic and spinal centres, and to show the mechanism by which various phenomena observed during life may be explained. Let us examine different parts of the diagram separately.

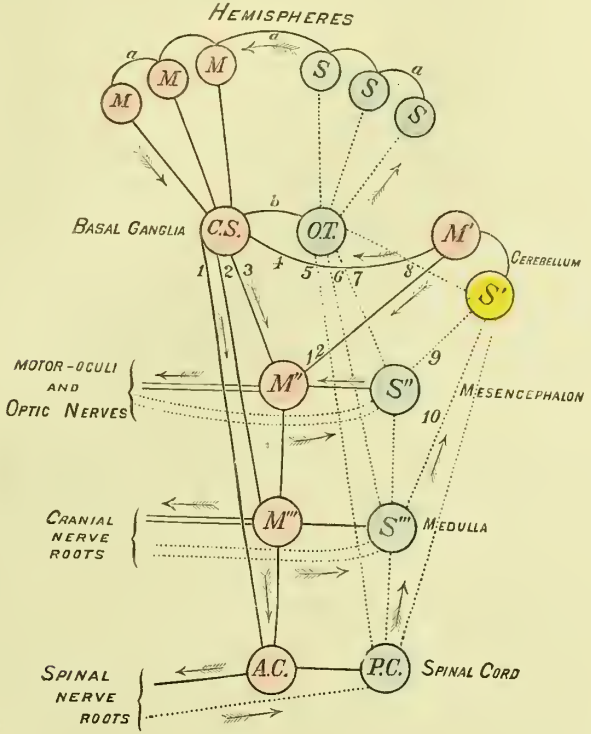


FIG. 35.—A SCHEMATIC REPRESENTATION OF THE CEREBRAL AND SPINAL CENTRES AND THEIR COMMUNICATIONS. *M*, *M'*, *M''*, *M'''*, motor centres; *S*, *S'*, *S''*, *S'''*, sensory centres; *C. S.*, corpus striatum; *O. T.*, optic thalamus; *A. C.*, anterior cornua; *P. C.*, posterior cornua. The arrows indicate the direction of the currents. The text will further explain the significance of the diagram.

THE CEREBRAL HEMISPHERES.

1. The circles (*M*) represent the *motor* and *psychic centres* of the convolutions; the circles (*S*) represent the *sensory centres* of the same. The lines in the diagram which connect these centres with the basal ganglia are the fibres of conduction to and from the cells of the gray matter of the convolutions (cerebral cortex). These fibres are the so-called "peduncular," "radiating," and "converging" fibres of different

authors. Taken as a whole, they constitute the so-called "corona radiata."

The fibres of both the *internal* and *external capsule* of the cerebrum *do not become associated with the cells of the basal ganglia.* They pass without interruption from the cells of the cerebral cortex to those of the gray matter of the spinal cord. Although not separately shown in the diagram, they can be imagined as passing over *C. S.* and *O. T.* in the cut as unbroken lines.

The lines marked (*a*) represent the so-called "associating fibres" of the cerebral hemispheres.

Throughout the diagram, all *efferent* fibres, or those which carry impulses *from* the various centres, are represented by unbroken lines, and all *afferent* fibres, or those which carry impulses *to* the various centres, by dotted lines. The arrows also show the direction of the currents. The fibres connected with the centres of the hemispheres can be traced in the drawing downward to their union with the cells of the basal ganglia, the mesencephalon, the medulla, and the spinal cord. In any of these regions separately, or in all simultaneously the cerebral cortex can probably exert its direct influence; the smaller centres are then either overpowered or controlled in their respective automatic actions by the great centre of intelligence—the cerebrum.

THE BASAL GANGLIA.

2. The *corpus striatum* (*C. S.*) is shown to be associated with the motor regions of the cortex (*M*). A direct communication probably also exists between it and the optic thalamus (*O. T.*), as shown by the line (*b*). The cerebellum is thought by some to communicate indirectly with it by means of the "processus e cerebello ad cerebrum" (4)—known also as the "superior cerebellar peduncle."

These three sets comprise its *afferent* fibres. Its *efferent* fibres (1, 2, and 3) pass to the cells of the crus, medulla, and spinal cord. The connection shown between the cerebellum and the so-called "motor tract," will help to interpret the view held by some that that ganglion assists the cerebrum in its control over the muscular apparatus of the body.

The *optic thalamus* (*O. T.*) has *afferent* fibres, which arise from the spinal cord, medulla, and mesencephalon (5, 6, and 7), and from the cerebellum (8). It is thus brought into relation with all sensory impulses conveyed by the spinal nerves, and also by those cranial nerves which are not motor in function. Impressions derived from sight, smell, hearing, and taste, as well as tactile impressions, and the sensation of pain, are probably more or less intimately associated with this ganglion. The efferent fibres of the optic thalamus are shown to lie in the posterior part of the corona radiata, and to distribute themselves among the sensory

centres of the cerebral cortex (*S*). The fibres of direct communication between the optic thalamus and the corpus striatum (*b*), help to explain the ability of an animal to perform automatic coördinated movements after the cerebral hemispheres have been removed and the basal ganglia left intact. These phenomena are in marked contrast to the forms of reaction which take place within the hemispheres between the sensory and motor centres of the cortex; since *consciousness* and *volition* are evoked by the latter, while the former is purely automatic. Conscious appreciation of sensations and voluntary motion are only possible when the cerebral hemispheres are present.

THE CEREBELLUM.

3. This diagram shows, in an imperfect way, the relations of the cerebellum to the paths of sensory and motor conduction. The sensory and motor centres of this ganglion (*S'* and *M'*) have not been anatomically differentiated from each other, but we have reason to believe that both varieties exist. The afferent fibres of the cerebellum (*9*, *10*, and *11*) probably bring it into direct relation with tactile impressions by means of the spinal cord, with sensations of pain (?) by the same channel, and with various other impressions by means of nerves of special sense. Its efferent fibres (*4* and *12*) are related in an imperfectly understood way, to the path of motor conduction. The most delicate feats of equilibrium are probably impossible without an intact cerebellum. This subject will be discussed hereafter. Each hemisphere of the cerebellum is now believed to be associated with the fibres of the opposite hemisphere of the cerebrum.

THE MESENCEPHALON.

4. As shown in the diagram, this term includes all the parts comprised between the cerebrum above and the medulla below. The collections of gray matter represented by the circles (*M''*) and (*S''*), comprise chiefly the so-called "substantia nigra" and the "red nucleus of the tegmentum." The fibres associated with them (*1*, *2*, *3*, *4*, *5*, *6*, *7*, *8*, *9*, *10*, *11*, and *12*), constitute, collectively, the *basis* and *tegmentum cruris* of Meynert, which are separated by the substantia nigra. The red nucleus lies beneath the corpora quadrigemina in the *tegmentum* (the sensory portion of the crus), and is in intimate relation with the fibres of the superior cerebellar pedunele (*4*). The corpora quadrigemina (not shown in the diagram) should be also included among the ganglionic masses of this region. The third cranial nerve is represented as structurally related to the mesencephalon. The optic nerve has also intimate relations with some of its parts. Fibres of many of the cranial nerves, which spring from the medulla, are prolonged through the pons and crus to reach the cerebrum.

The functions of the mesencephalic centres are too complex to justify any generalizations. All of the complex forms of muscular activity which are more especially elicited in response to some form of impression received from without by means of the nerves of special sense, such as locomotion, emotional expression, etc., are to be attributed partly, if not wholly, to these ganglionic centres. The special attributes of the red nucleus of the tegmentum and the substantia nigra are, as yet, somewhat conjectural.

THE MEDULLA OBLONGATA.

5. Within this ganglion, the nuclei of origin of many of the cranial nerves have been found, and special centres which preside over important physiological functions have also been demonstrated. The circles (*M'''*) and (*S'''*) in the diagram are supposed to represent the sensory and motor collections of gray matter, which give to this portion of the central nervous system its peculiar powers. The motor centres (*M''*) are represented as in communication with certain cranial nerve roots, and also with motor fibres which serve to connect the medulla to the corpus striatum and the ganglionic masses of the mesencephalon above, and the segments of the spinal cord below. The sensory centres (*S'''*) are shown to be in relation with the sensory cranial nerve roots (the term "sensory" being used in its broadest sense to include all fibres bearing afferent impulses), as well as with the paths of cerebral and cerebellar sensory conduction (*6* and *10*). Thus it is that the cerebellum as well as the cerebrum probably is made cognizant not only of tactile sensations and of other varieties of sensory impulses transmitted along the spinal tracts, but also of other facts which our special senses reveal to us. The view that the cerebellum acts in part as an "informing depot" (Spitzka) for the cerebral hemispheres can be comprehended by a study of this diagram.

The fibres which are drawn in the diagram between the motor and sensory centres of the medulla help us to comprehend the probable mechanism of many forms of complex coördinated reflex actions, of which the medulla is capable when all the nerve centres above it have been removed. It is apparent that each of the segments of the nervous system here depicted is capable (by means of associating fibres) of an action of its own which is independent of those centres above it, but which may be controlled or overpowered by the higher centres when they are called into action.

THE SPINAL CORD.

6. The diagram shows the cells of the anterior horns of the spinal gray matter (*A. C.*) to be in connection with the fibres of the direct motor tract which we have now traced from the cerebral cortex downward (although some have been deflected from the direct path by the cells of the

mesencephalon and medulla). These motor fibres of the spinal cord are prolonged by means of the interposed cell (*A. C.*) as fibres of the anterior or motor roots of the spinal nerves. The cells of the posterior horns of the spinal gray matter (*P. C.*) are likewise shown to receive the afferent impulses conveyed to them from without by the posterior or sensory roots of the spinal nerves (as shown by the arrow), and to transmit them upward by means of fibres which connect them with higher ganglionic masses (*5*, *6*, and *7*). The exact paths of motor and sensory conduction through the spinal cord are not positively settled. The antero-lateral columns of the cord are commonly regarded as the chief motor paths, although all observers are not in agreement respecting the anterior columns. The sensory tracts probably run partly in the central gray matter of the cord, and partly in the lateral and posterior columns. Sensory impulses travel on the side opposite to that on which the nerves enter, with the exception of impressions of the so-called muscular sense (Starr). The views held respecting the functions of the spinal columns have been given in preceding pages.

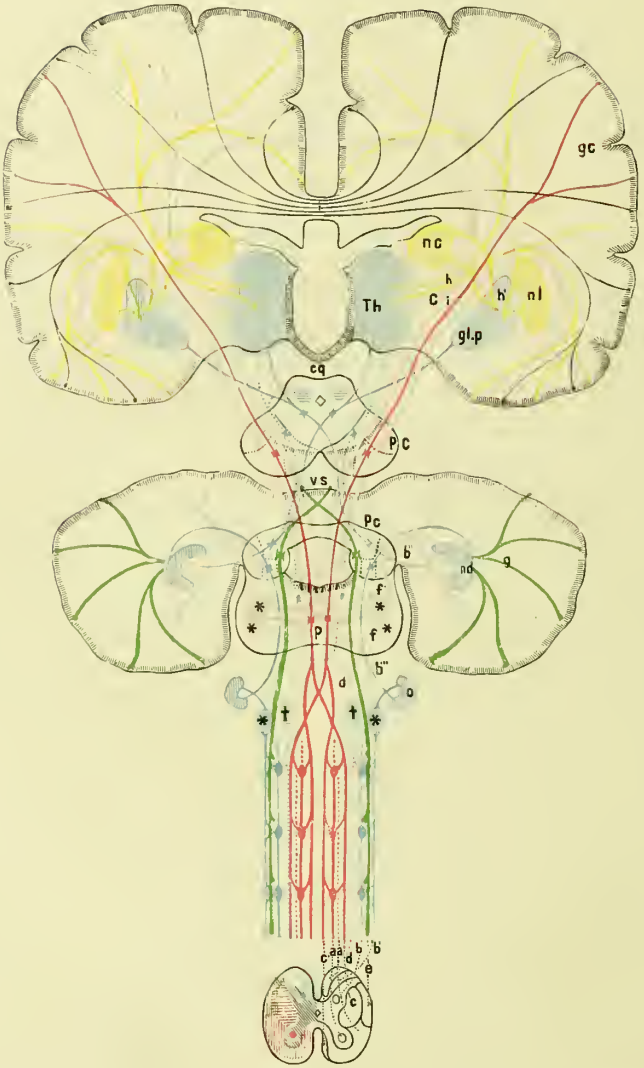
Finally, it will be observed that the motor and sensory cells of the spinal cord communicate. This arrangement allows of an *automatic spinal action*. Beheaded animals can be made to exhibit definite muscular movements when any irritation of the sensory nerves of the skin is employed to call them forth. A frog so mutilated will scratch with the opposite foot a spot on the leg which has been touched with an acid. Robin has observed similar phenomena in a beheaded criminal. These movements are purely reflex in type, because all parts which we know to be essential to consciousness or volition have been taken away. They can only be attributed, therefore, to a communication (not yet well understood) between the sensory and motor cells of the spinal segment. Many of the acts which constant and long-continued practice enable us acquire during life—as, for example, the running of scales upon a piano—are unquestionably performed automatically by the spinal cord, without assistance of the higher ganglia in many instances.

In closing this section, the Author feels that much has of necessity been omitted; and that some of the views advanced are apt to be modified or possibly overthrown by subsequent investigation.

He trusts, however, that the difficulties of the task will not be lost sight of by the reader; and that the chapter, as a whole, may prove of material assistance in fathoming the mysteries of obscure neuroses.

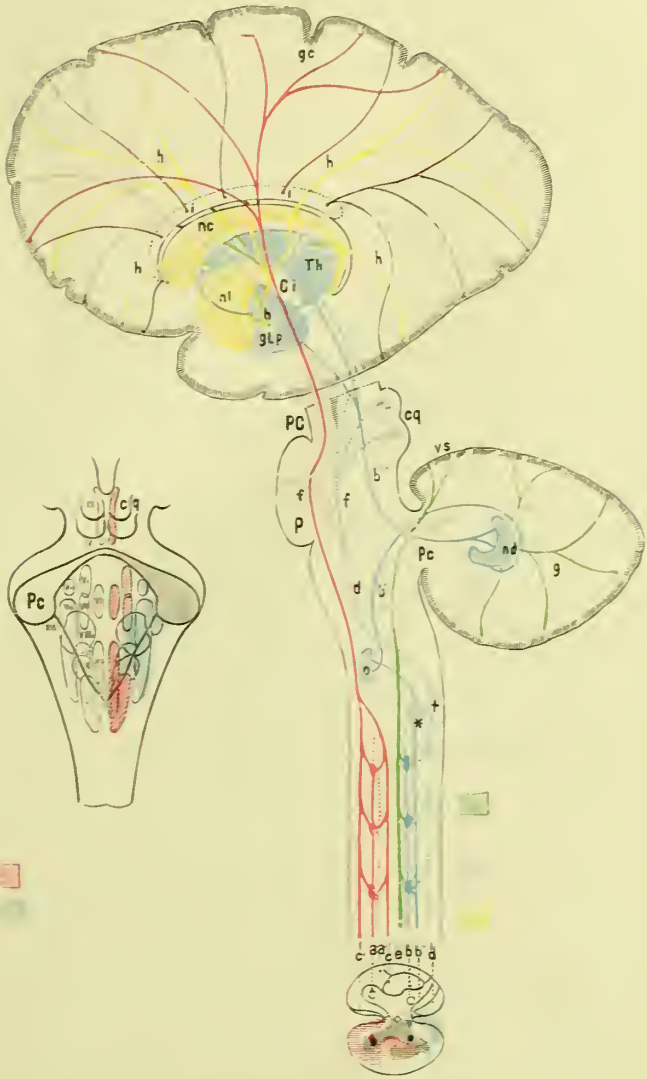
The two diagrams which follow are copied from Aeby. They present, to the Author's mind, the main points in cerebro-spinal architecture with singular lucidity.

FIG. 36.



FIGS. 36 and 37.—A DIAGRAM OF THE COURSE OF THE NERVE FIBRES IN THE SUBSTANCE OF THE BRAIN AND SPINAL CORD. (After Aeby.) I, view of a transverse section; II, Profile view; III, the nuclei of the medulla (partly after Erb). The crosses of color corresponding to the lines upon which they are placed, designate the point of section of each tract as it passes through different levels (the crus and pons). C i, *internal capsule*, with radiating fibres (in yellow), pyramidal fibres (red), and fibres going to the pons (in purple); P C, the *crura cerebri*, with the pyramidal fibres and the fibres going to the ganglia of the pons anteriorly, and posteriorly, the substantia nigra, the fillet tract (in dotted lines), the fibres of the superior peduncle of the cerebellum (in blue); Pc, the *peduncles of the cerebellum*, showing the fibres going to the cerebrum, the pons, and the medulla; P, *pons Varolii*, with its ganglia on either side (in purple). In III, the *nuclei of the cranial nerve roots* are numbered to correspond with the nerves. Red is used for the motor nuclei, and

FIG. 37.



blue for the sensory nuclei. The *tracts in the cord* are designated by the area similarly colored in the cross-section of the cord beneath. c', Column of Türck; c, crossed pyramidal column; a, anterior horn; a', anterior root zone; e, direct cerebellar column; b, posterior horn; b', column of Burdach; d, column of Goll. Higher up are seen b'', the inferior peduncle of the cerebellum; d', the fillet or lemniscus tract; f, the fibres connecting the ganglia of the pons with the cerebrum and cerebellum, b''', the fibres of the superior cerebellar peduncle; h, the caudo-lenticular and thalmo-cortical fibres; i, the commissural fibres (see Fig. 6); Th, optic thalamus; nc, nucleus, caudatus; nl, nucleus lenticularis; gc, central convolutions.

In this diagram, the course of b'' seems to be in error in not undergoing a decussation (Author's note).

SECTION II.

PRACTICAL HINTS REGARDING THE CLINICAL EXAMINATION OF PATIENTS AFFLICTED WITH NERVOUS DISEASES, AND THE VARIOUS TESTS WHICH MAY BE EMPLOYED AS AIDS IN DIAGNOSIS.

SECTION II.

THE METHODS OF EXAMINATION EMPLOYED IN THE DIAGNOSIS OF NERVOUS DISEASES.

The majority of practitioners apparently join in the feeling (which happily conduces largely to the benefit of specialists in neurology) that nervous anatomy and physiology is "too complex a subject for them to master," and that they must be, therefore, given over to those who are devoting themselves particularly to the department of nervous diseases.

While this may be true in part, I believe that it is not only possible but comparatively easy for any medical practitioner (who is willing to make the necessary effort) to grasp certain general principles which are applicable to the examination of cases afflicted with nervous diseases.

These can be applied without expensive apparatus, and with decided benefit both to himself and his patients. They will tend to render his diagnosis more scientific and accurate. They will aid him in properly directing his treatment. Finally, they will often save him the humiliation of seeing his patient seek advice from other hands.

The intelligence of laymen is always strongly impressed by evidences on the part of the physician of great care and marked skill, as shown in the first examination. The impressions left upon the patient's mind by the results of the first interview are of the greatest importance to both parties. While the doctor is studying the patient, the patient is, as a rule, studying the doctor with even greater interest.

Every step which is taken by the physician, as a means of forming a positive and final judgment, is watched with an earnestness on the part of the patient that invariably accompanies mental anxiety. Each question that is asked regarding the previous history of the patient, the possibility of similar troubles in his parents or blood-relations, the origin and course of the more important symptoms, etc., are even more indelibly impressed upon the mind of the patient than upon that of the physician, who keeps the written record.

When, later in the examination, the power or electrical reactions of the muscles, and the sensibility of different regions of the body to touch, temperature, and pain are being tested in various ways, and the results of such tests are being recorded in the case-book of the physician, the reasoning faculties of the patient are even more keenly alive, and seek to penetrate (as far as his intelligence will permit him to do) into the mysteries of the science, and to draw conclusions regarding the clinical

significance of certain symptoms, of which, perhaps, he was unconscious up to that time. It will often be necessary, therefore, for the physician to quiet evidences of alarm on the part of the patient, from time to time, as the examination of the symptoms proceed, by judicious explanation or words of encouragement.

It should ever be remembered by the physician that any omission on his part to investigate the condition of the motor or sensory nerves, the pulse, the respiration, the temperature, the spinal reflexes, etc., in each and every case, will sooner or later be remarked by some patient, who has either read extensively or had, from time to time, different medical advisers. Moreover, interested friends (sometimes very intelligent from past experiences of their own) may often drop hints to the patient which will tend to strengthen or weaken his or her views that have previously been formed of the accuracy and care of the first examination of the symptoms.

It is my intention to give here, with some detail, the description of the various steps that are commonly employed by specialists in neurology in the examination of their patients; and to suggest a simple method of recording symptoms, as a basis for the diagnosis and subsequent treatment of nervous affections.

I shall discuss the subject under the following heads:—

First.—The clinical history of the patient, and how to record the chief symptoms of each case.

Second.—The symptoms revealed to the physician by his sense of sight.

Third.—The symptoms revealed to the physician by instruments of various kinds, and other tests.

I.

THE CLINICAL HISTORY OF THE PATIENT.

Every physician should be provided with a case-book. In it the main features of each patient's case should be first recorded, and a memorandum of the treatment and modifications of the symptoms should be subsequently jotted down at each visit. In this way only can the results of an extended experience be made useful for scientific purposes at some later date. It will furthermore aid the doctor in utilizing his leisure hours by studying the cases which he meets during the busy routine of his office work. One case well studied is worth a hundred casually glanced at and hastily prescribed for.

It will help to economize time if the case-book is printed in such a way as to have the more important symptoms already upon the page; spaces being left blank to allow of a record of any modifications of these

that may exist. This plan adds to the legibility of the notes, and also admirably adapts them for comparison with those of previous or subsequent cases. Each physician may alter the arrangement of the pages of his case-book to suit his individual practice, but it is best for a general practitioner to have it adapted for all classes of patients.

In a subsequent portion of this chapter I will suggest a form of case-book which seems to me to be well adapted to the requirements of a specialist in nervous diseases.

Let us now suppose that a patient enters the office of a physician for medical advice relating to a nervous malady. After the usual questions have been asked the patient regarding the name, the age, the condition as to marriage, the nationality, and the occupation, and the answers recorded, the patient should be brought rapidly to a *concise statement of the more important symptoms* for which he seeks medical relief. This can be usually accomplished by a little tact; and much valuable time is saved by so doing. These symptoms can then be separately recorded upon a page in your case-book.

With these especially marked symptoms as a starting-point, questions may then be asked regarding certain of them which the physician deems the most important from a clinical aspect; seeking in each instance to learn all about the present and past history of *one symptom at a time*, and the modifications which have been observed concerning it, so far as the patient's memory will prove of assistance.

Now, the ability on the part of the doctor to ask questions that are pertinent to each symptom will depend entirely upon the knowledge which he himself possesses of the subject. I have often tested medical students and young practitioners in this regard, and have been amused to see how rapidly their stock of pertinent inquiries became exhausted.

In order to intelligently ask about pain, for example, the physician must know all the axioms of nerve-distribution which Hilton so ably advanced; he must be a master, in the second place, of the course of separate nerves which enable definite regions to tell the doctor (by the presence of the sense of pain) of disease that is sometimes far remote from the painful area; again, he must be able to correctly trace the course of affected nerves, and thus to seek for abnormal conditions along the line of each nerve which might produce local pressure upon them; he must be familiar, in the fourth place, with the individual peculiarities of pain in special diseases, as, for example, the characteristic pains of rheumatism, neuralgia, locomotor ataxia, etc.; finally, he must be familiar with all the possible causes of pain in different regions of the body or extremities.

When we shall have discussed the various symptoms revealed by inspection of the patient, as well as the tests employed to determine ab-

normal states of the motor or sensory nerves, and the reactions of muscles to different electric currents, many points will have been given that may prove of assistance in suggesting pertinent questions, to be employed in obtaining the clinical history of patients so afflicted; but it will require continued practice, much study, and close observation to excel in the art of quickly and accurately gathering pertinent facts, from which conclusions can be drawn regarding the diagnosis and treatment of nervous diseases.

A few general hints may, however, be thrown out here as to special lines of inquiry, each of which may afford us valuable information respecting nervous maladies.

The Duration of Existing Symptoms.—It is important to ascertain the exact date of the commencement of the symptoms for which the patient seeks relief, or of others which may be detected by the physician at the first interview.

This will often decide as to the acuteness of the attack, and also afford in some instances information respecting the seat and type of the disease.

In the chronic varieties of spinal disease (as, for example, progressive muscular atrophy, locomotor ataxia, disseminated sclerosis, etc.), the patient cannot, as a rule, fix the date at which the symptoms commenced because the development has been extremely slow and gradual.

On the other hand, a hysterical fit may be followed immediately by an attack of hysterical paralysis; a hemorrhage into the brain or spinal cord, that has ploughed up the substance of these organs, causes paralytic symptoms that develop instantly; inflammatory changes of the brain or cord are usually attended by a more gradual onset, although it may be comparatively rapid.

As an illustration of the clinical bearing of the duration of symptoms, let us take the following: Two patients present a deformed hand from atrophy of the muscles of the thumb and interossei. The one has been slowly developed, and is probably the result of progressive muscular atrophy; the other has been very rapidly developed, and is probably due to some disease or local injury of the ulnar nerve. Should the deformity have occurred in years past, and have shown no evidences of steady progression, the existence of progressive muscular atrophy could be then safely excluded.

The Exciting Cause of Existing Symptoms.—If there has been any external violence received, it is important to ascertain the exact nature, seat, and severity of the injury.

Concussion of the spine may cause severe and often fatal disease of the spinal cord. Violence to the head may depress the inner tablet of the skull without any evidence of depression upon the exterior. The brain

may be seriously injured, when the bones that encase it may escape. Some of the spinal nerves may be implicated in a wound or bruise, and thus paralysis may be induced independently of the nerve centres.

Mental anxiety or overwork is a frequent cause of brain diseases. Eye-defect acts very frequently as an etiological factor in many cases of headache, neuralgia, hysteria, epilepsy, chorea, and some obscure visceral derangements. Some defects in the eye are inherited (as are peculiarities in feature and mental traits). This field will be discussed later in this chapter.

A family tendency to gout or rheumatism, etc., may suggest the possibility of an abnormal blood-condition as an important factor in creating nervous disturbances.

The urine should always be carefully examined, as well as the heart, to exclude the possibility of renal or cardiac disease as a factor in the nervous derangement.

The Age of the Patient.—Much may be suggested to the mind of the physician by the age of the patient; because some diseases are more common at one period of life than at another.

During early childhood we are particularly liable to encounter the symptoms of idiocy, epilepsy, and chorea, as well as those of an inflammation of the anterior horns of the gray matter of the spinal cord, the so-called "poliomyelitis anterior." The acute variety of the latter disease is most common between the ages of one and four, and it is seldom developed except in childhood. In the vast majority of cases, the condition termed "pseudo-hypertrophic paralysis" (because the muscles are overgrown like those of an athlete) is developed during the first few years of life. Again, the tubercular form of inflammation of the meninges, both of the brain and spinal cord, occur in the young child. Among the rarer forms of disease of the spinal cord, a congenital variety of the so-called "spastic paralysis," and also of "locomotor ataxia," is encountered in young children. Reflex paraplegia is also occasionally seen in very young subjects. Cases of disseminated sclerosis of the spinal cord have been reported in the child.

Between the ages of puberty and the fully developed adult, Pott's disease of the vertebra may develop and create compression of the spinal cord; and attacks of rheumatism may render the development of embolic hemiplegia and aphasia possible. Meningitis of the brain and spinal cord are not uncommon during this interval. Hysterical paraplegia occurs in young females in connection with uterine disturbances. Between the ages of twenty and thirty, cerebro-spinal sclerosis is most commonly developed.

In the adult, progressive muscular atrophy, myelitis, meningitis of the cord, locomotor ataxia, the chronic form of poliomyelitis, and amyo-

trophic lateral paralysis are among the spinal diseases often encountered. Cerebral meningitis, and softening, tumors, and embolism of the brain are frequently recognized. Shaking palsy seldom occurs except in advanced life. The symptoms of Duchenne's disease, and the paralysis of the insane are most commonly developed between the ages of thirty and sixty.

Linked with adult life, also, comes apoplexy associated with paralysis; and a late rigidity of the paralyzed muscles is developed whenever the injury excites a descending degeneration of the fibres that are torn across by escaping blood, or deprives the cerebrum of its power of control over the cerebellum. Excessive indulgence in eating and drinking, coupled with the absence of proper physical exercise, and the possibility of acquired syphilis, render males more subject to paralysis than females.

The Sex.—Males suffer much more frequently from organic nervous affections than females.

This fact is to be accounted for partly by the liability of that sex to injury, exposure to cold or dampness, and excessive mental or physical labor. But habits of indulgence in alcohol and venery, with its danger of syphilitic infection, are also far more common in males than in females, and are often prominent factors in the causation of morbid conditions of the nerve centres. Certain occupations, tending toward great muscular strain, or lead, arsenic, and mercurial poisoning, may be exciting causes of serious nervous affections. Prolonged exposure to compressed air (as in the case of divers) is often followed by paralysis. Many such cases have occurred among workmen in submarine excavations.

The Heredity.—After you have exhausted the special lines of inquiry indicated by the prominent symptoms that each patient seeks relief for, questions should then be propounded to the patient touching upon the possibility of hereditary predisposition to nervous affections or of some hereditary "diathesis."

Some nervous affections exhibit a marked dependence upon a hereditary predisposition, either to the disease actually present, or to some allied disorder. Epileptics, for example, are frequently the offspring of tubercular or syphilitic parents, or of epileptics. Again, chorea and hysteria may be developed from the most trivial excitement (even from imitation of others so affected) in subjects predisposed to nervous excitability or debility. Apoplectic subjects not infrequently beget offspring who manifest in adult life a decided tendency to vascular disease. Certain spinal affections seem to be particularly associated with heredity. A predisposition to cancer and tuberculosis is unquestionably transmitted, and these conditions are not infrequently found in the brain and spinal cord, or their envelopes.

A marked hereditary tendency toward some spinal affections seems to be well established. Pseudo-hypertrophic paralysis is transmitted

through the mother. Locomotor ataxia occasionally runs in families, and progressive muscular atrophy is markedly hereditary. Quite a large proportion of hysterical women can be shown to have sprung from ancestry in which tuberculosis, epilepsy, or insanity has existed; and idiotic children and epileptics sometimes owe their disease to a so-called "hysterical temperament" on the mother's side. I believe that, in many cases, this predisposition can be traced to an inherited defect in the ocular muscles, or a refractive error in the eye itself, which creates eye-strain when binocular vision is attempted. This view is based upon an examination of quite a large number of such cases.

Habits of the Patient.—These should be the next subjects of inquiry.

Alcoholic subjects are always surrounded by dangerous possibilities. Inflammation, when once started in such patients, is liable to be of a severe and fatal form. Trivial injuries often excite serious complications in such subjects, and hereditary or acquired diseases, which have been comparatively dormant for some time, may be kindled into activity by a "spree."

Again the habitual use of drugs for nervousness, sleeplessness, and all the other ailments with which the laity often experiment at the suggestion of friends, but without the knowledge of their doctor, may be a factor in nervous symptoms that have become aggravated or actually developed by their injudicious use. Some patients can use tobacco without apparent injury, while it is a rank poison to others; tea and coffee are likewise injurious to many patients. The long-continued use of chloral, the bromides, opium, or other drugs may result in nervous affections of a serious character.

The Occupation of the Patient.—This may be a possible factor in the development of nervous diseases.

Sewing-girls frequently develop ulceration of the stomach from the pressure exerted upon that organ by stooping. Painters are peculiarly liable to lead-poisoning; and in some arts, where mercurial, phosphoric, and arsenical preparations are extensively employed, symptoms of these forms of poisoning may be developed. Constant or prolonged exposure to cold or dampness is very often an exciting cause of spinal affections. Excessive exercise or occupations demanding an unusual strain upon the muscles may induce actual disease of the muscles, peripheral nerves, spinal cord, or brain. Extreme mental labor or anxiety is a frequent cause of brain inflammation and changes within the coats of the blood-vessels of that organ.

The Acquired Diseases.—Finally, the previous history of the patient in respect to acquired diseases is especially important as an aid in deciding as to the probable cause of the existing symptoms.

All attacks of illness which have been passed through should be carefully inquired into.

The presence or absence of latent syphilis should always be investigated as perhaps one of the most common causes of nervous affections. The presence or absence of tubercular deposits in the lungs, or of cancer in the breast or viscera, should be decided by a physical examination, because similar deposits may exist elsewhere in the body. Some of the fevers often cause sequelæ that create impairment of the senses of sight and hearing, as well as other nervous phenomena. Cerebro-spinal meningitis may leave after-effects upon the nerve centres that last for an indefinite period. Kidney diseases may result in serious changes in the blood-vessels, and thus be a factor in the development of brain troubles. Diphtheria is frequently followed by paralysis of the throat and limbs. Diabetes may itself indicate an existing brain disease; or, as the result of imperfect performance of the digestive processes, create, in turn, symptoms referable to the nervous mechanism. In point of fact, few, if any of the more common diseases are entirely exempt from a more or less direct association with nervous phenomena.

There is a prevalent opinion among the laity (and unfortunately, with some of the profession also) that the nervous system is a distinct and separate part of the human organization; an entity entirely independent of the other organs and having functions peculiarly its own. They seem to forget that it is nourished by the same source as muscle, bone, organs, etc., *e.g.*, the blood; also that every part of the body is capable of sending telegraphic communications to the brain and spinal cord by means of the sensory nerves; and, finally, that these organs are called into action rather as the servants of the other parts of the body than as independent organisms, by the various impressions which they receive from without. All the mental processes are based, of necessity upon some impressions of the outer world gained by means of the organs of sight, smell, hearing, touch, taste, or the nerves of general sensibility.

The apparent disassociation which exists between the nervous centres and the viscera often misleads the practitioner of medicine, and causes him to disregard the importance of a complete examination of the various organs before a final judgment is expressed concerning nervous phenomena that are brought to his notice.

Some of the more common forms of nervous affections are purely functional. Text-books abound in cases where some disease of the intestine, ovaries, uterus, kidneys, bladder and urethra have been the exciting cause of paralysis, and of serious effects upon the nerve centres. The eye is also a very frequent factor in functional nervous diseases—although the fact is not generally recognized by authors. This field will be discussed later. Hysteria is often associated with an attack of paralysis

that is not easily differentiated from the types of paralysis produced by destructive processes within the brain and spinal cord. Epilepsy and St. Vitus' dance are purely nervous diseases, and yet they may sometimes be the indirect result of a defective assimilation of food, general debility, some poverty of the blood, and many other causes that are not directly associated with the nervous system proper.

On the other hand, diseased conditions of the nervous centres may induce so-called trophic changes, or changes of nutrition, not only in the muscles—as is evidenced by atrophy of a more or less complete kind—but also in the skin, the various organs, the joints, and even in the bones.

The peripheral nerves preside, not only over the muscles to which they give the power of contraction, and the tactile organs of the skin, to which they contribute the ability to perceive all varieties of impressions, such as the tactile sense, the sense of cold and of heat, the feelings of pain, etc., but they have another equally important function, which they exercise chiefly by means of the so-called vaso-motor filaments, viz., the regulation of the blood supply to the viscera, organs of special sense, muscles, bones, joints, and skin. Now, when the nerve centres become involved by any form of destructive process that cuts off these so-called "trophic fibres" from connection with certain parts of the spinal cord or brain, definite regions of the body may waste away without exhibiting paralysis, the eye and ear may lose their marvelous functions, and the skin may develop different forms of eruptions, bed-sores, etc.

Finally, the spinal cord and the medulla oblongata (which is its uppermost portion) contain certain collections of nerve cells or "reflex centres" that preside over the more important functions, or those essential to life.

By means of an excitability which is present in these collections of cells, the heart is kept pulsating; the respirations go on, even in spite of any voluntary efforts made to arrest them; the pupil dilates and contracts when exposed to different degrees of light; and the bladder and rectum expel the excretions that accumulate within them. In the same way the sexual act is rendered possible in the male; the stomach and intestine keep up a perpetual worm-like movement; swallowing is performed in such a way that the food does not enter the air-passages or pass upward into the nose; the calibre of the blood-vessels is constantly altered, so as to meet the demands of different parts of the body when active or at rest; and the acts of vomiting, hiccough, sneezing, sighing, laughing, etc., are rendered possible, and often involuntary.

In closing this section of the chapter, I take the liberty of presenting a sample page of my own case-book, specially designed for the recording of the results of the first examination of patients afflicted with any form of nervous malady. Some of the headings will be discussed in subsequent

pages. Their bearings upon diagnosis will then be made clear. The page which faces the printed one is left blank to allow of subsequent record of the treatment and any new symptoms that may arise.

NAME.....AGE.....OCCUPATION.....DATE.....188....

Hereditary tendencies:
- Parents.....
- Brothers and sisters.....
- Near relatives.....

Clinical history:

Acquired diseases—
- Fevers.....
- Lungs.....
- Kidneys.....
- Pelvic organs.....
- Venereal.....

Habits, as to diet.....
- " " alcohol.....
- " " tobacco or drugs.....
- " " venery.....

Motor phenomena.....

Sensory phenomena.....

Attitude.....

Gait.....

Sense of smell.....

Eye:
- Pupils.....
- Lids.....
- Ocular movements.....
- Vision.....
- Condition of fundus.....

Ear:
- Deformities.....
- Hearing.....

Mouth:
- Taste.....
- Articulation.....
- Deglutition.....
- Attitude of lips.....
- Movements of tongue.....

Brain:
- Memory.....
- Emotions.....
- Logical powers.....
- Sleep.....
- Aphasia.....
- Vertigo.....

Spinal cord:
- Superficial reflexes.....
- Deep reflexes.....
- Delayed sensation.....
- Anæsthesia.....
- Hyperæsthesia.....
- Pain.....
- Co-ordination.....

Pulse.....

Temperature.....

Respiration.....

Voice.....

Tremor.....

Fibrillary twitchings.....

Handwriting.....

Condition of arteries.....
- of urethra.....
- of bladder.....
- of uterus.....
- of ovaries.....
- of urine.....
- of heart.....

DIAGNOSIS AND REMARKS.

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II.

SYMPTOMS OF NERVOUS DISEASES REVEALED TO THE PHYSICIAN BY HIS SENSE OF SIGHT.

When a patient and his medical adviser meet for the first time there are many medical facts which may be detected simply by a glance, without a question being asked. Sometimes information thus gained is invaluable to the doctor, and of the greatest importance in diagnosis. To become skillful in this line, however, both study and practice are requisite.

Some years since I published, in the *New York Medical Journal*, a contribution to the study of medical physiognomy which has been honored by two foreign translations; and, in my late work on "Medical Anatomy" (Wood's Library for 1882), I have devoted an entire chapter to the subject. In this article, however, I shall only touch upon such points as are related to the diagnosis of nervous diseases.

This section of my article I shall discuss under the following heads: 1, The study of the features and general appearance of the patient. 2, The study of the gait and the attitude of the patient, when sitting, standing, or reclining.

The Features and General Appearance of the Patient.—One glance at a face affected with such striking alterations as those produced by Bell's paralysis, Duchenne's palsy in its advanced state, marked atrophy of the facial muscles, and some other nervous conditions which are associated with extreme facial deformity, would be sufficient with even an inexperienced practitioner for a diagnosis. But all diseases of the nervous centres, or of the cranial nerves themselves, independently of the brain, are not so forcibly evidenced in the face. Something of value can, however, usually be learned by a careful study of each of its parts, especially the forehead, eye, lips, tongue, and ear. It has been my custom for some years to have impressions from *untouched photographic negatives* made of many of my patients before any mode of treatment was commenced. I have found them very useful in many ways; and they certainly constitute the easiest and most reliable method of recording some medical facts.

A prominent and tortuous artery upon the temple may catch the eye of the doctor. It is well to know that such a condition often accompanies kidney disease.

A scanning of the face will show whether the complexion is ruddy, as in health, or pale from some cachexia; clear and free from eruptions, or sallowed and dingy; waxy and transparent, as in Bright's disease, or tinged with blue from imperfect oxygenation of the blood.

In children, certain lines or wrinkles may possibly exist that point strongly to some complicating disease of the brain, lungs, heart or digestive organs, the presence of persistent pain, and other valuable data. In adults, or the aged, these lines are of less clinical importance. I have discussed them in other articles, previously referred to.

A collar loosened or open may suggest some difficulty in breathing. An untied shoe may cover a dropsical foot; a slit in the region of the "great-toe" joint may have been made as a relief to gouty inflammation; one shoe badly worn at the toe may tell of an existing hemiplegia. Patients with enfeebled mental powers and drunkards are particularly liable to have their clothing wrongly or incompletely buttoned; the pants im-

perfectly closed or open; the shoe down at the heel; the hair uncombed, and to present other evidences of indifference to neatness of appearance.

Good, strong hair in abundance, and teeth that are free from defects, are evidences in the adult of a naturally vigorous constitution. Broad shoulders and deep chests are likewise an indication of inherited strength both of the organs and muscles.

The Diathesis.—The general appearance of the patient may afford some valuable information respecting an hereditary diathesis. Laycock has admirably described them.

Patients of the "*gouty*" *diathesis* usually have a heavy frame, well-developed muscles, a large head and jaw, strong hair and teeth, a robust appearance, and an erect carriage. They are peculiarly susceptible, in adult life, to diseases of the blood-vessels, apoplexy, aneurism, and heart troubles.

In contrast to this type, those of the well-marked "*strumous*" *diathesis* have a light, bony framework, which is often characterized by an enlargement of the ends of the long bones. The hand is sometimes unshapely from this peculiar defect, or the rings which will pass the joints are too large for the finger. The chest of such subjects is also small. The glands of the neck tend to become enlarged at about the age of puberty.

The hair of strumous subjects is apt to be thin and fine. The eyelashes are usually long and silken, although the lids may sometimes be diseased and the lashes more or less disfigured. As children, they are liable to be unusually precocious. The teeth are crowded into a narrow arch and are liable to decay early. The under jaw is light. Evidences of rickets in childhood may exist during adult life. Scrofulous children inherit "either a velvety skin, dark-brown complexion, dark hair, dark brilliant eyes and long lashes, with the lineaments of a face finely drawn and expressive; or a fair complexion, thick and swollen nose, broad chin, teeth irregular and developed late, inflammation of the Meibomian glands, scrofulous ophthalmia, eruptions of the head, nose, and lips, and enlarged cervical glands." These subjects are often "chicken-breasted" and "bow-legged." The "strumous diathesis" entails a peculiar liability to defective nutrition, glandular enlargements, and "consumptive" changes within the lungs during early manhood. Epilepsy and hydrocephalus often develop in such subjects during infancy or childhood.

The so-called "*nervous*" *diathesis* is commonly associated with small but perfect bones, an absence of fat, a well-formed cranium, small features, quick intelligence, and an active frame. They usually have a bright eye and small abdominal organs. They bear fatigue well, but are peculiarly susceptible to nervous excitability and depression. In adult life they

become the more common victims to neuralgia, epilepsy, hysteria, dipsomania, and many other nervous diseases.

Dark-haired and swarthy subjects are often of the so-called "*bilious*" *temperament.* They commonly possess large frames, strong muscles, and a tendency to moderate obesity. They are active rather than lethargic. The digestive organs are often disturbed by habits of over-indulgence at the table or excessive mental efforts. Such subjects commonly suffer from "sick-headaches" from early childhood, and often develop gouty symptoms in early adult life. They are not infrequently victims to vascular changes, kidney disease, and apoplexy, after the age of fifty years.

The "*lymphatic*" *diathesis* is generally met with in sluggish, lazy, and large subjects. They are commonly addicted to alcohol (because they suffer from fatigue) and to excessive eating. They have heavy bones, but soft and flabby muscles. They are often pale. They usually thrive best in invigorating climates.

Now, it must be remembered that it is seldom that the physician meets either of these types unadulterated. A man of the gouty diathesis, with a wife of the "nervous" type, will probably have children that exhibit certain characteristics of both. Hence it is often desirable, before making a diagnosis, to inquire into the peculiarities of build and temperament of the ancestors of patients afflicted with nervous diseases, as well as to their duration of life and the causes of their death.

The Cachexia.—These are diseased conditions. The ones which are most frequently recognized by the neurologist are those of syphilis, cancer, gout, mercurial or lead-poisoning, and malaria. In all of these there is poverty of the blood, because the red corpuscles are more or less destroyed and the constituents of the blood-plasma are altered. If a cachexia is superimposed upon some special form of diathesis, a double danger to the patient is the result. A strumous subject, for example, may have his tubercular tendencies materially hastened, if not actually developed, by malaria, syphilis, and mercurial or lead-poisoning.

Special Physiognomy.—As the physician scans the features of his patient, it is best to inspect different parts of the face separately, as it were. Let us note what he should particularly observe.

The Forehead.—If the forehead be well developed, the "nervous diathesis" is liable to be present. If protuberant and overhanging a small and imperfectly-developed face, rickets, hereditary syphilis, or hydrocephalus have probably existed in childhood. If hereditary syphilis has conduced toward the cranial deformity, the teeth will be found to be defective. Ulceration upon the forehead, unless it be due to a wound, is invariably syphilitic. Scars of this region or copper-colored spots are equally significant and suggestive. Depressed fractures over the frontal region are not necessarily associated in the adult with injury

to the brain, even if extensive, because the frontal sinuses are developed after puberty, and the front wall of the sinus may be then crushed in without disturbing the back wall or the underlying brain. A very small cranium and a retreating forehead are often present in imbeciles.

The Eye.—In the aged, if the cornea be cloudy, you should lift the upper eyelid and seek for an arc of a lighter shade—the so-called "*arcus senilis.*"

If it exist, and its edges are indistinctly defined, there is reason to suspect that the tissues of the body (especially the heart) are undergoing fatty degeneration.

The *pupils should be examined* to see if they are equal in size, and if their movements are in any way impaired.

There is one condition, called from its discoverer the "*Robertson pupil*," that is of the greatest significance to the neurologist, because it indicates a hardening, or "sclerosis," as it is called, of the spinal cord. It occurs only when this disease has involved the "cilio-spinal centre" of the cord. This condition is indicated in the eye by preternaturally small pupils that *do not respond to light*, but which still move when efforts to accommodate the vision to near objects (*i.e.*, within a radius of twenty feet) are demanded.

To test this fact, place the patient at a window and instruct him to look fixedly at some object more than twenty feet off whenever his eyes are open, so that the pupil need not contract in order to focus the vision. Now tell him to close the eyes and keep them closed until instructed to open them. After sufficient time has elapsed for the pupils to have become dilated, tell him to open his eyes. Watch carefully at this moment for a response in the pupils, as they will contract instantly in health. If they fail to do so, the existence of spinal sclerosis is almost positively indicated.

Abnormalities of the pupils may afford the practitioner material aid in diagnosis.

The pupils are found to be dilated during attacks of dyspnœa and after excessive muscular exertion, in the later stages of anæsthesia, and in cases of poisoning from belladonna and other drugs of similar action. A contracted state of the pupils exists during alcoholic excitement, in the early stages of anæsthesia from chloroform, and in poisoning by morphia or other preparations of opium, physostigmine, chloral, and some other drugs. Paralysis of the third cranial nerve creates a dilated condition of the pupil of the same side, since that nerve controls the circular fibres of the iris.

Again, one pupil may dilate irregularly in a weak light. This suggests the existence of adhesions of the iris, as a result of past inflammation. Iritis is often syphilitic, and this symptom may tell of past

infection. The inner surface of the eyelid is a valuable guide to detect the presence of anæmia, as it shows a pallor that is in marked contrast to the redness of health. Alcoholic subjects are apt to have a vascular redness of the eyeball. Bright's disease often causes a drop of fluid beneath the conjunctiva that might be mistaken for a tear. It can be moved, however, while a tear cannot without causing its disappearance.

In connection with hemianopsia (see previous section) there may be an absence of pupillary movement upon one lateral half of each eye—the so-called "hemiopic pupillary reaction."

The *movements of the eye* should be a subject of special inquiry. Brain diseases sometimes manifest their existence very early by some form of paralysis of the ocular muscles. Strabismus or cross-eye may exist when the third or sixth cranial nerves are impaired. We meet it chiefly in connection with hydrocephalus, apoplectic clots, brain-tumors, cerebral meningitis, growths within the orbit, and as a congenital or acquired deformity. This subject will be fully discussed later.

It is a fact well known among oculists, and one which often helps them materially in diagnosis, that the defects of vision occasioned by a serious impairment in the power of some of the muscles which control the eyeball, cause the patients unconsciously to assume an *abnormal position of the head*, which tends to assist them in the use of the affected eye. So diagnostic are some of the attitudes assumed by this class of afflicted people, that the condition which exists may be told at a glance, as the patient enters a room, by one thoroughly familiar with diseases of this important organ. The explanation of this tendency on the part of this class of patients lies in the fact that a loss of power in the ocular muscles may immediately show itself in the perception of every object, as it were doubled; and it is to overcome these double images that patients almost instantaneously discover their ability to get rid of the annoyance by some special attitude, which, of course, depends upon the muscle that is weakened or paralyzed.

It will be necessary, in order to clearly understand the mechanism of this peculiarity, that the separate action of the six muscles which directly act upon the globe of the eye be considered.

The action of each of the ocular muscles may be given, then, as follows, with the proviso that many of the motions of the eye are not the result of the contraction of any single muscle, but often of a number acting either in unison or successively.

The superior oblique muscle turns the eye downward and outward; the inferior oblique muscle turns the eye upward and outward; the superior rectus muscle turns the eye downward and inward; the internal rectus muscle turns the eye directly inward; the external rectus muscle turns the eye directly outward.

This statement as to the above muscles reveals nothing which would not be immediately suggested by the insertion of each, with the exception of the superior and inferior recti muscles, which, besides the action that their situation would naturally suggest, tend also to draw the eyeball inward, on account of the obliquity of the axis of the orbit, and the same obliquity of the muscles, since they arise at the apex of the orbit. The action of the oblique muscles is, as any one familiar with their origin and insertion would naturally surmise, to control the oblique movements of the eyeball.

Now, as soon as any one of these six muscles becomes pressed upon and weakened by the presence of tumors, inflammatory exudation, syphilis, or other causes, the patient at once perceives double images, and, in order to get his eye into such a relative position with that of the healthy side as to enable them both to focus upon the same object in a natural manner, the patient soon learns to so move his head as to compel the two eyes to look in parallel directions.

A very simple rule can be suggested by which the physician may be enabled not only to tell in what direction a patient would move his head in case any special muscle be rendered weak or utterly useless, but also to diagnose the muscle affected, when he looks at the patient, without any knowledge of his history. The rule may be thus stated: *In paresis of any of the ocular muscles, the head is so deflected from its normal position that the chin is carried in a direction corresponding to the action of the affected muscle.*

Thus, in paresis of the external rectus,* the chin would be carried outward toward the affected muscle; while in paresis of the internal rectus muscle the head would be turned away from the side on which the muscle fails to act. In case the superior oblique muscle is impaired, the chin would be carried downward and outward; while in case of the inferior oblique muscle, the chin would have to be moved upward and outward to benefit the vision of the patient. The superior and inferior recti muscles, when impaired by disease or other causes, would likewise create a deflection of the head in a line corresponding to that of their respective actions.

Paresis of the external and internal recti muscles occasionally causes, in addition to the facts already described, another point of very great value in diagnosis, viz., an alteration in the apparent size of the objects seen from what they would be in health. The condition of vision

*While this statement would be absolutely true in theory in all cases, we must acknowledge, as a clinical fact, that patients learn to utterly disregard the image in the affected eye when the internal or external rectus is the seat of paresis, and to use the normal eye only for the purposes of vision, thus rendering this attitude of the head less diagnostic than when the oblique muscles are affected.

termed by oculists "megalopsia," or "macropsia," often signifies paresis of the external rectus; while the opposite condition, called "micropsia," may indicate a loss of power in the internal rectus muscle.

In the former of these conditions, the objects seen by the patient seem to be greater in point of size than the intelligence of the patient assures him is the case; while in the latter, objects seem smaller to the patient than they really are.

THE EYE AS A WHOLE.

I take the liberty of inserting, in this connection, an extract respecting the eye from my brochure on medical physiognomy:—

The intimate communications between the fifth, the seventh, and the sympathetic nerves, through the media of the ciliary, optic, and Meckel's ganglia, would lead us to expect that the eye should exhibit in its altered appearance the derangement of internal structures. "When a glance of this organ is caught, what a field of mute expression is open to the mind! This silent or instructive index of the whole man may be bright or dull, heavy or clear, half-shut, or unnaturally open, sunken or protruded, fixed or oscillating, straight or distorted, staring or twinkling, fiery or lethargic, anxious or distressed; again, it may be watery or dry, of a pale blue, or its white turned to yellow."

The pupils may be contracted or widely dilated, insensible to or intolerant of light, oscillating or otherwise, unequal in size, or changed from their natural clearness of outline. "The noble arch of the brow speaks its varied language in every face of suffering humanity. It may be overhanging or corrugated, raised or depressed; while the lid of the eye, an important part of this vault, exhibits alternations of puffiness or hollowness, of smoothness or unevenness, of darkness or paleness, of sallowness or brown discoloration, of white or purple. Lines intersect this region, and the varied tints are perpetually giving new color, new feature, new expression, by their shadows." If the frontal muscle acts in connection with the corrugator supercilii, an acute deflection upward is given to the inner part of the eyebrow, very different from the general action of the muscle, and decidedly expressive of debilitating pain, or of discontent, according to the prevailing cast of the rest of the countenance. An irregularity of the pupils of the two eyes indicates, as a rule, pressure upon nerve centres or upon the optic nerve itself. In adynamic fevers the eyes are heavy and extremely sluggish, and are, as a rule, partially covered by the drooping eyelid; while in certain forms of mania they are seldom motionless. This latter peculiarity is also often noticed in idiocy.

In the so-called "Bell's paralysis," due to failure of the facial nerve, the eyelids stand wide open, and cannot be voluntarily closed, since

the orbicularis palpebrarum muscle is paralyzed. This condition may be further recognized, if unilateral, by a smoothness of the affected side, since the antagonistic muscles tend to draw the face toward the side opposite to the one in which the muscular movement is impaired; an inability to place the mouth in the position of whistling, because for this act the two sides of the face must act in unison; loss of control of saliva, which dribbles from the corner of the mouth; and a tendency to accumulation of food in the cheek since the buccinator muscle no longer acts.

When the third pair of nerves are affected upon either side, the upper eyelid cannot be voluntarily raised, for the levator palpebræ muscle fails to act; and the eye is caused to diverge outward, because the external rectus muscle, not being supplied by the third pair and having no counterbalancing muscle, draws the eye from its line of parallelism with its fellow. In photophobia, attempts to open the eye create resistance on the part of the patient, since the entrance of light causes pain; while, as death approaches, or in the state of coma (save in a few exceptions), the eyes are usually open. In cardiac hypertrophy, an unusual brilliancy of the eye is perceived, since the arterial system is overfilled from the additional power of the heart. A peculiar glistening stare exists during the course of scarlet fever, which is in marked contrast with the liquid, tender, and watery eye of measles. Many diseases of the eye itself tend to greatly alter the normal expression of the face. Prominently among these may be mentioned cataract, glaucoma, cancer, staphyloma, exophthalmus, iritis, conjunctivitis, amaurosis, etc., but the special peculiarities of each need not be here described.

THE EYE AS A FACTOR IN THE CAUSATION OF SOME COMMON NERVOUS SYMPTOMS.

Although something has been written within the past few years in relation to the deleterious effects of errors of refraction and accommodation of vision and the condition known as "muscular insufficiency" upon the functions of the nervous system and the viscera,* the profession at

*Priority in this field (save in respect to ocular defect as a cause of headache, which has been recognized in a somewhat imperfect way for many years) is justly claimed, as far as I know, by Dr. George T. Stevens of New York. Although his views have been regarded by some as extreme and untenable, those who have carefully and accurately investigated the eyes of nervous subjects cannot, I think, deny that defects in refraction and accommodation, and insufficiency of the ocular muscles, are very important and generally neglected factors of causation. Authors cannot afford to-day to utterly discard all mention of the tests for muscular insufficiency from neurological works, as they have done in the past. In point of fact, even the tests for errors in refraction are not described in the standard works on nervous maladies. Most authors seem to have been content with showing a cut of some ophthalmoscope and dismissing the subject with a few lines. It is safe to infer that such writers are either not familiar with the field here discussed, or not in the habit of employing the tests herein described upon their patients. I am sure (if this is not the case) they could not remain so apathetic and apparently indifferent to the results obtained

large is not yet thoroughly awakened to the importance of the detection and correction of such errors. I deem it of the greatest importance, therefore, to call attention to it again in this connection, and to give a full description of the testing of vision and of the eye muscles.

Most of you know that some persons can be made dizzy by looking from a height or inspecting a water-fall; you have doubtless seen laymen suffer pains in the head and be made "sick at the stomach" by trying on a pair of spectacles which gave relief to a friend.* You doubtless know that a "squint" in the eyes is very often due to some defect in the refraction of the eye or a weakness of its muscles; but possibly some of you do not know that a squint will occasionally disappear at once when the proper glasses are given to such a patient, without recourse to cutting the muscle. Perhaps it has never occurred to most of you that sight is the *only special sense which we use constantly* except during the hours of sleep. There is not a moment of the day or evening when we are not acquiring visual impressions of some kind.

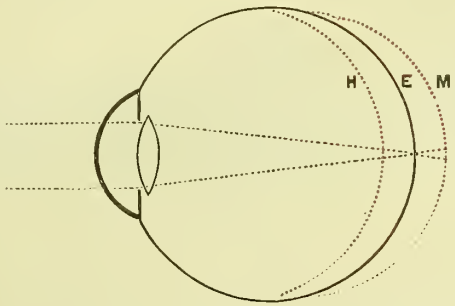


FIG. 38.—DIAGRAM TO ILLUSTRATE CONGENITAL OR ACQUIRED DEFECTS IN THE ANTERO-POSTERIOR DIAMETER OF THE EYE.—The *black line*, E, represents the *normal line* of the eye; H, represents the *hypermetropic* eye; M, the *myopic* eye.

Fortunately for our nervous system, the normal eye takes pictures of surrounding objects *without any muscular effort* when the object is more than twenty feet away; hence, during the larger part of each day, the *normal eye is passive*, and is practically at rest, although performing its functions. How different is the condition of the far-sighted or "hypermetropic" eye, however, from the normal! For this eye (since it is *too short* in its antero-posterior axis) all objects *have to be focused by muscular effort*, irrespective of their distance from the eye. Such an eye is never passive. It has no rest while the body is awake. It is always straining more or less intensely to bring properly upon the retina the images of objects seen.

* Let a healthy child try on its grandfather's spectacles and wear them for a time, and the effects of "*eye-strain*" will be very clearly exhibited *by distressing symptoms in a few minutes.*

THE HYPERMETROPIC EYE.

The "hypermetropic" condition of the eye, or "far-sightedness," as it is called, is a very common defect. It is especially frequent in persons of tubercular parentage.* It is well, therefore, to suspect the existence of this defect in children or adults whose ancestors have died of "consumption."

Hypermetropia cannot be corrected too early in life. It is unquestionably one of the *most frequent causes of "sick-headache,"* which, as you know, runs in families. It is commonly encountered also (among other optical defects) in subjects afflicted with chorea and epilepsy.† It is a congenital defect, and will never be "outgrown."‡ as many people think. A hypermetropic child, from the days of babyhood, suffers (unconscious perhaps of the fact) from a variety of symptoms which indicate the "strain" to which it is subjected in consequence of its efforts to see distinctly. Its eyes are liable to become easily suffused when it plays or looks steadily at near objects. A slight cast in the eye is sometimes developed. It occasionally "sees double" after it learns to read. It usually prefers and excels in out-of-door sports, which require only slight efforts at accommodation of vision. It finds that study and close application to books bring an indescribable sense of weariness and discomfort; hence, study becomes irksome and play brings a sense of peculiar relief. Some years ago Dr. Loring, of this city, wrote an article for *Harper's Monthly* which treated of hypermetropia and myopia in a charmingly lucid and popular manner.

* This is probably due to the shallowness of the orbits.

† Dr. George T. Stevens was the first, so far as I know, to advance the general proposition that ocular defect was an important factor in causing functional nervous diseases, that muscular insufficiency (chiefly of the externi) was particularly apt to cause such disturbances, and that they could be relieved by tenotomy. I have an epileptic child under my care at the present time whose attacks have averaged four a day for several years. *The fits will cease at once when the child is at sea*, possibly because efforts of accommodation are almost entirely dispensed with when on deck. Hypermetropia, astigmatism, and external insufficiency exist in this patient. The use of atropine caused a complete cessation of the fits for several days. Why cannot the eye act as a disturbing element as well as phimosis, sexual excesses, ovarian irritations, etc., concerning which so much has been written?

‡ It is a well recognized fact that people who are victims to sick-headaches early in life tend, as a rule, to suffer less from such attacks after the age of forty. This is not generally attributed (as in my opinion it should be in many cases) to the enforced use of glasses in writing, sewing, reading, and other forms of near eye-work. Most of this class of sufferers are hypermetropic to a marked degree; hence they are compelled to relieve their "accommodation" by a glass earlier than most adults.

These subjects, therefore, do not "outgrow their malady;" nor does the eye improve in regard to its refractive error as age advances. They simply aid the eye at last with a glass, which it has too long needed; not voluntarily in most instances, but from compulsion, because the focusing muscle of that organ is unable after a while to continue to work under the strain which the refractive error has entailed upon it.

Now, one peculiar fact should be noticed here—viz., that *hypermetropic subjects often have remarkable acuteness of sight.* They are very apt (when young adults) to boast of their power of vision. They can often read all the test-types made for distance (twenty feet or more) without an error. If the defect exists in a child, the parents will frequently tell you how the child can see things with distinctness which possibly they themselves cannot see at all; how they have tested its eyes from time to time; how absurd the idea seems to them and their friends that the vision of the child is defective; and how unnecessary the use of glasses seems to them (even if the eye is abnormal) so long as the child can get along without them. In some cases no amount of explanation or pleading will persuade the parents to have atropine used upon the child's eyes in order to positively decide the question of the existence of "latent" far-sightedness.

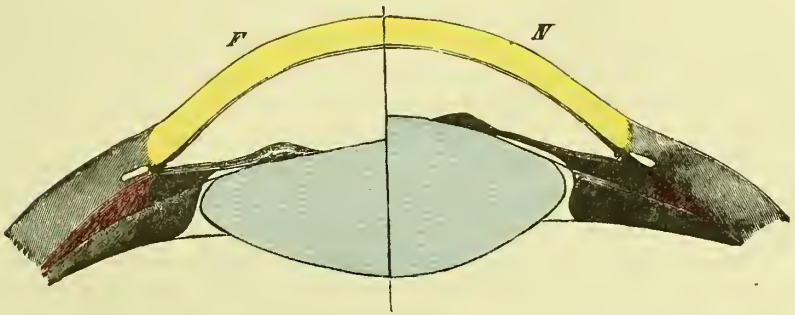


FIG. 39.—SECTION OF THE FRONT PART OF THE EYE, SHOWING THE MECHANISM OF ACCOMMODATION. (Fick.) The left side of the figure (*F*) shows the lens adapted to vision at distances of over twenty feet; the right side of the figure (*N*) shows the lens adapted to the vision of near objects, the ciliary muscle being contracted and the suspensory ligament of the lens consequently relaxed.

Some years ago I pleaded with a medical man to allow some oculist of reputation to examine his children's eyes, all of whom had weekly attacks of sick-headache, inherited from both the mother and father, and in whom a tubercular tendency was strongly marked. I was refused, and the statement was made that never, while the father lived, should a child of his wear glasses with his consent. One of these children wears to-day a convex glass with a twelve-inch focus for distance; another wears the same glass with five degrees of prisms added. These only partially correct an insufficiency of the muscles which exists in addition to the hypermetropia. A third child is highly hypermetropic and astigmatic. In every one of these subjects immense relief has been afforded by the correction of an optical defect which had rendered their early life one of suffering. This is not an uncommon experience. I could cite many more, if I deemed it necessary to prove what is already accepted by ophthalmologists as proved—viz., that hypermetropia and eye-defect of other forms may prove to be *fruitful sources of headache.*

There is a prejudice among laymen and some medical men that glasses are an injury when they can be avoided; because, as they say, "a person becomes so dependent upon them when he once puts them on." This argument should be exactly reversed, and construed as follows: *Because nature becomes dependent upon a glass which gives relief and corrects an existing strain upon the eye, no time should be lost in affording this relief.*

Should a hip-splint be avoided (when the pain in the joint is arrested by it) because the patient feels his dependence upon the splint? Should a child be allowed to go through life with a deformed eye simply because the defect is not apparent to himself or his friends on account of an unnaturally-developed ciliary muscle (see Fig. 39), which for a time renders the eye capable of getting along tolerably well in spite of its deformity?

More harm is being done to-day to the community at large by this fallacious argument than it is possible to compute. Thousands of sufferers from sick-headache and neuralgia are to-day struggling along through life with an optical defect uncorrected, and, in many instances, after costly experimentation with drugs and doctors, are left in despair of cure.

I speak strongly upon this point because I believe that the gastric symptoms which accompany typical attacks of sick-headache are not to be explained (as they commonly are) on the ground that the "liver is inactive," or that "dyspepsia exists," or that "the gastric juice is weak," or that "the patient uses tobacco to excess," or that "he has been living too high." Every one who has suffered for years with these attacks knows that they often occur without explainable cause; that they are cured sometimes by eating, drinking, and smoking, and made worse at other times by similar indulgences or excesses; that every known remedy is apt, sooner or later, to prove inoperative, and that a sure specific for them is unknown among the drugs of our Pharmacopœia. These subjects also know that life is rendered almost unendurable by the attacks at times. They are tractable patients, and will try anything, live in any way specified, and bear any privation without a murmur, if it will insure a cure.

I believe, from a personal experience of my own of this kind (which it is unnecessary to relate here), and from some experience also in examining the eyes of this class of sufferers, that the symptoms of sick-headache are reflex in character to a large extent, and are due primarily in almost every case to some ocular defect. We can easily demonstrate that disturbed brain-action from "eye-strain" may produce in a healthy child and in some adults all of the symptoms of these attacks in a few minutes. Why is it irrational, therefore, to affirm that a brain (disturbed by the constant efforts made to use eyes which are abnormal in respect to the refraction, accommodation, or the equilibrium which should exist

between its various muscles) may manifest its disturbed state by nausea, headache, vomiting, dizziness, constipation, and other evidences of imperfect performance of the functions of the viscera? Does not our central nervous system regulate and directly control those functions? Is it not as probable that the master when upset disturbs the servants under him, as to advance the argument that the servants themselves are the all-important factors in causation?

THE MYOPIC EYE.

When the eye is too long from before backward, the patient is said to be "myopic," or *near-sighted.* Distant objects are more or less indistinct to such an eye in proportion to the excessive length of the antero-posterior axis of the eye over the normal standard. No amount of muscular effort can overcome or improve this defect in vision; hence these individuals are not subjected to the muscular strain which far-sighted persons constantly and unconsciously exert in order to see at a distance. Again, the near-sighted eye can read or perform any of the functions required of it (when brought sufficiently close to the object) without any muscular effort of an unnatural character. In contrast, the far-sighted eye has to exert a still greater muscular effort to see near objects distinctly than when employed upon distant objects; hence the fatigue, the blurring of letters upon a printed page, the watering of the eyes, the pain in the eyes and head, and the many other ills previously described.

Near-sighted subjects are generally conscious of an eye-defect, because they cannot see across a room with distinctness or recognize familiar faces on the street. They are apt to become very fond of occupations which brings the eye close to their work, because they have no difficulty in seeing the object. Near-sighted children are liable to be considered precocious beyond their years, because they prefer to read rather than to play out-of doors. It is generally safe to conclude that a child is near-sighted when it avoids out-of-door amusements in order to gratify a taste for reading or in-door occupations.

Near-sightedness is less liable to induce nervous disturbances than far-sightedness, provided it is not accompanied by astigmatism or muscular insufficiency. Yet it should be remembered that myopic subjects are more frequently sent to the oculist for relief than hypermetropic subjects are, because the defect in vision is very apparent to all in the former class, and is more often unsuspected than recognized in the latter.

THE ASTIGMATIC EYE.

You may find, in the third place, when you have examined the eyes of patients or friends who suffer from headache, persistent neuralgic attacks, etc., that a condition of the eye known as "*astigmatism*," may

be detected, co-existing with far- or near-sightedness, or independent of these refractive errors. In such subjects the cornea or the lens of the eye (see Fig. 39) has a *greater curvature in some meridians than in others;* hence the images of all objects seen are more or less distorted when they fall upon the retina. To this class of sufferers some letters in the tests employed will be distinct, while others will not. If a number of dots are made upon a blackboard or a sheet of paper, some will appear as ovals, with a hazy border, or as lines, while others will more closely resemble the normal appearance of the dots. Finally, if a card, with lines running from its centre to its periphery (the "clock-face test"), is used, some of the lines will appear blacker than the rest and more clearly defined. Now, there can be no comfort to such subjects in their visual efforts. They learn by practice and experience to properly interpret, after a while, the imperfect images of objects seen, and they are aided in so doing by the fact that the outlines of letters, etc., become clearer in some positions, as regards the eyes, than in others; but, in spite of all that may be said to the contrary, the strain of using imperfect eyes tells upon most astigmatic persons sooner or later, and tends to excite reflex nervous phenomena of various kinds. To properly correct astigmatism by glasses is often an extremely difficult matter. It requires experience, a thorough knowledge of optics, and a familiarity with the practical use of the ophthalmoscope. There are comparatively few physicians (outside of the specialists in ophthalmology) who are capable of managing a bad case of this kind with perfect success. You can, however, easily detect its existence in most cases. When you discover it, I would advise you to intrust its correction to skillful hands.

Certain abbreviations are employed by oculists to designate various forms of astigmatism which may be detected. These are of use in recording the results of an examination:—

Ah stands for *simple hypermetropic* astigmatism.

Am stands for *simple myopic* astigmatism.

$H + Ah$ stands for *compound hypermetropic* astigmatism.

$M + Am$ stands for *compound myopic* astigmatism.

$M + Ah$, or $H + Am$, stands for *mixed astigmatism.*

THE ASTHENOPIC EYE.

Finally, it is very important that you determine (in each patient whose eyes are examined by you) the *condition of the muscles* of the eye. The term "asthenopia" is commonly applied to that condition of the visual apparatus which entails suffering in consequence of a defective "equilibrium" in the muscular power exerted upon that organ when a fixed position of the eye is maintained for any length of time. When a state of perfect equilibrium is impaired from a weakness in some muscle

of the eye, the effects become manifested sooner or later by pain and great discomfort after the eyes are used for any length of time. I have seen patients who could not attend a place of amusement, or read or sew, for even a short time, without great distress from this cause. These patients may or may not have a refractive error. In some instances, no glasses but *prismatic ones* will benefit them.

A high-couraged horse feels the will, as well as the support, of his driver through the reins by means of the bit. Although his course and rate of speed are changed from time to time at the will of the driver, the reins are never slackened. The horse becomes acquainted with the desires of his master by a sense of increased or diminished tension upon the reins. He is guided to either side by a difference in the tension of the two, although the driver does not entirely relax his hold upon the opposing rein while he uses the guiding one, and the difference in tension may be very slight.

So it is with the normal eye. It is both controlled and supported while performing its movements within the orbit by the eye muscles (which are its reins). The brain is the driver. At its command the eye revolves or remains stationary at any desired point. The tension of muscles, opposed to any movement of the eye required, is so modified by the brain as to insure the requisite support to the eyeball, and to steady it as it moves. Thus a perfect equipoise is constantly established between opposing forces, adjusted with the nicest care to meet the full requirements of the organ under all possible circumstances. The normal eye does not tremble or wabble when it moves or the attempt is made to hold it in any fixed attitude. It is a piece of machinery, perfect in all its parts, reliable in its movements, perfectly controlled by its master.

The eye with "muscular insufficiency" is like a horse with an inexperienced and incompetent driver; the proper tension upon the reins is not maintained at all times, as it should be; there is no equilibrium between antagonistic muscles; fixed attitudes are maintained with difficulty for any length of time; the brain becomes more or less disturbed by its inability to properly control the eye movements, and exhausted by the continual strain imposed upon it by the efforts required to do so even imperfectly.

Asthenopic subjects are *very frequently encountered* in the practice of a neurologist. The oculist, perhaps, sees them still oftener, because they are generally conscious that something is wrong with their eyes. Still, there are exceptions to this rule. I have examined patients who showed, in response to appropriate tests, very high degrees of muscular "insufficiency," that came to me for the relief of symptoms which had never been referred by themselves or their physician to any possible eye defect. I recall the case of an epileptic who was placed under my charge.

His family assured me he had "wonderful eyes;" and they were surprised when I examined them with care. The results of this examination showed, however, that twenty-five degrees of external insufficiency existed (as measured by the vertical diplopia test), and that he was hypermetropic and astigmatic to a marked degree.

Insufficiency of ocular muscles seems to me to be a *congenital defect* in most cases—possibly in all. It is encountered in very young subjects. It is not a paralysis or a true paresis. It is not uncommon to note wide variations in the same case, if examinations are made from time to time. Possibly this fact helps to explain why competent observers do not always estimate the degree of insufficiency in a given case alike, even when similar tests are employed and equal care is given to the case. *We have no way as yet of determining "latent" insufficiency,** as we

*Because this term was used by me, in a prior publication, I have received several communications from oculists of prominence denying the existence of "latent" or hidden insufficiency, and taking me to task for the use of such an expression. I would state, therefore, that there are, to my mind, most positive evidences that the condition thus described does exist in some cases; in fact, I would go so far as to assert that it is the rule, rather than the exception, to find a certain amount of masked insufficiency, in connection with "functional" nervous maladies, that cannot be elicited by any means yet known to the science of optics.

My experience in relieving ocular "insufficiencies" by tenotomy of the recti muscles has shown me that the amount of tissue divided is almost always *greatly in excess of the apparent defect* to be overcome. Again, after the eyes have been *perfectly balanced* by a tenotomy (as shown by careful tests made after the operation), it is very frequently found that more "insufficiency" develops within a short time than was detected before surgical interference was resorted to as a step for its correction. In the third place, I have found that repeated tenotomies (performed as often as indicated by the tests described) *eventually bring the patient to a point where the eyes remain permanently corrected*—a fact that proves quite conclusively the error of supposing that the tenotomy was in any way responsible for the lack of equilibrium which developed later. In the fourth place I have found it to be advisable in some cases to cut the muscles to excess, so as to *over-correct an error in equilibrium*—knowing that by so doing I anticipate a certain amount of "latent" insufficiency, which will assist in making the results more satisfactory to the patient within twenty-four or forty-eight hours.

If it were necessary, in my opinion, to argue this question at greater length, I might add (1) that a persistent wearing of prisms for the correction of insufficiency almost invariably results in the development of a latent weakness of the muscles not discovered at first; (2) that persistent daily exercise of the eyes by prisms usually accomplishes the same result; and (3), that my views are in accord with all who have had much experience in the use of prisms—irrespective of partial tenotomies, which demonstrate the facts even more satisfactorily.

I take the liberty of quoting from the late work of Prof. H. D. Noyes (pp. 87 and 89) the following passages:—

"Give due opportunity for disclosure, and what at first seemed to be a moderate degree (referring to insufficiency) may at length declare itself in much larger proportions."

"While great advantage is gained by Graefe's test, it is not true that *latent* insufficiency is always thus brought to view."

Again, I may quote from a late article by Dr. G. T. Stevens as follows: "Muscular anomalies of the orbit may be totally or partially latent."

do latent hypermetropia by atropine. Should a patient show us an insufficiency counteracted by a prism of a certain angle to-day, it only proves that he has *at least* that amount, not that he has no more. This statement can, I think, be demonstrated. It is an important fact to remember when the results of examinations of such patients made by yourself are at variance with the observations made by another.

Without further preparatory remarks, I pass to the consideration of the steps commonly taken to determine if the eye (regarded purely as a piece of mechanism) is perfect or imperfect. The study of the eye, when any of its component parts *become the seat of disease*, has no bearing upon the subject under discussion. This field is properly relegated to oculists.

THE TESTS OF VISION AND OCULAR MOVEMENTS.

The steps which should be employed in examining the eye for errors in refraction and accommodation, as well as those employed to detect defect in the power of ocular muscles, have not thus far been discussed. I expect to offer nothing new, but I hope to make the details of such an examination simple and within the comprehension of all.

The importance of this department of diagnosis can hardly be overestimated in nervous maladies. It has been my custom for three years past to examine the vision of nearly every patient sent to me, as my experience has shown me many times that remarkable cures may be made by the light thus shed upon the causation of obscure nervous symptoms.

Unfortunately for the sick, in many instances, physicians in general seem to think that the examination of the eye is too difficult a field for them to intrude upon without some special preparation for it. While this is undoubtedly true, in case the ophthalmoscope is to be employed, it is by no means a difficult matter for a person acquainted with physics to acquire a practical and satisfactory knowledge of the few tests here described in a comparatively short time, and with but a limited number of patients, provided that he works faithfully and intelligently. The healthy (?) as well as the sick can often be used to familiarize the beginner with the practical adjustment of prismatic, spherical, and cylindrical glasses, and also with the tests employed to detect "asthenopia" or anomalies of the eye-muscles.

Defective vision does not always produce ill health; hence among your friends or in your immediate family you may find a field for investigation and practice.

Now, in the first place, it is not necesssary to have a complete Nachet case of lenses. Such a case is very expensive. By selecting a limited assortment of lenses and prisms, different combinations can be made to meet the needs of almost every eye-defect encountered in medical practice.

There is furnished, with the various small cases designed by prominent oculists, a sheet of Snellen's test-types for distance, and also one containing several paragraphs printed in an assortment of types of various sizes to be used as a test for reading power. Each paragraph is numbered so that a record can be kept of the one read by the patient as a test. These test-type slips can be purchased separately, however, of any optician. It is best to have each *mounted on card-board*, and it is well to have the one used in testing for distance a double one with different letters on the opposed sides. If you suspect that the patient is *using his memory* during the tests employed rather than his sight, the board can then be exposed upon different sides at various periods of the examination.

You will find that the letters are mathematically made for testing distant vision. Above each line a numeral or Roman character is placed to designate the *number of feet at which the normal eye should read the line with ease.* Thus, the large letter on the top line will be designated usually by 200, or C C, while small letters of the lower line will be marked 10, or X. This shows that the top letter should be read easily at two hundred feet by the normal eye, and the lower line at ten feet. After you have provided yourself with a good trial-case, a set of prisms, and the necessary test type, let us see how you should proceed with an examination of a patient's vision. We may illustrate the steps by using one of the class as a patient.

I first hang upon the wall, as you see, the test-type card; and I place the patient with his eye on the same level and at a distance from it of exactly twenty feet. I then take the triple-grooved spectacle frame from the trial case and insert a plate of metal in the left rim of the frame, so that when it is used by the patient the left eye will be covered. I then place this frame upon the patient, and ask him to read aloud the letters on the testing card from the top downward, line by line. This act tests his vision in the right eye. I note (while he reads) the following facts: (1) If he *calls all the letters properly;* (2) if he *reads without apparent effort;* (3) at *what line he fails* to read. I then make a record as follows: O. D. (oculus dexter, or right eye) $V = \frac{20\ (\text{feet})}{\text{—}\ (\text{type})}$. The dash in the fraction is filled with the number which indicates the last line which the patient reads. When the vision is normal, the fraction will be as follows: $V. = \frac{20}{20}$ or $\frac{20}{xx}$. If the patient fails at the line next above the normal point the fraction would be expressed by $\frac{20}{30}$ or $\frac{20}{xxx}$. Remember that the *numerator* represents the distance (in feet) between the patient

and the test-type, and that the *denominator* represents the numeral on the test card placed above the last line of type read by the patient (which indicates the normal distance in feet at which it should be legible to the normal eye. Now, if the vision of the right eye is found to be defective, try and improve it, and, if possible, to render it normal, or as nearly so as possible, by testing the effects of concave or convex glasses upon it as the case seems to indicate, beginning with the weakest lenses and gradually increasing their strength until the vision reaches its highest acuteness. This takes some little practice and experience. If *convex glasses* are found to be indicated, note the *strongest* which gives the best vision to the patient; if *concave*, record the *weakest* glass that overcomes the defect.

In some cases you may find yourself unable to obtain normal vision in either eye by means of cylindrical or spherical glasses. I presuppose a certain degree of acquired facility on your part with glasses of the forms specified, and a carefully made effort to overcome the existing defect.

In such a case it is well to consult some expert oculist (if near at hand), and thus to ascertain the results of an *ophthalmoscopic examination.* The patient may have some mechanical impediment to vision, such as an opaque lens within the eye (cataract), or an opacity of the cornea; or he may have a high degree of astigmatism, which can often be estimated with some accuracy by the ophthalmoscope. Again, he may be found to be suffering from morbid changes within the optic nerve or the retina.

When it is found that a patient is so blind in an eye as to be unable to recognize any of the letters on the testing-card at any distance, you should note (before sending him to an oculist) if he can recognize with accuracy the *number of fingers* which you hold before the eye, and record the results of such investigation. You should make this test with the fingers in all possible positions in reference to the diseased eye (directly in front, above, below, and to either side of it).

We might record the results of an examination of a suppositious case up to this point as follows:—

$$\text{O. D. V.} = \frac{20}{\text{XXX}} \text{ (manifest) made } \frac{20}{\text{XX}} \text{ by } + 30 \text{ glass.}$$

The word "*manifest*" in this record means that the far-sightedness or "hypermetropia," which *apparently* exists, is overcome by a convex or (+) glass which focuses at thirty inches. After the use of atropine, any increase over this amount which may be developed is recorded as "*latent*" far-sightedness. I use here the old style of numbering glasses for the sake of perspicuity, although I personally prefer the metric system (dioptre), as it allows of more rapid combinations when the trial-case contains only a limited supply of lenses.

You will understand, when I exhibit the method of recording such observations more fully to you, why it is that the right and left eyes have to be separately examined and corrected (as already described) before the binocular vision is tested with and without the needed correction. I usually make upon the page of my own case-book a note relating to each eye of the patient, prior to the use of atropine somewhat as follows:—

O. D. (right eye) V. $= \frac{20}{xxx}$ (manifest) made $\frac{20}{xx}$ by + 30.

O. S. (left eye) V. $= \frac{20}{xl}$ made $\frac{20}{xx}$ by — 30

Binocular V. $= \frac{20}{xxx}$ made $\frac{20}{xx}$ by *this combination.*

Such a record of a suppositious case would show that the patient was *far-sighted* or "*hypermetropic*" in *the right eye*, and *near-sighted* or "*myopic*" in *the left eye*. It would lead me to believe also that the right eye (when under the influence of atropine) might show a still greater defect, which is now rendered "latent," or hidden, by an excessive development of the muscle of accommodation.

In all far-sighted eyes Nature tries from the date of birth to compensate for the congenital defect (an eye which is too flat) by a hypertrophy or *enlargement of the ciliary muscle* (see Fig. 39); hence, when this muscle is temporarily paralyzed by atropine, the true refractive condition of the eye is no longer masked. Far-sighted patients, therefore, lose their clearness of vision more or less at once when atropine is used. The normal or the "myopic" eye, on the contrary, is but little affected (as regards the outline of objects seen at twenty or more feet from the eye) by the use of atropine, although excessive light may annoy the eye in any case.

Let us now suppose that during the examination of a patient we first have examined each eye separately, carefully corrected all existing error found, and succeeded in getting $\frac{20}{xx}$, or normal vision, for each eye separately; that we have then tried both eyes together with the glasses best adapted for each, and found the patient able to read the normal type for distance without fatigue or conscious effort; and, finally, that we have made a careful record of each point noted during our observations. Are we now prepared to order glasses for the patient? Have we noted all that is important to note? To both of these inquiries I would say to the beginner, emphatically, "No." Several steps still remain to be taken, even before the use of atropine (which it is generally best to employ before a final decision is arrived at).

This brings us to the *tests for the detection of muscular anomalies in the orbit.*

Until within a comparatively few years the necessity of carefully measuring the power of adduction and of abduction of the eyes, and of determining the presence or absence of muscular insufficiency in "nervous" subjects, seems to have been practically disregarded even by oculists. Even to-day this defect (which probably is, as a rule, congenital) seems to be omitted from prominent mention among the enumerated list of ætiological factors of nervous symptoms by almost all authors of note. In some cases I have known it to be overlooked even by opthalmologists of world-wide reputation, simply on account of a careless and hasty examination for the defect. It is an extremely common defect of the eye; and may prove a very serious one to the patient. It is an important factor in many subjects afflicted with headache; it often exists to a high degree in epileptics; it is frequently found among children who suffer from chorea; it may unfit a patient for sewing, reading, attending places of amusement, or using the eyes in any way for any length of time. I have known it to cause vomiting and so-called periodical "bilious attacks" by exciting a reflex irritability of the central nervous system. One patient of mine (a close student) was completely cured of chronic dyspepsia by the use of prisms which corrected an insufficiency of 6° of the external recti muscles. He subsequently had tenotomy performed and now uses his eyes without fatigue. All bodily ailments have disappeared without the use of drugs.

In order to properly determine the condition of the ocular muscles, several tests have to be made. I do not personally regard *any of these alone as sufficient for diagnostic purposes.* The tests which I advise you to invariably employ are as follows:—

1. Direct the patient (as you see me do with a member of the class) to look fixedly with both eyes at some small object (say the end of a pencil), *and to follow it with the eyes as I move it* before the face of the patient at a distance of about ten inches. I watch both eyes carefully at the same time and note if a *tremulous movement* in either eye is present in any position of the eye as it moves about, and if the two eyes act in perfect unison with each other.

2. While the patient is instructed to *fixedly gaze* at the same object, I next *shield one eye* with a card or sheet of paper so as to exclude the object from view. Now I shift the card rapidly from one eye to the other, and I observe at the same time any deflection or trembling of the covered eye, which may show itself as I shift the card. If deflection or trembling occurs, it indicates a weak muscle.

3. Deviations of the visual axis of an eye in a vertical direction are not always revealed by the two tests previously mentioned, nor are they

always apparent to a careful observer of faces. They are of the greatest clinical importance, however, and should be looked for early in the examination. A pair of prisms of five or more degrees each are placed in a spectacle frame with their bases inward* in order to overcome the power of fusion of images by the externi, and the patient is directed to look through them at a candle flame placed twenty feet from the patient's eyes and on the same level. The head is placed in natural position for distant vision and steadied by a photographer's head-rest. If either of the two candle flames (seen by the patient in consequence of the prisms) be higher than the other, a prism is selected, which, with its base upward or downward, when placed before one eye overcomes the defect. The angle of this prism (in degrees) is then noted and recorded in the case-book.

4. I next place upon the patient a spectacle-frame previously arranged with a disc of ordinary glass, tinted red, to cover one eye, and a prism of 5°, with its base directed accurately upward or downward, before the other eye. I then direct the patient's vision upon a candle-flame at a distance of twenty feet. The prism causes *two candles to appear* (one being colored red by the glass of that hue), both of which to the normal eye should be seen as if *in a vertical line.* If the red image is seen to the same side of an imaginary vertical line dropped through the white image as the eye covered with the red glass, the external recti are insufficient; if the red image is seen on the opposed side of the vertical line, the internal recti muscles are weak.†

5. Any deviation of the candle which exists can be remedied easily by placing a prism with its *base outward* before one eye for external insufficiency, and with its *base inward* for internal insufficiency. The strongest correcting prism that can be worn without an over-correction marks the degree of the "manifest" insufficiency only;‡ hence we will

*Dr. Stevens had devised an rectangular and elongated form of glass for this purpose. Its great advantages must be apparent to all who have worked in this field.

† I have lately employed in my consultation room a device which seems to me to be of great assistance to patients while their eye-muscles are being tested. It consists of two pieces of white tape which are stretched upon a dark background at right angles to each other; so that one lies exactly vertical and the other horizontal. The flame from a small gas burner at the tip of a porcelain candle lies directly opposite to their point of intersection and between them and the eye of the patient, all of which should be on the same level.

During the tests described, the patient can tell at once if either line appears double as well as the image of the candle flame.

‡ My experience with tenotomy, as a means of producing an equilibrium between opposing forces in the orbit, has convinced me that the amount of insufficiency detected by prisms is but a *small proportion* of *what actually exists* in some cases. A persistent use of prismatic glasses will often develop a degree of insufficiency which the patient did not at first apparently possess. In my experience this is the rule rather than the exception. It is comparatively rare for me to encounter a case where a full correction of an existing insuffi-

note variations from time to time. I usually note both the weakest and the strongest prism which corrects the candle-deflection.

6. I next test and measure the *power of adduction and abduction* (convergence and divergence) of the eyes by means of prisms. To do this I set a lighted candle twenty feet from the patient on a level with his vision when seated. I then hold before one eye a prism, *with its base* directed *outward*, of sufficient angle to cause two images of the candle to appear when both eyes look at the object. I then instruct the patient to make an endeavor *to draw the images together* and to fuse the two into one image. This is the *test for adduction* or *convergence*. The normal eye should overcome a prism of at least 23° to 25°. It may overcome 60° in some instances.

In the same way a prism with its base directed inward is used to test the *power of abduction* or *divergence*. The external recti muscles should not fail to overcome a prism of at least 8°. By combining prisms of varying angles, one of the requisite angle can be easily obtained with but a few prisms in your trial-case.

The power of *abduction* and *adduction* should always be recorded when accurately determined. One fact should be stated, however, in this connection—viz., that several sittings are usually required before the patient learns to use his eye-muscles to the best advantage; hence the records of daily tests should be kept for purposes of comparison for a short time (when practicable to do so).

7. The power of convergence and divergence of the eyes can be *estimated for near objects* by means of a stereoscope modified by Professor Henry D. Noyes, into which prisms may be dropped at will. I have used it of late with some satisfaction. I find that the accommodation often modifies the power of ocular muscles (as determined by the previous test at twenty feet distance). Prisms vary, moreover, according to the glass used in their construction.

8. The power of fusing images of the test-object at twenty feet when a prism is placed with its base up or down before each eye is next determined. This is recorded as the "*sursumduction*" *test* for the right or left eye. This test should not be employed successively upon the two eyes without some minutes of rest have been given the patient. It aids us in determining the relative strength of the superior and inferior recti of the two eyes, and offers suggestions regarding the proper muscle to divide for the relief of vertical deviations of the visual axis (see test 3).

ciency of the ocular muscles by prismatic glasses insures a perfect equilibrium of the eyes for any great length of time. When we attempt to correct this peculiar muscular defect in the eye by weakening the stronger muscle (as I am constantly doing with brilliant results) the existence of "latent" insufficiency cannot, in my opinion, be doubted. Unfortunately for science, we have as yet no way of fully developing it, as we do "latent" hypermetropia by the aid of atropine.

9. It is well to exercise the muscles of the eye with prisms before the results of the diplopia tests are finally recorded. I have found that, after a flexibility of the eye muscles has been obtained by the aid of prisms, an insufficiency of the internal or external rectus muscle will sometimes manifest itself where it was not apparent at first. That this is not simply the result of fatigue seems proved by the fact that the insufficiency remains more or less apparent during subsequent examinations.*

I have lately adopted some new terms suggested by my friend Dr. George T. Stevens, in recording the results of my tests made to determine the condition of ocular muscles.

I quote the article referred to in full, because I deem it of great practical value. Dr. Stevens says:—

"The relations of the eyes to each other, in the act of vision, exercise important influences, not only in occasioning the condition known as asthenopia, but in the causation of many other important nervous disturbances.

"If this statement is admitted, it will be evident that the subject of irregularities in the actions of the ocular muscles must assume a greater importance than when disturbances of equilibrium were regarded as only occasional factors of asthenopia, and when these disturbances were looked for mainly in a single direction, in case they were not entirely disregarded. It is true, even at the present time, that 'insufficiency of the interni' is the only disturbance of the ocular muscles, excluding strabismus or some of the results of paralysis mentioned in the majority of the text-books upon the eye. Indeed, the importance of even this defect is hardly dwelt upon at any considerable length in many of these works, and it is not at all uncommon for the oculist to overlook the condition in his practical work.

"Defects which result in lasting difficulties and perplexities in the performance of binocular vision are not to be ignored; and the *rôle* of the ocular muscles in the causation of many nervous disturbances is undoubtedly of very considerable importance.

"As the investigator in this department of ophthalmology proceeds in his researches, or attempts to record his observations, he is met by the fact that the terms now in use are not only frequently inaccurate and misleading, but wholly inadequate to describe many of the states observed.

"To illustrate the two factors of the proposition just made, a few ordinary conditions may be adduced:—

"1. The expressions employed to designate the deviations from the state of physiological equilibrium are often incorrect and misleading.

"The term 'insufficiency of the interni' is used to express a state of the muscles of the eyes which is shown by the equilibrium test of Graefe at reading distance. In this test the images seen by the two eyes are separated by a prism held vertically, with its base exactly up or down before one of the eyes. If, under these circumstances, the images deviate laterally in directions opposite to the two eyes—that is, if the image of the right eye deviates to the left, and of the left eye to the right—there is said to be 'insufficiency of the interni' of as many degrees as equals the strength of a prism which, with its base toward the nose, will bring the two images in a vertical line. The expression, 'insufficiency of the interni,' in this relation, is used to indicate the fact that the internal recti muscles are 'insufficient' to counterbalance the external recti; and it also carries the idea that the externi are, in proportion to their physiological state, stronger than the

* This fact also confirms the views expressed in the previous foot-note (p. 138).

interni, or that the interni are, proportionally to the others, abnormally weak; tending thereby to balance the eyes outward, so as to cause an unusual and excessive demand upon the internal recti in close work.

"The fact that a great many cases, in which the equilibrium test of Graefe shows the conditions described, are really 'insufficiency of the externi' and not of the interni, must occur to any careful observer. Such a one will often find that, if he makes his test of equilibrium while the ocular muscles are in a comparative state of repose, as when looking at an object at a distance of six metres or more, he may find very marked 'insufficiency of the externi.' He may even observe that, if a screen is passed before one of the eyes while the other continues its gaze, at the distant object, the covered eye will deviate in a marked manner inward. If the screen is quickly changed to the opposite eye, he will see the lately covered eye move outward in order to fix the object. He may make various other tests which will demonstrate beyond a doubt that the real balance of the eyes is inward, and yet, when he makes the test of the dot and line of Graefe, or any similar test, at near point, he has marked 'insufficiency of the interni.'

"It is manifestly incorrect to say of such a muscular arrangement that the interni are 'insufficient,' and especially when by such a term it is generally understood that the outer are the stronger of the two opposing sets of muscles.

"Again, in certain cases of what is known as 'insufficiency of the interni,' one of the eyes actually deviates inward while the other deviates outward, while in a still greater number an apparent 'insufficiency of the interni' results from irregularities in the superior or inferior recti.

"Many other illustrations of the truth that this term as employed is misleading might be cited, but, without further expenditure of time or space, we may pass to the other factor of the proposition.

"2. The term 'insufficiency' is quite inadequate to express the conditions of deviation from the equilibrium as they may be observed.

"Graefe, as one of the great pioneers in modern ophthalmology, and as the greatest authority on the subject of muscular asthenopia, recognized some of these deviations, and not only regarded 'insufficiency of the interni' as a condition of notable importance, but wrote also of the 'insufficiency of the externi.'

"Notwithstanding his remarkable observations, much remained to be learned in this department of ophthalmology. While Graefe's great authority is to be fully recognized, the knowledge of these important conditions may yet be greatly extended.

"A class of deviations not at all uncommon, and one which induces great nervous perplexity, is that in which the tendency is for the visual line of one eye to deviate above that of the other.

"In examining some thousands of cases of 'insufficiencies' I have found a very important proportion of such tendencies. There is no term now in use which definitely expresses this condition. We cannot say that it is insufficiency of one or other superior or inferior rectus, for it is, in the great majority of cases, impossible to determine through what special influence the equilibrium is lost. We have not here, as in paralysis of the muscles, the definite guides of restricted motions by which we may determine the exact location of the trouble. Indeed, the defect may include an inclination on the part of the one eye to deviate upward, and on the part of the other to deviate downward. We might, perhaps, call such a condition 'insufficiency in a vertical direction,' with the right (or left) line of vision inclined to deviate upward.

"This would be a descriptive and somewhat extended expression. It would still be inaccurate, for it implies a weakness of some muscle, when the actual state may be an excess of tension on the part of some other muscle.

"Again, there may and often does exist a combination of faulty tendencies in more than one direction. The eyes may incline to deviate in both the vertical and the horizontal planes, the result of which will be a tending of the visual lines to deviate in an oblique manner. It must be apparent that the term 'insufficiency' is inadequate to express all these tendencies.

"There may be some propriety in using the expression 'insufficiency of the interni' in many cases, but in these just cited it would be impossible for us to speak of insufficiency of this or that oblique muscle without more accurate information than we are likely to possess. Indeed, in the majority of cases these muscles may not be influential factors in the condition described.

"Some term better adapted to express just what is intended, and nothing more, is needed. It is after much hesitation and doubt whether a suggestion involving the use of new terms in connection with a subject which has already engaged the attention of many able investigators might not be regarded as needless and presumptuous, that I have ventured to propose such an innovation. If, however, a change is to be made at any time in the classification and nomenclature of these defects, such change should be made before the literature becomes still more extended.

"The first need in a scientific classification of these muscular defects is the possession of such terms as, with proper modifications, shall justly express the conditions described.

"The terms which have already been employed are all unsatisfactory, and are not uniformly employed by different writers to describe precisely similar conditions. No terms now in common use occur to me as being in all respects desirable.

"We may, therefore, select some word which shall convey the general idea and which, with its proper modifications, will express our meaning with specific variations. Such a term should not, like the word 'insufficiency,' attempt to describe the exact nature of the muscular conditions, for this is often, if not generally, a subject of uncertainty. It should rather indicate the resultant facts as shown by the tendency of the visual lines to deviate from the physiological equilibrium. Nor should the term convey the idea of an actual turning, or deviation of one of the visual lines from what should be the common point of fixation. It should express a *tendency* to such deviation of such character that, should the force of the will be removed, this actual turning would result.

"The visual lines, in the conditions under consideration, are held in such relations to each other as to permit of more or less perfect binocular vision, but at an expense of a certain excess of nervous effort. In this we have the distinction between these conditions and those known as strabismus; for, while in these there is habitual binocular vision, in strabismus there is habitual diplopia, either conscious or unconscious. It is true that a fusion of images is possible in many cases of strabismus, and that slight diplopia may become to a certain degree a habit, in the conditions under consideration. Nevertheless, a condition of habitual diplopia should in general be regarded as distinguishing strabismus from these conditions.

"The Greek word *φόρος* (*a tendency*) seems to fulfill the conditions required, and accurately expresses our meaning in regard to this class of defects. With this for our central idea we may easily express every variety of tendency to deviation, as well as the absence of such tendency. Thus the two generic terms *orthophoria* (*ὀρθός*, right, *φόρος*, a tending) and *heterophoria* (*ἕτερος*, different) would express respectively a tendency straight forward and a tendency in some other direction.

"In order that these terms should possess precise signification, the relation of the visual lines to which they are applied should be determined under the uniform conditions which are here given.

"The eyes should be directed toward an object situated at a given distance from

them, and the head should be in the position known as the 'natural' or 'primary' position. The most convenient distance for the object is that at which tests for refraction are commonly made; that is, twenty feet, or six metres. This distance is, therefore, chosen as the standard for the determination of orthophoria and heterophoria. The best object for use in these determinations is a lighted candle against a dark background. It should be on a level with the eyes and at a distance of twenty feet. If ametropia exists, the eyes should be supplied with suitable correcting glasses. In the 'natural position' the body aud head are erect, the eyes are in the same horizontal plane, and the median line (a horizontal line at right angles with the line connecting the two eyes) is directed exactly toward the object. Under these circumstances there should be in orthophoria the minimum of muscular innervation.

"These conditions being observed, we may ascertain the existence of muscular equilibrium or its absence by means of prisms in the manner familiar to all oculists.

"The determination of the muscular conditions at near points will occupy our attention as we proceed. It is to be remembered that the results in such examinations are by no means absolute. Heterophoria may, like hypermetropia, be partly or entirely latent. Indeed, as in actual hypermetropia, we sometimes have apparent myopia, so with an actual inward tendency an apparent outward tendency may be observed.

"The different relations of the visual lines which may be now found may be defined and arranged as follows, a state of the most complete relaxation of muscular effort attainable being always supposed:—

"I. *Generic Terms.—Orthophoria:* A tending of the visual lines in parallelism. *Heterophoria:* A tending of these lines in some other way.

"II. *Specific Terms.*—Heterophoria may be divided into:—

"1. *Esophoria:* A tending of the visual lines inward.

"2. *Exophoria:* A tending of the lines outward.

"3. *Hyperphoria* (right or left): A tending of the right or left visual line in a direction above its fellow.

"This term does not imply that the line to which it is referred is too high, but that it is higher than the other, without indicating which may be at fault.

"III. *Compound Terms.*—Tendencies in oblique directions may be expressed as *hyperesophoria*, a tending upward and inward; or *hyperexophoria*, a tending upward and outward. The designation 'right' or 'left' must be applied to these terms.

"In recording the respective elements of such compound expressions I have employed the sign ⌞. For example, if it is desired to indicate that the right visual line tends above its fellow 3°, and that there is a tending inward of 4°, the facts are noted thus: *Right hyperesophoria*, 3° ⌞ 4°.

"In the absence of any means of producing a uniform state of relaxation of the long ocular muscles, such as we possess in atropine for the ciliary muscles, we must resort to every known device to ascertain as nearly as possible the true relations of the muscles. Methods other than that of measuring the deviation when diplopia is produced should, however, be regarded as auxiliary, and the record of ortho- or heterophoria should be made from the diplopia test.

"The powers of the different pairs of muscles to overcome prisms should next be determined. Some confusion has existed in the use of terms to express this power. Thus, the words *adduction* and *abduction* have been employed by Graefe and succeeding writers to express the power of the eyes to overcome respectively a prism with its base out or in. They have, however, been employed to express this power both when the object of fixation has been at a considerable distance. and when at the ordinary reading distance.

"The same words are also used to express the limits of excursion of the eyes outward or inward in the act of fixation.

"The words convergence and divergence have similarly been employed to express different classes of phenomena. As the words adduction and abduction are necessary to express the power of moving outward and inward of either eye singly, and as the terms convergence and divergence must in all cases imply the approach or the separation of the axis of the two eyes, whether in the act of overcoming a prism or otherwise there might be an advantage in employing the word *convergence* to indicate the highest degree of power of blending images at a distance of twenty feet when a prism with its base out is interposed; and the term *divergence* to indicate the limit of power to overcome a prism with its base in. This latter would also be less liable to objection for the reason that, while each eye is habitually directed in abduction and adduction, the two are rarely by voluntary effort caused to diverge except by the influence of a prism. The fact, however, that Graefe in his classic treatise on muscular asthenopia employed the words abduction and adduction to indicate the ability to overcome prisms must, beyond a doubt, determine the point, and these words should, therefore, represent the diverging and converging power with prisms. The standard of distance should, however, be uniform with that for the test for ortho- and heterophoria.

"It often happens that images can be united when a prism is placed before an eye with its base up or down, but that diplopia is produced if the prism is reversed, or if it is placed in the first position before the other eye. In other words, the tendency of one visual line being higher than the other, the power to blend images is greater when the prism is placed in one than when placed in the opposite direction.

"This condition is one of great importance, and no examination of muscular equilibrium should be regarded as complete in which its presence or absence is not determined. The ability to overcome a prism with its base down may be called *sursumduction*, and the eye before which the prism is placed is indicated by the word 'right' or 'left.'

"It remains to consider the relations of the muscles when the eyes are directed to objects at the usual reading distance.

"These relations may be uniform with those manifested at a distance, or they may vary in degree or in the direction of greatest apparent energy. To these conditions it might at first appear best to apply the familiar terms 'insufficiency of the interni' or 'externi.'

"The objections are that the terms have already been employed to express the relations of the eyes in accommodation and also in repose, and that only two of many conditions can be described.

"The relations of the visual lines in accommodation do not always depend upon the comparative strength or weakness of the opposing muscles, but upon a peculiar state of innervation of the muscles.

"The habit of maintaining an excessive tension upon the outer muscles in order to overcome esophoria frequently manifests itself in the near test as 'insufficiency of the interni.'

"These considerations render it desirable that a uniformity in the descriptive terms for the near and distant tests should be maintained. The terms already employed for distance may, therefore, be properly used if the modifying phrase 'in accommodation' is added. Thus we should have for insufficiency of the interni *exophoria in accommodation*, etc.

"The relations of the ocular muscles should, as Graefe has shown, occupy a prominent place in the record of all examinations of the eyes for asthenopia or kindred troubles.

"If the system of words here introduced at first appears to be superfluous, and, therefore, unnecessary, a careful consideration of the subject will be likely to convince a candid observer that new and more definite terms are needed to convey uniform meanings, and to express more conditions than are described by terms now in use. The terms here proposed

are explicit in meaning, and the system, by arranging the various deviating tendencies into classes, suggests to the examiner the conditions concerning which he should inform himself."

Thus far, then, in the examination, our record page in blank would stand as follows:—

Name..... Residence..... Date.....

V. { R. = $\frac{20}{\cdot\cdot}$ ..... corrected by ..... *glass.*
{ L. = $\frac{20}{\cdot\cdot}$ ..... " " ..... "

ASTIGMATISM ..... " " .....

Esophoria..... in accommod.....

Exophoria..... in accommod.....

Abduct..... Adduct.....

Hyperphoria, R..... L.....

Sursumduct, R..... L.....

Reading power at fourteen inches, corrected by ..... glasses.

VISUAL FIELD.	OPTHALMOSCOPE.
.....	....
.....	.....
.....	.....
.....	.....

All the data indicated for record in this table, excepting the estimation of the degree of astigmatism and the outline of the visual field, have been referred to, and the tests for each have been given with some detail.

The *estimation and correction of astigmatism* is a difficult matter for a novice, and sometimes for an expert. It will be better understood by reference to and close study of the standard text-books on ophthalmology. Moreover, the ophthalmoscope is often required to properly estimate the degree and kind of astigmatism which exists. I would say, in passing, that a *high degree of astigmatism should never be disregarded* or left uncorrected, especially if present in connection with abnormal nervous phenomena. It is a very common cause of headache and asthenopic symptoms.

In *estimating the visual field*, an instrument specially designed for that purpose (the *perimeter*) greatly simplifies the step, and gives us at the same time an accurate representation of its outline for subsequent reference. A drawing can be roughly made, however, of the visual field of any patient, by means of a blackboard and a piece of chalk, through a simple method described in most of the text-books. In some nervous cases it is very desirable that a register of the visual field be taken from time to time and preserved for reference.

Now, when we have carefully examined our patient respecting all the data indicated in the preceding table, are we safe in passing an opinion respecting the condition of the eyes? I would again say, "No."

We have now reached a point where we should *administer atropine* to the patient. I usually employ a solution of gr. iv of sulphate of atropine to an ounce of distilled water. This can be kept constantly in your office in a phial with a rubber-top dropper substituted in place of a cork. A drop or two in each eye will suffice in most subjects to dilate the pupil widely and to paralyze the power of accommodation of vision for near objects in about three hours. In occasional instances it becomes necessary to keep the patient under its influence for several days, but this is not the rule.

It is well to caution the patient, after using this drug, that he may possibly suffer from the sunlight, and that colored glasses will relieve him of this annoyance. It is also best to tell him that his vision may become very blurred for distant objects in case he is far-sighted; and that, in any case, he will *be unable to read or to write by the aid of vision without glasses* for several days. I have known hypermetropic patients to become greatly alarmed at the rapid loss of vision which has followed the use of atropine; all of which could easily have been avoided had they been prepared for it by timely words of explanation. It is always well to explain to far-sighted subjects the difference between "manifest" and "latent" hypermetropia, and to make them intelligent as regards the effect of atropine upon the "focusing" muscle before you administer it. If they are forced by their business to use their eyes for near-work while under the influence of atropine, a pair of cheap glasses may be given them for temporary use while under its influence.

I cannot impress too strongly upon you the necessity of using atropine upon a patient (if young) for diagnostic purposes when an error of refraction or of accommodation is suspected. Personally, I *do not regard an examination as complete without it.* It solves the question of the presence of "latent" hypermetropia—a very common defect and a very important one (from the standpoint of the neurologist) if allowed to go unrecognized. It reveals the existence of a previous ciliary spasm. It often arrests headache as if by a magic touch, and solves the nervous origin of many other similar symptoms.

Patients who boast of their acuteness of vision, and who apparently justify their statement by reading test-type at a distance without the aid of glasses, are often astonished and sometimes alarmed at the immediate loss of this power which is brought about by the use of atropine. This surprise is heightened when (by the use of proper lenses) their power of vision for distance is immediately restored, and they become conscious

for the first time of the muscular effort which they have been compelled in the past to exert in order to see without them. I shall never forget, personally, the sensation which I experienced of "seeing without effort" when a latent hypermetropia was discovered in my own eye, and corrected by glasses.

These experiences are well-known facts among oculists, but to the profession at large they often occasion as much of a surprise as to the patient.

I could point to case after case in my own practice where the cause of neuralgic attacks, excruciating headache, vomiting, extreme nervousness, and many other symptoms (not apparently connected with eye-defect) would have remained unrecognized if atropine had not been employed. There is a rule given by most oculists—viz., to give to a hypermetropic patient the *strongest convex glass** with which he can comfortably read the normal test-type (xx) at a distance of twenty feet. It is impossible in many cases to decide this fact without atropine or an ophthalmoscope. The former method is unquestionably the most accurate one, because the accommodation of the oculist, as well as that of the patient, has to be excluded in the latter; and it has the advantage, moreover, that it can be employed by the general practitioner as well as by the specialist.†

Now, after the patient returns to you with widely dilated pupils, you should *carefully repeat each step of the previous examination.* You should record the results of these tests and then compare them with those obtained before atropine was employed. If the eye is a normal one, the vision will be $\frac{20}{xx}$ after atropine has been used, as it was on the first examination; but, when an error of refraction or accommodation exists, changes of a greater or less degree may be noted. You may find, moreover, that the power of adduction and of abduction of the eye will be modified in some patients by the action of the drug upon the accommodation of vision, and that a different degree of muscular insufficiency may be detected. You can now decide intelligently as to the glass which is best adapted to restore vision for distant and near objects in each eye of the patient, and you are prepared to advise the patient respecting the use of the glasses

* The advisability of a full correction by glasses of existing hypermetropia can only be decided after the condition of the patient, his age, his susceptibility to reflex irritation from eye-strain, etc., have been carefully considered. It is not usually advisable to force a young subject to wear a glass which fully corrects the *latent* hypermetropia. I am in the habit of correcting all latent hypermetropia *in excess of one dioptre.*

† Personally, I have of late discarded the ophthalmoscope as a means of estimating errors in refraction, except in children and feeble-minded persons. It cannot always be relied upon, even in the hands of an expert, for this special object.

selected. You can decide also respecting the question of the utility of prisms or of tenotomy if the patient has marked insufficiency of the muscles. You can judge more accurately respecting the proper angle of the prism required in case their use is indicated. I would caution you, however, against deciding this latter point *before the error of refraction* (if such exists) *is corrected*, and not until the "diplopia tests" have been employed, after such lenses as are required to correct it have been placed before the patient's eyes. I have seen patients who gave evidence of marked insufficiency (5° to 8°), when the refractive error was uncorrected, exhibit no such defect when glasses which corrected that error were worn. Prisms in such a case would inflict injury upon the patient rather than afford relief.

In closing, I would remark that views which I have advanced respecting the dependence of abnormal nervous phenomena upon eye-defect are not new. They are in antagonism, however, to those of some authors, and have been more or less actively combated of late, especially in regard to eye-defect as a cause of chorea and epilepsy. I do not think the relationship between "eye-strain" and attacks of headache or neuralgia can be denied, although it is only hinted at by Anstie and is omitted by most authors who have written on the causes and cure of these distressing maladies. Some of our best neurologists, as well as most oculists, are now investigating with renewed interest not only the ametropic conditions of the eye, but also the eye with "insufficient" muscles. Facts are being daily substantiated beyond dispute which met with ridicule some years since. Every day, in my own experience, I am strongly impressed with the curative effects of glasses and partial tenotomies of the ocular muscles in various forms of functional nervous disturbances. In my opinion, the neurologist of to-day who fails to familiarize himself thoroughly with the examination of the eye omits an evident line of duty both to himself and his patients. No neurologist can send all of his cases to an oculist for an opinion, and, even if he could do so, he should at least be able to verify the opinion thus gained respecting the *refractive errors* found and the *state of the eye-muscles*. He requires a case of lenses and prisms in his office as much as an electrical outfit, and he should know how to use both—the one as an aid in diagnosis, and the other as a means of cure. Personally, I have come to regard the examination of any patient sent to me as incomplete until I have tested the state of refraction and accommodation, and examined with care the condition of the ocular muscles. This view has not been hastily formed, and my daily experiences confirm me in it. I believe the time will come when the tests employed in eye-examinations will rank in importance in neurology with the knee-jerk test, which for generations, as Gowers remarks, simply "amused school-boys."

The Ophthalmoscope.—In connection with the eye, it may be well to mention the instrument which is employed to detect abnormalities of that organ, viz., the ophthalmoscope.

All forms of this instrument consist (1) of a concave mirror which is perforated at its centre, in order that the observer may look directly into the illumined field; (2) a series of lenses by which it is possible to correct errors of refraction in the eye of the patient or observer; (3) a bi-convex lens, which brings the deeper parts of the eye into prominence, and enables the observer to inspect them minutely.

Hutchinson (as quoted by Hamilton) gives some concise and practical suggestions respecting the use of this instrument, which will bear repetition. He says:—

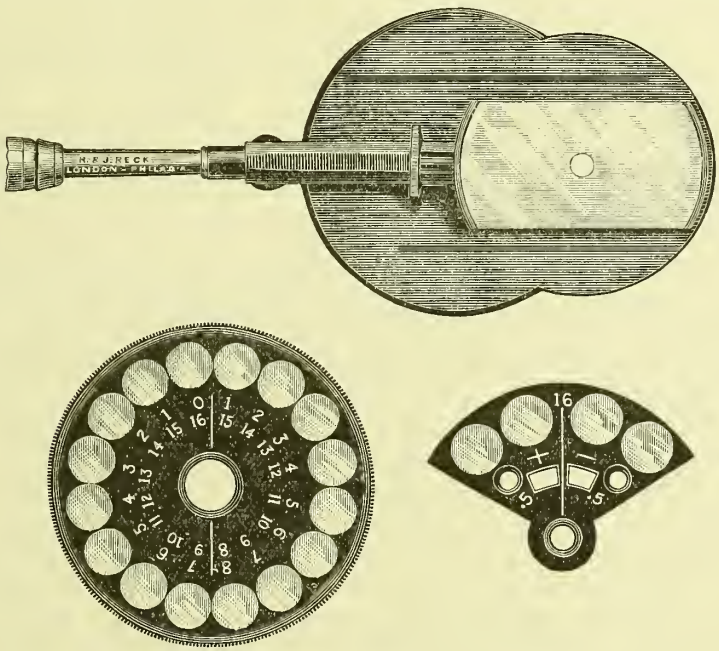


Fig. 40.—The Ophthalmoscope. (Loring's Pattern.)

"Having placed the patient's head in such a manner that the light (a lamp, candle, or gas-light) is on a level with his temple, and slightly behind it and his face, as a consequence, in shadow, the observer sits in front and applies the ophthalmoscope mirror to his own eye. He should keep both eyes open that he may see where the light falls, and then move the mirror until the light falls full upon the pupil of his patient. In a moment he will perceive the first fact which this instrument reveals, that the fundus is not black, as it has always appeared to be before, but that it is of a brilliant fire-red. He will, however, see nothing of the fundus distinctly, only a general reflex Now at this point the student must

stop awhile and use his mirror to inspect first the transparency of the cornea, and next, that of the lens and vitreous, and to do this he must make the patient move his eye in various directions. After a little practice he will be able to manage his light well, and to throw it with precision wherever he may wish, and to keep it steadily on any given part. At a first lesson, he may even with advantage practice for awhile by illuminating the second button of the patient's waistcoat. Tact in directing the light having been obtained, we may now proceed further. Instruct the patient to look, not full in your face, but over one shoulder; if you are inspecting his *right* eye, over your *left* shoulder. You will, when he does this, notice at once that the tint of the light reflected from the fundus is changed, that it is no longer fire-red, but canary yellow. The reason of this is, that a different part of the fundus is exposed to view, that, namely, of the optic disk itself, which is much lighter in color than the rest. The area of yellow is very large,—occupies, indeed, the whole of the field, while we know that the disk itself is very small. This proves that the objects thus distinctly seen are immensely magnified. Magnified by what? By the patient's own eye, which, as we have said, is equivalent to a lens of one-inch focus.

"Hitherto we have seen nothing distinctly, but if the observer now brings his head very close to the patient's face, he will be able with more or less facility to observe the details of the bottom of the eye, the trunks of the vessels of the retina, the optic disk, etc. What he sees is now equivalent to type looked at through a one-inch lens, placed exactly one inch in front of it."

In the ophthalmoscope now generally employed, a revolving disk containing a series of lenses is placed behind the mirror. These are intended for the purpose of correcting any error of refraction in the eye of the observer or patient. It is important that such error be determined first with accuracy and properly corrected, before the fundus is examined.

The *ophthalmoscope* is an important and valuable aid to those who are skilled in its use in detecting changes in the deep parts of the eye, chiefly those of the optic nerve and the vessel of the retina, by means of the sense of sight.

Dr. William C. Ayres has lately published in *The American Journal of the Medical Sciences* (1881) an exceedingly valuable and complete article upon this branch of diagnosis

By means of the ophthalmoscope the neurologist determines the presence or absence of a neuro-retinitis, or a "*choked disk*" as it is called, which is peculiarly suggestive of some brain lesion, that is creating a gradually increasing pressure within the cavity of the skull. Again, the *vessels of the retina* are derived from the same source as those of the brain; hence changes in the one are liable to be associated with similar changes in the other.

THE EYELIDS.—These may afford valuable aid in diagnosis. The upper lid sometimes drops over the eyeball and cannot be raised, constituting the condition termed "ptosis." This indicates a paralysis of the third cranial nerve. Again, when the facial nerve is paralyzed, the eyelids of the affected side cannot be closed. Puffiness of the lower eyelid, especially in the morning after rising, suggests the possibility of kidney disease. Alcoholic patients often exhibit a quiver of the muscular fibres of the eyelids. Spasm of the lids produces the peculiar winking so often seen in St. Vitus' dance and other nervous affections. In imbeciles and cretins the lids are often obliquely placed.

The expression of the eye is influenced to a large extent by the eyelids and may often be characteristic of certain nervous diseases. Melancholiacs exhibit the downcast eye. Maniacs may look excited, suspicious, or distrustful. A vacant stare is often present in dementia. Some forms of brain disease exhibit in the eye an air of exaltation. Masturbators seldom direct their gaze at the questioner, but look furtively about as if to avoid scrutiny.

THE MOUTH.—The lips are sometimes paralyzed. The pronunciation of the labials is then rendered indistinct or impossible, and a facial deformity is also created. The various diseases in which the mouth is affected may be considered separately with advantage.

A. In *Bell's paralysis* the lips are rendered incapable of movement on one side only and the mouth is drawn toward the opposite side by muscles which are no longer antagonized, on account of the facial paralysis. The act of whistling is rendered impossible, because "puckering" of the lips requires a contraction of the symmetrical muscles of the face. The saliva is no longer retained, and the patient "drools." All expressions except that of repose are those of a face alive on one side and dead and motionless on the other; hence, they would be particularly grotesque and striking (were it not so frightful and distressing) even to a casual observer.

In those rare cases where the facial nerve of both sides is impaired, symptoms similar to those mentioned above exist, except that the tongue has its normal capabilities of movement, save in the perfect articulation of the labial consonants only, and that a complete absence of facial expression is present.

Certain rules, which prove of value in making a diagnosis of the seat of the exciting cause of the condition have been given in the preceding chapter (p. 85). They are based entirely upon anatomical facts, and are therefore very important, because they admit of no exceptions:—

B. The lips and tongue are particularly affected also in that disease of the medulla called *Duchenne's disease* (glosso-labio-laryngeal paralysis). So marked is this loss of power, in severe cases, that a most

characteristic facial deformity is induced. As this disease is commonly bilateral, the lips usually hang apart from each other and cannot be approximated. The tongue lies trembling and immovable in the floor of the mouth, if the paralysis be complete; but if paresis only exists, it can be imperfectly protruded with difficulty, and is tremblingly and slowly retracted. If the paralysis be unilateral, the healthy side of the tongue becomes full and prominent, in comparison with the affected side, when called into action. Speech and mastication are seriously embarrassed. The saliva is constantly expectorated, because swallowing is performed with extreme difficulty.

C. The facial muscles, as well as the tongue, exhibit a peculiar tremor in *paralytic dementia.* Small bundles of fibres composing parts

Fig. 41.—Bell's Paralysis. (After a Sketch from Life by the Author.)

of the tongue, or the delicate muscles of the face, are thrown into non-rhythmical contractions by emotion, or the performance of any voluntary movement, as when showing the tongue or teeth. These fibrillary tremors may sometimes exist even in the quiescent state of the muscles. The tongue occasionally exhibits coarser movements of a convulsive character. Late in the disease it may become atrophied or shriveled.

The effects of this form of tremor upon speech are aggravated by an imperfect coördination of the muscles of the tongue and lips, which is simultaneously developed. Long or difficult words are omitted in conversation by these patients in a half-unconscious way, and the terminal syllable of other words is commonly left off. The speech becomes thick, and of a tremulous character. The shortest words possible are employed

by the patient to convey his ideas. A distinct pronunciation of consonants and polysyllabic words, such as "constitution," "infallibility," "prognostication," etc., is impossible; hence, a test is thus afforded between carelessness of utterance and a physical inability to articulate.

An unnatural quietude of the muscles of the face and a slight disparity of the pupils are prominent features of its stage of development.

It is well to note, in this connection, a test which is of some value in deciding as to the existence of this special form of disease. Extend the patient's fingers and place them between your own, and a delicate, "parchment-like" fremitus will be felt, which is due to an otherwise imperceptible tremor of the hand muscles.

D. The *lips* participate to a marked degree in severe types of *facial spasm.* In the clonic form of the muscles on one side of the face, are violently contracted and as suddenly relaxed. The eye is commonly affected simultaneously with the angle of the mouth. The spasms are marked by distinct paroxysms, whose duration varies from a few seconds to an hour or so. If the spasm is of atonic variety, mastication and articulation are interfered with, and the paroxysms are of longer duration.

It is always well to search carefully for carious teeth in these cases; but the spasms may be due to cold, wounds, injuries to the trigeminal nerve, or chorea.

E. The lips may indicate some form of *defect in the heart's action* if blue or purple in color.

Scars at the corners of the mouth are strongly suggestive of previous syphilitic ulceration, a point of importance in the treatment of some forms of nervous disease.

F. The *gums* should always be inspected. If pale, anæmia exists. If blue along the line of junction with the teeth, lead poisoning is present. If the teeth are loosened and the gums are soft and bleed easily, mercurial poisoning may be suspected; this is rendered positive if the breath has the "mercurial odor" and the saliva is excreted in very large quantities. Various cachexias, phosphorus poisoning, purpura, and scurvy, produce marked and often characteristic changes in the gums.

G. The *teeth* may afford much valuable information respecting the possibility of hereditary syphilis. Hutchinson has described the characteristics of such teeth with accuracy and detail. It is impossible to quote his deductions here, but the peculiarities of syphilitic teeth are now generally well recognized, and are often a valuable aid to the neurologist, both in diagnosis and treatment.

H. The *tongue.* Some diagnostic points regarding the tongue have been touched upon already. When the face exhibits any form of paralysis, it should be always carefully noted if the tongue exhibits fibrillary tremors; also whether it can be protruded in a straight line and

moved freely in all possible directions. In testing speech those words should be employed that require the normal power of movement of the lips (the labials) and of the tongue (chiefly the consonants). It should be also noted whether the words are clearly, rapidly, and distinctly articulated, or if the utterance of words is slow, thick, or slurred.

Ragged edges in the tongue indicate epilepsy, because it is frequently bitten during the paroxysms.

Imperfect mastication of food and difficulty in swallowing may be due to loss of power in the tongue.

A "furred condition" of one lateral half of the tongue indicates some irritation of the branches of the fifth cranial nerve; hence, the presence of decayed teeth, diseases of the gums, or the maxillary bones, etc., should be carefully searched for. The tongue may be paralyzed on one side or on both. This condition is not infrequently due to hemorrhage, softening, or tumors of the brain, and it occurs in connection with embolism or the general paralysis of the insane.

FIG. 42.—SYPHILITIC TEETH. (Hutchinson.)

Spasm of the tongue may be perceived in connection with chorea, epilepsy, hysteria, facial spasm, and as a result of slight compression or irritation of the hypoglossal nerve. Fibrillary tremors of the tongue are often encountered in patients afflicted with paralytic dementia.

I. The *soft palate* may be implicated when the facial nerve is injured above the origin of the petrosal nerves. It is important to examine the palate, therefore, whenever Bell's paralysis exists, since the seat of the exciting cause may be decided by it. It may participate also in atrophic changes.

THE EAR.—Deposits of urate of soda are often found in the ear in gouty subjects. These may cause ulceration. Again, the lobe of the ear may become swollen, red, and glistening as an attack of gout is being developed.

Among insane patients, the ear frequently becomes deformed from œthæmatoma.

Persistent discharge of pus from the ear indicates disease within the temporal bone. Fatal brain complications may arise from an extension of such ear troubles to the coverings of that organ.

THE FACE AS A WHOLE.—Among the diseases of the nervous system, there are certain types of physiognomy which are so characteristic as to be of the most positive value in diagnosis.

In attacks of *epilepsy* the neck at first becomes twisted, the chin raised and brought round by a series of jerks toward one shoulder. The features are greatly distorted. The brow is knit; the eyes are sometimes fixed and staring, at other times rolling about in the orbit, and again turned up beneath the eyelid, so that the cornea is covered, and only the white sclerotic is to be seen; the mouth is twisted to one side and distorted; the tongue is thrust between the teeth, and, caught by the violent closure of the jaws, is bitten, often severely; and the foam which issues from the mouth is reddened with blood. The turgescence of the face indicates obstruction of the venous circulation; the cheeks become purplish and livid, and the veins of the neck are visibly distended.

During the fit of exacerbation, in an attack of *tetanus* or lockjaw, the aspect of the sufferer is sometimes frightful. The forehead is corrugated and the brow knit, thus expressing the most severe type of bodily suffering; the orbicularis muscle of the eye is rigid, and the eye itself staring and motionless; the nostril is widely dilated, indicating the extreme distress of breathing; the corners of the mouth are drawn back, exposing the teeth, which are firmly clinched together; and the features, as a whole, have a fixed and ghastly grin—the so-called "risus sardonicus." During such paroxysms as in those of epilepsy, the tongue is liable to become protruded between the teeth and to be severely bitten.

In *chorea* the facial muscles participate in the general eccentricity of movement. Watson thus describes the peculiarities of this strange affection: "The voluntary muscles are moved in that capricious and fantastic way in which we might fancy they would be moved if some invisible mischievous being, some Puck or Robin Goodfellow, were behind the patient and prompted the discordant gestures. With all this the articulation is impeded; there is the same perverse interference with the muscles concerned in the utterance of the voice. By a strong figure of speech the disorder might be called 'insanity of the muscles.'"

In *catalepsy* the patient lies often with eyes open and staring, yet without expression indicative of life; more like a wax figure or a corpse than like a living subject. The features may be made to assume any expression, no matter how absurd, as the tissues have their normal pliability, and they will remain so placed until again mechanically altered. This same peculiarity is also present in the muscles of the extremities, and

forms one of the distinguishing tests of the disease. The mental faculties are in abeyance, and all power of voluntary motion is lost. The sensibility of the body seems also to be lost.

In *hystero-epilepsy* the contortions of the face and body are often frightful. In rare cases consciousness may be retained throughout the attack.

The *deformities of face* and intellect which seem to be the result of residence in special atmospheric conditions, or of certain well-defined localities, are illustrated in that race of people found in Valais and the adjoining cantons of Switzerland, called "cretins." Many of these wretches are incapable, according to Watson, of articulate speech; some are blind, some are deaf, and some suffer from all of these privations. They are mostly dwarfish in stature, with large heads; wide, vacant features; goggle eyes; short, crooked limbs, and swollen bellies. The worst of them are insensible to the decencies of nature, and in no class of mortals is the impress of humanity so pitiably defaced. They are usually the descendants of parents afflicted with goitre.

Fig. 43.—The Hand in Progressive Muscular Atrophy.

The Hand.—Among certain forms of nervous disease characteristic deformities of the hand are sometimes encountered. These will be considered separately. Tremor and spasm of the fingers are also developed, in some cases, and require a hasty description.

The *deformities of the hand* that are commonly observed include (1) that of so-called "progressive muscular atrophy;" (2) that of a spinal disease known as "amyotrophic lateral spinal sclerosis;" (3) that of injury of the ulnar nerve; (4) that of injury of the musculo-spinal nerve; (5) that of injury of the median nerve; (6) that of paralysis agitans; and (7) that of gout and rheumatism.

The *tremors of the hand* include (1) those of a condition called "athetosis;" (2) those of chorea or St. Vitus' dance; (3) those of paralysis agitans; (4) those of the general paralysis of the insane; and (5) those produced by circumscribed lesions of the nerve centres.

In *progressive muscular atrophy* the ball of the thumb is often the starting-point of the disease. This eminence gradually shrinks and disappears. It should be remembered that the disease affects symmetrical and homologous parts; hence both hands are liable to present the same deformity. Gradually the muscles between the bones of the hand shrink, so that the bones stand out unnaturally. Fibrillary twitchings over the affected muscles should always be looked for, as they are seldom absent. The temperature is lowered over the atrophied muscles.

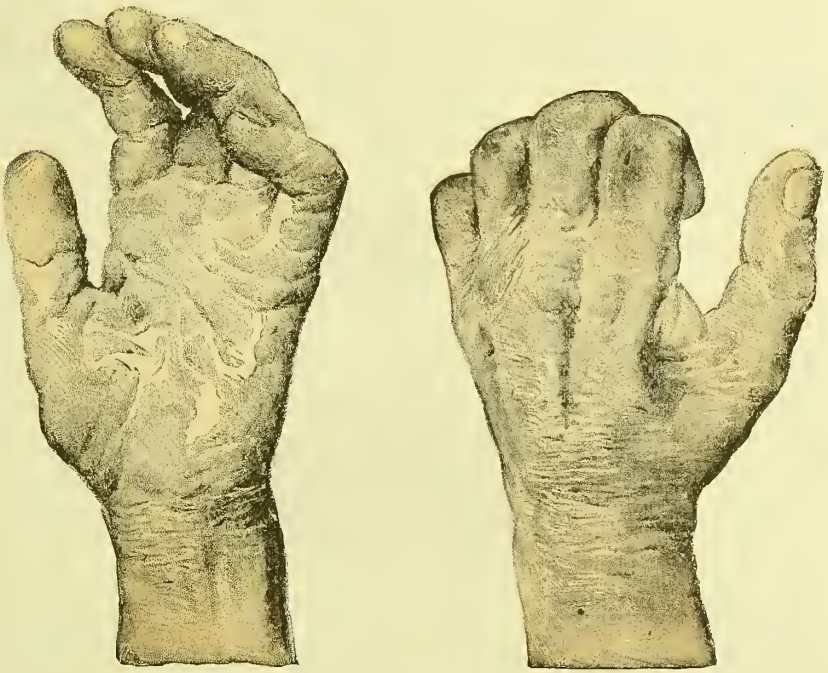


FIG. 44.—ULNAR PARALYSIS. (After Bramwell.)

In *amyotrophic lateral spinal sclerosis* the hand, when affected, is strongly flexed upon the forearm, the fingers are shut tightly upon the palm, and the thumb is drawn inward toward the fingers. Attempts to straighten the fingers, thumb, or hand, will be strongly resisted, and cause pain. In course of time the affected muscles become markedly shrunken, thus adding to the deformity described. Fibrillary twitchings may be easily excited in the affected muscles, provided they do not spontaneously exist.

When paralysis of the *ulnar nerve* exists, adduction of the hand is no longer performed in a perfect manner, since the flexor carpi ulnaris can no longer act in unison with the extensor carpi ulnaris. Flexion

of the hand is performed imperfectly, and by means of the flexor of the radial side of the forearm only, since that muscle is supplied by the median nerve. The ability to move the little finger is almost entirely abolished. Complete flexion of the inner three fingers is rendered difficult and sometimes impossible. The fingers cannot be separated from each other, or compressed into a close lateral juxtaposition, owing to paralysis of the interossei muscles; and flexion of the first phalanx and extension of the two terminal phalanges of all the fingers are rendered impossible, for the same reason.

When the ulnar nerve is paralyzed above the wrist, so that the interossei and lumbricales are alone paralyzed, the hand assumes a diagnostic attitude, the so-called "claw-hand," in which the extensor communis digitorum muscle extends the first phalanges of all of the fingers,

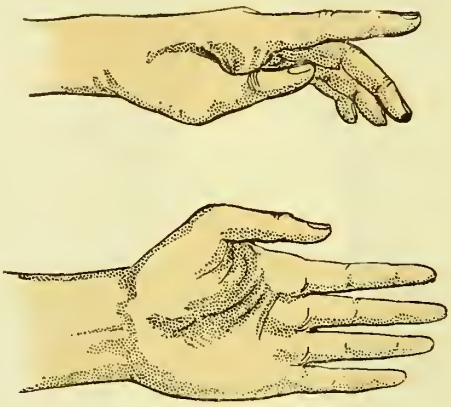


Fig. 45.—Median Paralysis.

while the other two rows of phalanges are flexed by the common flexor muscles of the fingers (the interossei and lumbricales being no longer able to flex the first row of phalanges, or to extend the two other rows). This same condition of the hand may, however, be produced by a condition of progressive muscular atrophy of these muscles.

It must be remembered that this condition, if dependent upon ulnar paralysis alone, is more marked in the two inner fingers than in the three outer, since the lumbricales are supplied in part by the median nerve. This clinical fact seems to stamp the action of the lumbricales as similar to that of the interossei. Finally, the effects of ulnar paralysis may be manifested in the movements of the thumb, since it supplies two muscles which control it. This will be most apparent when the patient is requested to press the thumb forcibly against the metacarpal bone of the index finger, or to adduct the thumb.

When paralyiss of the *median nerve* exists, the seeond phalanges of all the fingers, and the third phalanges of the index and middle fingers cannot be flexed, and the thumb cannot be flexed or brought into eontact with the little finger. On the other hand, flexion of the first phalanx, with extension of the other two, can be performed in all the fingers by the aid of the interossei, which are supplied by the ulnar nerve. The position of the thumb is peculiar; it is extended and adducted and thus elosely applied to the index finger, as in the hand of the ape. The hand, when flexion at the wrist is attempted, is strongly adducted by the action of the flexor earpi ulnaris, because the antagonistie musele of the radial side is paralyzed. The act of phonation of the hand is seriously impaired. The inner three fingers can be brought into a partially flexed condition, since the flexor profundus digitorum musele is partly supplied by the ulnar nerve. These combined effeets give to the hand and fingers,

Fig. 46.—Musculo-spiral Paralysis.

and especially to the thumb, a position so peeuliar that paralysis of the median could hardly be mistaken by an anatomist for any other deformity. when the paralyzed museles begin to show the results of atrophy, the deformity in the forearm and in the ball of the thumb will further assist in the diagnosis of this affeetion.

The *musculo-spiral nerve* is more frequently affeeted with paralysis than any of the nerves of the upper extremity. It is particularly liable to both peripheral and eentral causes of paralysis; thus in the eerebral hemiplegia, the museles supplied by this nerve are, perhaps, more commonly affeeted than those supplied by any other nerve, while paralysis of these museles is eommon as the result of ehilling of the upper extremity, traumatism, and lead-poisoning.

The anatomieal situation of the musculo-spiral nerve and the peeuliarity of its eourse around the humerus, probably explain the frequent oeeurrenee of paralysis, sinee it may be easily eompressed by sleeping upon the arm. It is eommon to meet with this type of paralysis in

patients who have used their arms as a pillow, or in drunkards who have slept in some constrained position upon benches, steps, etc. Persons who have fallen exhausted and have rested upon the arm, and soldiers who have slept upon the damp ground, often arise with this form of paralysis. It is stated by Brenner that the coachmen of Russia, who are in the habit of sleeping upon the box with the reins wound around the upper arm, are victims to this condition; and Bachon reports the same result as common among the water-carriers of Rennes, since they pass their arm through the handle of the heavy water-pails to more securely compress them against the chest. The habit of the Russians of tightly bandaging the arms of infants to the body, and allowing them to sleep upon one side for long intervals, seems to promote the frequent occurrence of this trouble.

Among the other forms of traumatism which conduce toward this form of paralysis may be mentioned the use of poorly-padded crutches, the kicks of animals, cuts, stab-wounds, fractures of the humerus, dislocation of the humerus at the shoulder-joint, and the development of an excessive amount of callus after a fracture.

Rheumatic affections and a neuritis of the musculo-spiral nerve are reported as causes by Bernhardt and others; and cases of hysterical origin have been rarely but positively authenticated.

Finally, lead-poisoning must be mentioned as one of the most common causes of paralysis of the muscles supplied by the musculo-spiral nerve. The existence of this form of poisoning will have generally been indicated, previous to the appearance of paralysis, by colic, jaundice, and arthralgia, as the muscles are seldom affected until the latter stages. The extensor communis digitorum muscle is usually affected first, and the paralysis gradually extends to the other muscles supplied by the musculo-spiral nerve. The muscles of the arm are much less frequently affected than those of the hand and forearm; but in severe cases the muscles of the upper arm are involved, and the thumb and the index-finger cannot be extended or abducted; the patient cannot supinate the hand when the forearm is extended (this position being assumed in order to exclude the action of the biceps muscle), nor can the forearm be half bent and the hand half supinated by the supinator longus muscle; and, finally, when the patient is instructed to flex the forearm, when placed in a position of half flexion and semi-pronation, the supinator longus muscle lies flaccid, and does not become tense and hard as in health. The loss of power in the triceps muscle renders it impossible for the patient to extend the forearm upon the arm when the arm is first raised above the head; nor can the forearm be extended with the same degree of force as the healthy side in any position of the arm. When the hand is laid upon the table, the patient is unable to raise the hand from contact with it, but the lateral movements of the fingers can be performed as in health.

In *gout* the joints become enlarged and seriously crippled by deposits of urate of soda, that cause prominent nodules upon the fingers. These often ulcerate. The index and middle fingers are the ones most frequently deformed. Occasionally one finger will be drawn toward the palm by gouty inflammation in the sheath of its flexor tendon. If this deformity be found, always examine the other hand to see if a similar deformity is not more or less developed on both sides. If so, it is almost a positive sign of gout.

The diseased condition, called "*athetosis*" because the fingers do not maintain a fixed position, is characterized by a continual motion of the fingers and toes, and an inability on the part of the patient to retain them in any fixed attitude. These patients cannot keep the hand closed or open, even for a short period, although the fingers are to some extent

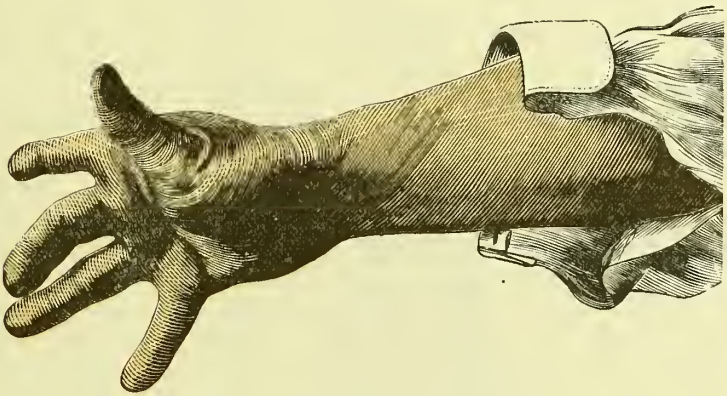


FIG. 47.—ATHETOSIS. (After Hammond.)

under the control of the will. The toes are not commonly affected to the same degree as the fingers. The movements of the fingers and toes are perpetual, not being entirely arrested during the hours of broken sleep.

The tremor of *paralysis agitans*, or "shaking palsy," is markedly aggravated by voluntary muscular effort or mental excitement. Except in very aggravated cases, it ceases during sleep. During the daytime it is more or less persistent and uncontrollable.

In *imbeciles*, rhythmic movements of the hands are commonly met with. They are in marked contrast to the irregular and spasmodic movements observed in St. Vitus' dance.

During an attack of *acute hydrocephalus*, or "water on the brain," the thumbs are usually flexed upon the palm.

Langdon Down has described the so-called "*woolly hand*" of the idiot; the skin being too abundant for its size, and forming wrinkles upon it.

In all diseases which *cut off the nerve fibres from their centres of nutrition*, or "trophic centres" as they are called, or after injuries to the nerves, the skin of the hand, as well as of other parts, may become smooth, shining, and affected with eruptions or ulceration. The nails and hair may also give evidences of imperfect nutrition.

THE GAIT AND ATTITUDE OF THE PATIENT AS A FACTOR IN DIAGNOSIS.

Among the symptoms which are brought to the notice of the neurologist by his perceptive faculties, none are more positively diagnostic than the abnormalities of gait and attitude which are frequently encountered. It will simplify description to consider first the more common abnormalities of gait, and subsequently the characteristic attitudes produced by nervous affections.

Gait of Hemiplegia.—This condition (in which one lateral half of the body is paralyzed) is evidenced by a characteristic gait, if the paralysis is not so profound as to prevent all attempts at walking. The arm hangs at first limp, and, in some cases (later on), more or less rigid on the affected side. The shoulder droops upon the paralyzed side. At each step the paralyzed half of the body is lifted; in order, as it were, to swing the weak leg forward. This movement causes the shoulder to tilt toward the healthy side, and the pelvis to be raised; while at the same time, the leg is not bent at the knee as in health. The shoe of the paralyzed leg trails along the ground as it is swung forward, and the toe and outer part of the sole of the shoe becomes worn off rapidly, a clinical point not to be overlooked. The back is not arched, as in the spastic form of paralysis, and the feet do not tend to cross the median line. The term "sickle-walk" is applied by French authors to this variety of gait, on account of the swinging, semi-circular movement of the paralyzed foot. The patient usually carries a cane on the healthy side, to aid him in walking.

Gait of Paraplegia.—Both legs (or, to be more accurate, the lower half of the body) may be more or less paralyzed, and yet the patient can walk. How different is the gait, however, from that of health! These patients shuffle along without raising either foot from the ground to any appreciable extent, so that they cannot be said to step. The progression is extremely slow, because the length of the step (if it may be so called) is very short. The heel of one foot rarely passes the limit of the toe of its fellow, if the paraplegia is well developed. This gait differs from that of spastic paraplegia chiefly in the absence of the stiffness of the legs and the interlocking of the knees, which are both present in the other. The so-called "hopping gait" is not developed as in the other form. The back is not arched.

Gait of Spastic or Tetanoid Paraplegia.—In the early stages of this disease a combination of paresis, muscular rigidity, and occasional tremor exists. The feet are turned inward and appear to be firmly glued to the ground during attempts at walking, and are scraped along with a characteristic noise. They often cross each other in walking, and the knees are liable to become locked together. These subjects are particularly prone to fall in spite of the use of canes or crutches, because the

Fig. 48.—Paralysis Agitans, or Shaking Palsy. (Charcot.)

slightest irregularity in the pavement may catch their shoe as it is slid along the ground. These patients sometimes exhibit a "hopping gait" when the muscles of the calf become affected with spasm. The back is strongly arched and the chest is thrown forward. The patient throws his weight first on one cane and then on the other, in order to lift his body so as to move his feet.

Gait of Paralysis Agitans.—The tottering and trembling gait of these subjects, with a tendency to trot rather than walk when under full

headway, is characteristic. The shaking hands are usually held out in front of the body, which is bent forward as they run. These subjects are generally well advanced in age. The head is projected forward and held stiffly when walking, and the "vertebra prominens" stands out in bold relief. Fig. 48 illustrates this point very well.

Gait of Pseudo-Hypertrophic Paralysis.—These subjects are always children. The immense calf muscles are strangely in contrast with their paralytic symptoms. When they attempt to walk the gait has been aptly compared to the "waddling of a duck." The back is excessively curved

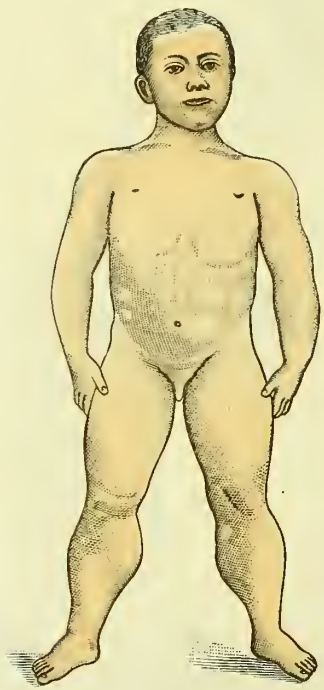


Fig. 49.—Attitude of Pseudo-Hypertrophic Paralysis. (Duchenne.)

in the erect posture, so that a line dropped from the shoulders falls behind the hips. The peculiarities of attitude of these patients will be considered later.

Gait of Locomotor Ataxia or Tabes Dorsalis.—These subjects straddle as they walk. The legs are flung about in an uncertain and apparently aimless manner, although the steps are taken with marked deliberation. The feet are brought down with the heel projecting, thus creating a "stamping" and "flopping" gait. These patients keep their eyes steadily upon the ground when walking. They have no motor paral-

ysis, as is shown by testing the various muscles separately; the abnormal gait being due purely to an inability to properly coördinate the various groups of muscles. These patients are frequently subjected to severe and unexpected falls after marked incoördination of movement is developed, and generally resort to the use of strong canes when walking.

Gait of Hysterical Palsy.—The feet are dragged or shuffled along in the paraplegia of hysteria, but one foot is usually more affected than its fellow. There is no "sickle-movement" of the leg, as in hemiplegic subjects. These subjects use a cane or crutch, or cling to articles of furniture as they sluggishly move about a room. It is liable to pass away suddenly and is usually developed as suddenly.

Gait of Progressive Muscular Atrophy.—When the thigh and calf muscles are affected, or those of the back or abdomen, the gait is seriously altered. As a rule, these subjects walk as a sailor does upon land, only the "roll" is exaggerated, and the trunk is peculiarly poised upon the legs. The gait is, however, modified by the seat and extent of the muscular degeneration, as it is produced in each case by the inability of a certain set of muscles to perform their normal functions.

Gait of Cerebellar Disease.—Like all ataxic subjects, these patients stand with their feet wide apart, to increase their base of support. When walking they give evidence of imperfect coördination of the muscles of the legs. Sometimes they stagger and reel like an intoxicated person. If the feet are exposed the toes will be seen to be in constant motion when an effort to stand on one spot is made, as if they were endeavoring to bury themselves in the carpet.

Gait of Reflex Paralysis.—This is generally of the "hemiplegic" variety. One leg is dragged along behind the other, in the large majority of cases.

Gait of Cerebro-Spinal Sclerosis.—In this disease we meet a very characteristic gait. Slight jerking movements of the head and neck can be perceived in the early stages. Later in the disease the symptoms of marked incoördination of the muscles are apparent. The gait is then extremely unsteady and irregular, but totally unlike that of locomotor ataxia, in that the muscles of the trunk as well as those of the legs are affected. These patients do not walk deliberately and in a straight line, but shoot suddenly forward or to one side, and are very apt to knock against articles of furniture in moving about a room and to fall violently.

THE ATTITUDES ASSUMED IN THE MORE SEVERE FORMS OF NERVOUS DISEASES.

Nervous diseases tend in some instances to produce abnormalities of attitude. The limits of this chapter will preclude more than a cursory view of this field. A volume would be required to properly exhaust the headings already touched upon.

Among the more common causes of abnormalities of attitude due to nervous lesions may be mentioned epilepsy, tetanus, hydrophobia, spinal meningitis, hysteria, catalepsy, hydrocephalus, chorea, athetosis, arthropathy, the many forms of cerebral and spinal paralyses, the different types of tremor and muscular atrophy, contracture, or reflex spasm.

Of the characteristic attitudes some are observed only in the erect posture of the patient, and others when the patient moves about. Some are recognized when the patient is sitting, while again others are present in patients confined to bed. Attempts at movement of any kind sometimes increases the deformity, while again walking may gradually limber up other patients and render the defects of movement less apparent.

In connection with the description of facial evidences of nervous disease, the characteristic facial attitudes of Bell's palsy, Duchenne's disease, paresis of the muscles of the eye, paralysis of the third nerve, the convulsions of epilepsy and tetanus, the condition of catalepsy, and the facial spasm of St. Vitus' dance have been alluded to and in part described.

When the hand was considered, the attitudes of progressive muscular atrophy, paralysis agitans, and the results of paralysis of the median, ulnar, and musculo-spinal nerves were described separately. The deformities of the hand in gout, and the attitude of the thumb and fingers in hydrocephalus, athetosis, and imbecility, were also mentioned. It is not necessary, therefore, to again describe them.

It remains for me to touch upon some of the more important attitudes which have as yet been omitted.

In *acute hyprocephalus* the tuberculous deposit at the base of the brain creates a characteristic attitude when the condition is well developed. These children bore their head into the pillow and roll it from side to side. The thumbs are flexed upon the palms during sleep, even before the severity of the attack is reached. The pupils are at first contracted, but they become dilated when coma develops from the pressure of the deposit of tubercle upon the brain. The abdomen is markedly retracted.

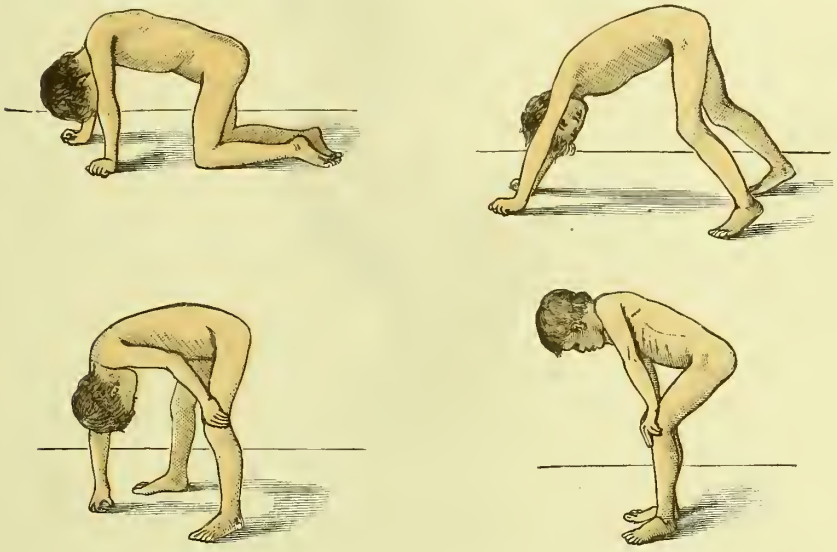
Linked with *cerebro-spinal meningitis* we notice the rigid contraction of the muscles of the trunk, resulting in a curvature of the back. The head is also thrown backward and the muscles of the neck are more or less rigid. Fever and an eruption are also present.

Sooner or later after a "*stroke*" *of paralysis*, a state of rigidity and contracture of the paralyzed muscles often develops. It causes, as a rule, a state of permanent flexion in the upper limbs and that of extension in the lower. This post-paralytic contracture, if developed late, is thought by some authors to indicate a descending degeneration (?) of those motor fibres of the spinal cord that have been cut off from their so-called

"trophic centre" by the exciting lesion. It is always associated with a marked increase of the spinal reflexes, a point of great clinical importance. This condition is known as "tetanoid" or "spastic" paralysis. The peculiar gait of these subjects has been previously discussed.

In *pseudo-hypertrophic paralysis* the child first gives evidence of the commencement of the disease by a weakness of the legs and a clumsiness in walking, which is exhibited by frequent stumbling and falls. Gradually the patient assumes a characteristic attitude and gait.

The attitude is very peculiar. In the standing posture the back is thrown beyond the proper position, so that a vertical line dropped from the shoulders frequently falls behind the sacrum; this antero-posterior



FIGS. 50, 51, 52, 53.—ATTITUDES ASSUMED IN PSEUDO-HYPERTROPHIC PARALYSIS DURING ATTEMPTS TO RISE. (Gowers.)

curvature entirely disappears, however, when the patient is in the sitting posture. The feet are placed wide apart so as to increase the base of support. The heels are usually drawn upward by a contraction of the tendo-Achillis. In the effort to preserve the balance the arms are held at the side with the hands extended, and the slightest touch may cause the patient to fall. Another remarkable feature of the disease is the difficulty which is experienced in rising from the recumbent, or even the sitting posture. The sufferer uses surrounding objects as a means of rising, drawing the body upward by the hands. When unable to reach such assistance, the steps which are taken to rise are thus described by Gowers: "If laid, for example, on his back upon the floor and told to

rise, he would first with great difficulty turn on his face; he would next get on his knees, his head being almost between his thighs; from this position he would gradually extend himself, so that he stands upon his feet and hands with all his limbs extended; finally he would extend the hip-joint by grasping the thigh with the hand and pushing up the body, as it were, by the arm." This movement of "climbing up the thighs," as it has been termed, is an indication of weakness in the muscles which straighten the knee, and also those which extend the trunk upon the thigh—the extensors of the hip-joint.

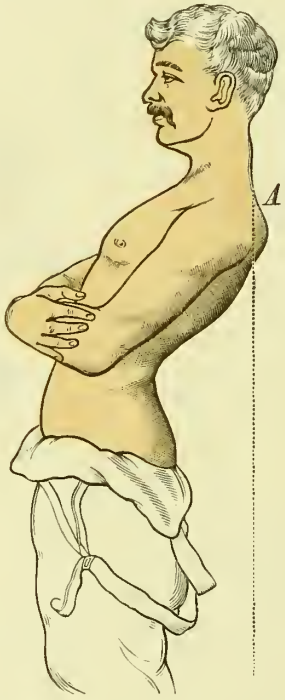


Fig. 54.—Attitude Created by Atrophy of the Back Muscles. (Bramwell.)

The gait of these patients is associated with an oscillation of the body from side to side, or a waddling movement. The advance made at each step is very small, and a difficulty seems to be experienced in flexing the thigh upon the abdomen.

The muscles of the calf exhibit early a firmness and increase in size which is not proportionate to their motor force—as that is far below normal. Soon they become excessively developed, as do also those of the buttock; while the other muscles of the leg commonly grow smaller from atrophic changes.

The latissimus dorsi and the lower part of the pectoralis major muscles exhibit marked wasting in a very large percentage of cases. In some instances all the striated muscular fibres of the body, including even the heart, may become affected.

In *spinal meningitis* of the acute form the patient lies with the legs and thighs flexed, and shows evidences of great suffering in the countenance. The muscles of the neck are attacked by spasms which draw the head backward. The patient dreads all movements, because they increase both the pain and the spasms of the muscles.

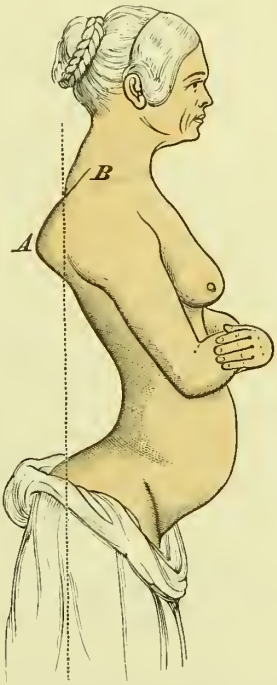


FIG. 55.—ATTITUDE CREATED BY ATROPHY OF THE ABDOMINAL MUSCLES. (Bramwell.)

Children affected with *acute poliomyelitis* are often delirious and have febrile symptoms. The paralyzed limbs lie motionless and the muscles are flaccid. Tremors and twitchings in the facial muscles and the tendons of the wrist are often observed, but they are the result of a rapid elevation of the temperature rather than a symptom of this special disease.

In *progressive muscular atrophy* the "bird-claw" appearance of the fingers attracts attention at once (Fig. 43). When the muscles of the arm and forearm are badly wasted the limb hangs at the side in a help-

less way, "as if it were tied to the body by strings." If the muscles of the lumbar region be attacked the belly becomes slightly prominent and tense, and the back is strongly arched in order to balance the trunk (Fig. 54). A line dropped from the shoulders falls behind the hips as the patient stands erect. If the abdominal muscles are atrophied the belly falls forward to a marked extent, and the back is arched in such a manner by the healthy lumbar muscles that a vertical line from the shoulders passes through the sacral region (Fig. 55). The muscles of the lower limbs are seldom so severely wasted as to prevent the patient walking.

In *hysterical paralysis* the patient (usually a young woman) is often confined to the bed. Todd has described the facial appearance of this class of patients as characterized by a "remarkable depth and prominent fullness with more or less thickening of the upper lip, and by a peculiar drooping of the upper eyelid." Sometimes the muscles of the limbs are flaccid, while in others the legs are stiffly extended and the feet are turned inward. The nutrition of the muscles is generally good, and marked atrophy is seldom present.

In *cerebro-spinal sclerosis* the face first attracts attention by a stupid and vacant expression, the half-open mouth, an oscillation of the eyeballs (nystagmus) in some instances, and a contracted state of the pupils in many cases. The speech is liable to be of a "drawling" character, and the tone of the voice monotonous. The head is often turned slightly to one side during attempts at walking, or perhaps is drawn a little backward—a point which is explained by Bramwell as an effort on the part of the patient to prevent unsteadiness of the head by an artificial stiffness of the neck. The gait has already been described.

SYMPTOMS OF NERVOUS DISEASES REVEALED BY THE EMPLOYMENT OF VARIOUS TESTS.

We are prepared by what has been said in the preceding pages of this volume, to consider intelligently the various tests which are employed (exclusive of the tests of vision which have been already discussed) as aids in the diagnosis of certain forms of nervous diseases. The following table may aid the reader in his study of the closing pages of this section:—

A TABLE OF THE MORE IMPORTANT TESTS OF THE NERVE OR NERVE CENTRES.

Tests employed to determine the REFLEX EXCITABILITY of the spinal cord:—

1. The "SUPERFICIAL" or SKIN REFLEXES.
2. The "DEEP" or TENDON REFLEXES.
3. The "ORGANIC" REFLEXES.

Tests of MOTOR PARALYSIS are employed for the following purposes:—

1. To determine its EXACT LIMITS.
2. To determine its DISTRIBUTION.
3. To determine the TROPHIC CONDITION of the affected muscles.
4. To determine the POWER OF CO-ORDINATION of muscular movement.
5. To determine the so-called "MUSCULAR SENSE."
6. To determine the IRRITABILITY of the muscles.

Tests to determine the "IRRITABILITY" of the muscles:—

A. MECHANICAL TESTS—

1. For "*diminished muscular tension.*"
2. For "*increased muscular tension.*"
3. For *fibrillary twitchings.*
4. For *tremors.*
5. For *contracture of muscles.*

B. ELECTRIC TESTS—

1. By the *faradic current.*
2. By the *galvanic current.*

Tests for the SENSORY NERVES enable us to decide respecting the following conditions:—

A. Abnormalities of TACTILE SENSIBILITY—

1. *Anæsthesia.*
2. *Hyperæsthesia.*
3. *Delayed sensation.*

B. Abnormalities of SENSIBILITY TO TEMPERATURE.

C. Abnormalities of SENSIBILITY TO PAIN.

D. Abnormal condition of the ORGANS OF THE SPECIAL SENSES.

THE SPINAL REFLEXES.

"SUPERFICIAL" OR "SKIN REFLEXES."—These are performed by different segments of the cord.

Stimulation of the skin of the sole of the foot by a scratch, prick, or touch with the nail,* for example, induces a contraction of the foot muscles (*plantar reflex*) through the lower part of the lumbar enlargement of the cord.

The skin of the buttock calls into action the glutei muscles (*gluteal reflex*) through a segment which corresponds to the escape of the fourth or fifth lumbar nerve.

The skin upon the inner aspect of the thigh causes the cremaster muscle to draw the corresponding testicle toward the external abdominal ring (*cremaster reflex*), by influencing the cord at the level of the first or second lumbar nerves.

The skin upon the side of the abdomen creates reflex movements of the abdominal muscles (*abdominal reflex*) by affecting a segment of the cord situated between the levels of the eighth and twelfth dorsal nerves.

* A sharp-pointed instrument is best adapted for the excitation of the skin reflexes.

The skin upon the side of the chest creates a reflex response in the region of the epigastrium (*epigastric reflex*), which depends upon a spinal segment extending from the fourth to the seventh dorsal nerves.

Finally, the skin between the shoulder blades causes the posterior axillary fold or the teres major muscle to contract (*scapular reflex*), by influencing the spinal segment between the levels of the fifth cervical and third dorsal nerves.

By *means of these reflexes* we are thus enabled to test the various spinal segments from the neck to the terminal extremity of the cord. Should any be found to be absent it should be remembered: (1) that the reflex excitability of the cord varies with individuals, and is always greater in youth than old age; (2) that the plantar, cremasteric, abdominal and epigastric reflexes are variable in health but are more constant than the scapular; (3) that cerebral lesions may impair them on the side of the hemiplegia, for reasons not as yet well understood;* and (4) that systematic lesions of Burdach's or Goll's columns (see Fig. 32) tend to diminish or abolish them.

Deep or "Tendon Reflexes."—These are also of great value as a means of determining the condition of excitability of different segments of the cord. The ones now commonly employed are called the *knee-jerk* or *patella reflex;* the *peroneal reflex;* the *foot clonus;* and the *tendo-Achilles reflex.* The method of obtaining these reflexes in the most satisfactory manner will be described separately. It is important, however, to remember one fact in connection with them before deciding as to their clinical significance, viz., that the *reflexes should be tested on both sides and compared with each other*, because any perceptible differences between the two sides are an indication of some pathological lesion of the cord.

In exceptional cases, the knee-jerk may be absent in health. These exceptions are not sufficiently common, however, to detract from the clinical value of the test.

*Gowers advances a theory to explain this fact, which is certainly ingenious and possibly its true interpretation. He starts with the assumption that the corpora quadrigemina, or the optic thalami contain a centre which inhibits or restrains the manifestation of the skin reflexes in man, as the optic lobes have been proven to do in the frog. He assumes, in the second place, that the higher or motor centres of the cerebral cortex are capable in health of overpowering or controlling in some way this centre.

Now, if the motor centres are prevented from exercising this function (by becoming themselves diseased or mechanically separated from the fibres that are functionally associated with them, as in the case of apoplexy, softening of the brain substance, tumors, etc.) the centre which inhibits the skin reflexes is enabled to act without restraint, thus causing them to become abolished. On the other hand, when the motor paralysis is due to some lesion of the spinal cord, the fibres through which the inhibiting centre acts upon the spinal segments below the seat of the spinal lesion are severed; hence the skin reflexes are no longer controlled by the higher centres, and are therefore enabled to respond to even more delicate tests than in health.

The *knee-jerk* has for years been recognized and employed by Charcot in diagnosis, although it was first systematically investigated as a clinical symptom by Westphal and Erb. Gowers remarks in a late work: "It is not a little curious that this knee-jerk, which for generations has amused school-boys, should have become an important clinical symptom."

To properly test this reflex movement of the limb, the muscles of the quadriceps extensor tendon must be put upon the stretch to a moderate degree, and the leg be unrestricted in its ability to respond. The common method employed is to have the patient cross the leg over the knee and allow it to hang passively at an angle of nearly ninety degrees. Perhaps a still better way, is that employed by Gowers, viz., to allow it to hang over the forearm of the physician when his hand is placed upon the opposite knee of the patient; because in this way the jerk is often elicited in stout people when it otherwise fails. The space between the patella and the tibia is then struck with a percussion hammer or the side of the physician's hand, upon the bare skin, with *sufficient force to slightly increase the state of muscular tension* which has resulted from flexion of the leg. This will cause a reflex contraction of the quadriceps extensor muscle and the foot will be jerked upward without the volition of the patient as a factor in the movement.

The *ankle-jerk*. If the muscles of the tendo-Achilles be put upon the stretch by flexion of the foot, a blow upon that tendon will cause a similar extension of the foot.

The *foot-clonus*. When the *excitability of the cord is excessive*, if the foot be firmly flexed and held so by the pressure of the hand against the sole, a series of rhythmical reflex movements of extension follows, which vary between six and ten per second. They can be traced upon a revolving drum, by attaching a pencil to the foot, as easily as a sphygmographic tracing is made. This clonus is more apparent when the knee is firmly extended than when flexed.

The *peroneal reflex*. The tendons of the peroneal muscles pass to the bones of the foot at the outer side of the ankle. A blow made upon them when the *foot is bent inward* so as to produce a moderate degree of tension of these muscles, will elicit a reflex movement, as in the case of the patella tendon.

The "*front-tap contraction*." Gowers has described a reflex test for increased spinal irritability that he considers particularly delicate. It consists in flexing the foot with the hand upon the sole, the knee being extended, and applying the blow to the muscles on the anterior aspect of the leg. It is followed by a reflex contraction of the muscles of the tendo-Achilles, which are not directly affected by the blow.

Although the deep reflexes are commonly tested only in the lower

extremities, the same phenomena may be elicited in the triceps or biceps muscle of the arm as in those of the thigh and calf, if subjected to the necessary position to insure tension of the muscles before the tap is given over the tendon.

Let us attempt to summarize the more important clinical deductions pertaining to these deep spinal reflexes.

1. A *persistent foot-clonus never occurs in health.* It indicates that the lateral columns of the cord are probably involved by some spinal lesion. In supposed hysterical affections this symptom will often decide the question of the existence of organic disease. It must not be mistaken for the involuntary foot-clonus which sometimes occurs when an unnatural posture is long maintained, even in health. It is usually *associated with exaggeration of all the other deep reflexes.*

2. All *reflex tests become abolished* when the muscles are separated from their connection with the spinal cord; hence, severing of a nerve, posterior spinal sclerosis, compression of the spinal nerve roots, destruction of the gray matter of the cord, poisons, etc., are often associated with their complete abolition.

3. Disease of the *lateral columns* usually *decreases the skin reflexes*, especially those of the trunk. This is particularly true of the so-called descending degeneration of these columns, which follows the development of cerebral lesions.

4. *Sclerosis of the lateral columns always increases the "deep" or tendon reflexes.*

5. When marked *incoördination of movements* is present and the *deep reflexes* are not abolished, it indicates that sclerosis of the lateral columns probably co-exists with similar changes in Burdach's columns.

6. *Spasm* is a marked symptom in many diseases of the spinal cord. It commonly indicates an excessive action of the reflex motor centres. It is particularly common as an acute symptom in spinal meningitis. In chronic organic diseases of the cord, it assumes the form of *contracture of muscles*, especially if the lateral columns of the cord are attacked; this condition becomes transformed into that of a genuine spasm when the slightest forms of peripheral impressions are experienced, as in delicately manipulating the muscles, for example.

The Organic Reflexes.—*The Bladder and Rectum.*—The bladder and rectum are more or less affected, in respect to the performance of their functions, by those diseases of the spinal cord that tend to impair or destroy the special nervous mechanism connected with them. The nocturnal incontinence of children, who "wet the bed" in spite of all precautions against the accident, is an evidence either of spasm of the bladder, excessive stimulation of the centripetal nerves connected with the so-called "vesical centres" of the spinal cord, or atony of the

sphincter muscle. If due to spasm, it may be excited by worms in the intestine. When the spinal cord is subjected to sudden injury low down, or is attacked by some disease process that involves the *lumbar region* of the spinal cord, the bladder and the rectum are liable to be paralyzed. In such cases, if the paralysis be complete, the urine has to be drawn with a catheter. Sometimes, if not drawn at regular intervals, it overflows, when the bladder becomes excessively distended. This compels the patient to wear some form of apparatus to prevent wetting of the clothing. Urinal overflow should never mislead the physician into the belief that the bladder is empty.

True incontinence is a rare condition in the adult. The term "incontinence" is not, however, restricted by many authors to that condition characterized by a continued escape of urine and emptiness of the bladder.

Bramwell gives the following table, as an aid in the diagnosis of two forms of incontinence that are commonly recognized :—

	Occurrence.	*Effect of effort, coughing, etc.*	*Age.*	*Urine.*	*Associated nerve-symptoms.*	*Effect of Treatment.*
Spasmodic Incontinence.	Occasional and intermittent.	Nil.	Generally young.	Clear, acid, and normal.	None, unless hysteria	Good.
Paralytic Incontinence.	Constant.	Forces away urine.	Any age: but generally old age.	May be ammoniacal and purulent.	If central, generally a similar affection of rectum and paraplegia.	Very often unfavorable.

In all cases, where either incontinence or retention of urine is developed in connection with abnormal nerve symptoms, the urethra and rectum should always be carefully explored for the purpose of detecting disease, or of eliminating, if absent, all local causes of these conditions.

The Sexual Reflex.—In the *lumbar region* of the spinal cord a centre is situated that governs the acts of erection and seminal ejaculation. It may be called into action either by impressions made upon the sensory nerves of the skin of certain regions, or by cerebral influences that are exerted upon the sexual centre as the result of some emotional impulse.

Destructive processes in this centre of the spinal cord cause a loss of power of erection and ejaculation,—*i.e.*, impotence. General spinal weakness from any cause may also lessen the duration and degree of erection or render ejaculation premature.

Linked with some forms of nervous disease comes *priapism*, or the state of erection without sexual desire. It may be a result of irritation or excessive stimulation of the following structures: (1) the sensory

nerves (as in gonorrhœa); (2) the sexual centre itself; (3) the nerves that convey the emotional impulses from the brain to the sexual centre through the spinal cord; (4) the parts of the cerebral cortex functionally associated with sexual emotions. The latter are, as yet, undetermined. Priapism may be complete and painful, or incomplete and painless. It may last for days. It occurs not infrequently in connection with disease in the lower cervical or upper dorsal regions of the spinal cord.

The Pupillary Reflex.—The last two cervical and the three upper dorsal segments of the spinal cord probably embrace the so-called "*cilio-spinal centre.*" From it sympathetic nerves pass to the muscular fibres of the iris. *Irritation* of this centre causes the pupil to dilate; *destruction* of it causes the pupil to contract. The tests for the "Robertson pupil" have been described already (p. 120). This peculiar condition of the pupil is one of the most valuable signs of the disease called "locomotor ataxia" or posterior spinal sclerosis. It is the only condition of the eye that allows of the movements of the pupil in attempts to focus near objects and destroys at the same time the response of the pupil to varying degrees of light.

TESTS FOR MOTOR PARALYSIS.

As mentioned in preceding pages, the physician may be called upon to recognize five forms of paralysis of motion in the trunk and extremities viz.: monoplegia, hemiplegia, paraplegia, hemiparaplegia, and complete paralysis. If the paralysis be of an incomplete or partial form in any type, it is called "paresis."

Cerebral diseases commonly produce either *monoplegia* or *hemiplegia* of the opposite side of the body, in case paralysis occurs either as a result of localized pressure upon the brain or of destruction of some of its component fibres.

In those cases where the lesion involves *both hemispheres*, the paralysis may be bilateral. Such lesions are generally present at the base of the brain.

When "crossed paralysis" is developed, definite information is afforded respecting the seat and extent of the lesion.

Spinal paralysis is bilateral in the great majority of cases, and is limited to the muscles of the legs (*paraplegia*). This is to be explained (1) by the fact that the motor tracts of the spinal cord are in a somewhat close relation to each other, and that acute diseases are seldom confined to one lateral half of the cord; and (2) to the fact that the muscles *below the seat of the lesion* are necessarily paralyzed in proportion to the amount of injury sustained by the motor fibres.

In those rare cases, where the spinal lesion is situated above the point at which the nerves to the upper extremities are given off, bilateral

paralysis is apparent in both extremities (arms and legs), constituting the condition termed "*cervical paraplegia*" by some authors.

The points to be tested in any case of motor paralysis have been enumerated in a preceding table. Without further explanation, we will now proceed to consider each separately.

The Seat and Limits of the Paralysis.—To ascertain the exact limits of the paralysis is important as an aid in the determination of the seat of the exciting lesion, be it cerebral or spinal. The *peculiarities of attitude and gait* will often aid, in a rough and imperfect way, in deciding as to the muscles that are chiefly affected; but a more detailed examination of the separate muscles, by instructing the patient to *perform designated movements* that shall call different sets successively into action, will be more accurate and scientific. To employ these tests in a skillful manner, however, the physician must first be thoroughly familiar with the action of the various muscles, both individually and in conjunction with others.

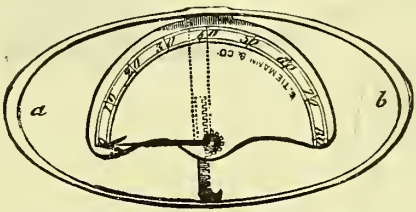


Fig. 56.—The Dynamometer of Matthieu. When taken in the hand and pressed, the two sides of the elliptical spring, *a*, *b*, are approximated, and the finger of the dial records the exact amount of force exerted upon the spring. One advantage of this instrument over all other devices of a similar kind is, that the index does not return to zero, but remains at the point indicating the greatest amount of force exerted by the hand of the patient. It is not necessary, therefore, to watch the index while the instrument is being used. Both hands should be tested separately and the results noted in the record of each case. A modification of this instrument has been devised to test the power of the muscles of the lower extremity.

In some forms of spinal diseases, where great accuracy in the diagnosis and localization of the lesion is required, it may become necessary to *test the motor condition of the various spinal segments* by means of the muscles that are governed by them. The investigations of Yeo and Ferrier upon the monkey tribe, as well as those of Marcacci and Bert upon dogs and cats, seem to have demonstrated that each pair of spinal nerves exerts an influence upon definite muscular movements.

The Degree of Motor Paralysis.—Complete paralysis, of course, abolishes all power in the muscles affected, but paresis does not, and therefore varies in degree. It is often important to decide as to the *force that can be exerted* by the partially paralyzed muscles before completing a diagnosis. This can be best accomplished in the muscles of the upper extremity, by the employment of an instrument devised by Matthieu, an instrument-maker of Paris, called the *dynamometer*. It is shown in the cut.

When grasped in the hand, the index shows the amount of power that is exerted upon the spring. The index remains fixed until mechanically replaced after it has been used; this enables the physician to direct his attention to other points in the case while the patient is trying his muscles. It is really a test for the "grasping power" of the flexor muscles of the forearm only. An apparatus for tracing the effects of muscular contraction is sometimes attached to the dynamometer. It is called the dynamograph. It shows irregularities of muscular contraction.

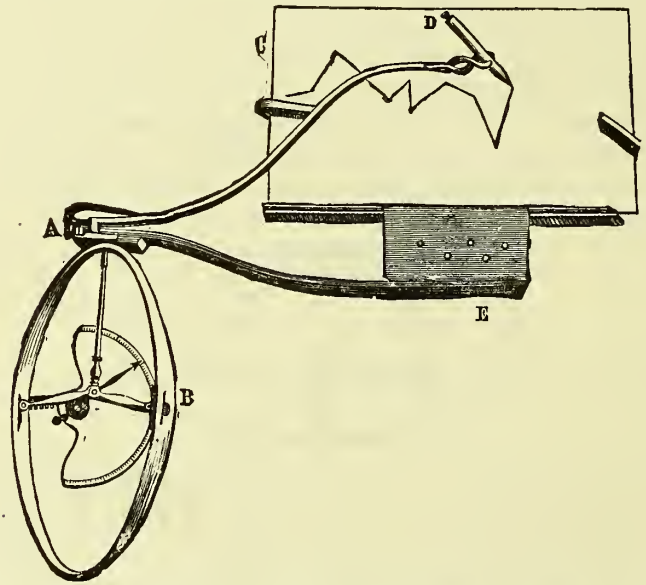


Fig. 57.—The Dynamograph.—This modification of the dynamometer enables the physician to observe and record the condition of the muscles in respect to their ability to maintain tonic contractions. In health, the line drawn by the pencil should be *perfectly straight* when the elliptical spring is steadily compressed by the hand for a few seconds. If the grasping power is intermittent or incapable of being maintained continuously for an interval of several seconds, the line described on the recording tablet will be irregular. The slips of paper, indicating the deviations of the pencil, can be preserved in the case-book, and by comparison they may show improvement or increase of the muscular debility.

The strength of the muscles of the calf can be tested, as Gowers suggests by requesting the patient to *jump on tip-toe.* And also by an ingenious instrument devised by Dr. Birdsall, of this city, called the "foot-dynamometer."

A third method of testing the various muscles is to request the patient to *exercise all possible resistance* to some special movements that the physician creates by manipulation; or, on the other hand, to request the patient to perform some special movement and estimate the *amount of force required to prevent his doing so.*

Bilateral paralysis requires that the power of the muscles should be compared with that of a healthy individual of about the same muscular

development as the patient, if great accuracy is desired. In unilateral paralysis, the healthy side can be used as a standard of comparison.

THE TROPHIC CONDITION OF THE MUSCLES.

The amount of atrophy or wasting that ensues simply from disuse of the muscles must be distinguished from that due to a loss of the so-called "trophic function" in the nerves that supply the muscles.

Rapid wasting of a muscle occurs when the nerve fibres that supply it are cut off from the so-called "trophic centre" of the spinal cord with which they are normally connected. The multipolar nerve cells in the *anterior horns of the spinal gray substance* are probably the "trophic centres" for the motor fibres found in the anterior roots of each spinal nerve. When these cells become the seat of disease, the muscles undergo extreme and rapid atrophy. Similar changes also occur when the nerves are cut off from the connection with them, as in wounds of a nerve, pressure upon a nerve, etc.

THE POWER OF COÖRDINATION OF MUSCULAR MOVEMENTS.

Disease of the cerebellum, the lemniscus or fillet-tract (Fig. 11), and of the columns of Burdach and Goll in the spinal cord (see Fig. 32) is commonly associated with a peculiar inability on the part of the patient to *perform certain muscular movements in a proper way*, because the muscles do not act in the sequence necessary to accomplish them. This is termed "incoördination of movement."

Various tests are employed in determining the degree of this abnormal state, because one that will answer for the lower limbs will not for the upper extremities, or *vice versa*. Besides, it is necessary in these cases to decide both as to the ability to perform complex movements with accuracy, and also as to the state of the so-called "muscular sense." Let us consider first the tests of the former.

When so-called "ataxic patients" are requested to *follow a designated line* in the floor or carpet as they walk across a room, they invariably keep their eyes fixed upon the floor, and have extreme difficulty in following the line. Now, ask such a patient to do the same with the *eyes looking straight ahead* of him, and the attempt will prove a still more lamentable failure than before. In advanced stages of the disease, the patient may fall.

A second manifestation of lack of coördination in the muscles of the legs lies in an inability on the part of the patient to *stand erect with the feet in close contact*, without swaying or falling. This is rendered still more difficult when the patient is instructed to close the eyes. It must be remembered, however, that an inability to stand erect and motionless with the eyes closed is not always due to ataxia. I have seen the

same result produced artificially in a healthy subject by freezing the soles of the feet to a degree sufficient to destroy the appreciation of its contact with the floor or carpet. The test is a reliable one only for the presence of *marked anæsthesia* of the soles of the feet; hence it is common in ataxic subjects, in whom sensation is always more or less impaired.

Considerable stress may be laid upon this point, because it is stated by some writers that this symptom or test is to be regarded as a positive sign of locomotor ataxia. That it is a valuable diagnostic point in that disease, when associated with other evidences of its existence, cannot be disputed; but it is by no means a pathognomonic symptom, as it might exist in any disease (cerebral, spinal, or functional) that could cause marked anæsthesia of both lower extremities.

In order to preserve the equilibrium during an erect posture when the feet are in close contact, it is necessary that the nerves of sensation allow the keenest appreciation by the nerve centres of variations in the amount of pressure exerted by the weight of the subject upon the different regions of the sole of the feet. When such information is withheld from any cause (chiefly by sensory paralysis or anæsthesia) the nerve centres can no longer properly govern the muscles to counteract a tendency toward a fall, provided that the sense of sight is prevented from giving them the necessary information. This explains why it is that ataxic patients often notice a difficulty in washing the face at a washstand when the eyes are closed; why they keep the vision fixed upon the ground as an aid in governing the movements of walking; and why they keep their feet well apart when standing still, in order to increase their base of support. All the other symptoms of ataxia may thus be mechanically interpreted and be employed as tests in diagnosis.

Among these symptoms may be mentioned: a difficulty in climbing a flight of stairs, on account of the feats of balancing required to do so; a difficulty in placing the foot rapidly and accurately upon some small object, as in mounting a horse by means of the stirrup; and many others of a similar kind.

The tests for incoördination of the muscles of the upper extremity have not as yet been described.

The handwriting is sometimes seriously affected in ataxic patients, by an inability to make *continuous curves* with accuracy, as in the case of the capital letters C, D, G, etc. This is because the acts required of the muscles in making these curves are complicated and must follow each other in a certain sequence, in order to properly execute them. Again, the *clothes are buttoned and unbuttoned with extreme difficulty*, because these simple acts require coördinated muscular movements of a complex character. Food and drink are carried to the mouth with difficulty in some cases, especially when the eyes are closed or in the dark. These

patients cannot touch designated parts of the face with the finger with accuracy and rapidity when the incoördination of the upper extremities is well developed, or the so-called "muscular sense" is destroyed. These tests will be mentioned later.

THE MUSCULAR SENSE.

By this term we mean the power which each individual possesses, in health, of discriminating in regard to the amount of muscular force required to accomplish certain ends. Thus, for example, if two objects are held in the hands, the difference in weight between them should be estimated with an approach to accuracy. Again, if the eyes be closed, the fingers can be made to touch rapidly any designated portion of the body with perfect certainty. Movements of progression should be also performed with the eyes closed nearly as well as when open, if the distance be short and the location a familiar one. Finally, the handwriting should not differ materially as regards the formation of letters when made with the eyes shut or open.

Now in some forms of nervous derangements, the muscular sense is impaired; hence it becomes necessary to sometimes test it before making a final diagnosis. Several tests are commonly employed. They may be designated as the "weight" test, the "movement" test, and the "handwriting" test.

In testing the power of discrimination of weights held in the hands, it is best to have them all of *uniform size*, in order to avoid the patient using the sense of sight as a factor in his decision. Hanging different weights from the foot in a handkerchief will test the muscular sense in the lower extremity. Metallic balls of different thickness but of uniform size, either covered or uncovered, answer the purposes of the "weight" test. The ability on the part of a patient to tell with the eyes closed the exact position of a limb in reference to other parts of his body, when different attitudes are assumed, may be interpreted as an exhibition of the muscular sense.

To test the accuracy of movement, direct the patient to close the eyes tightly, or blindfold him, and then instruct him to rapidly place the forefinger of either hand alternately on some spot upon his body which shall be designated in each instance, as the nose, upper lip, lower lip, ear of either side, etc. When the lower limbs are to be tested, he may be instructed to place his great toe upon the opposite instep, heel, knee-cap, etc., or to raise the foot to a given height when lying on the back, and then to slowly lower it till it rests upon some designated spot on the other foot.

The *handwriting* of a patient is often of value in diagnosis; especially when a sentence written with the eyes open is compared with

the same written with the eyes closed. In health the muscular sense should enable almost any one to perform both with a fair degree of precision. In motor paralysis or ataxia the changes are marked, especially in the latter, because incoördination of the muscles prevents the formation of continuous and well-formed curves, even when the eyes are open, and utterly destroys the legibility of the letters if closed. The existence of tremor, or the presence of profound motor paralysis, will, of course, interfere most seriously with the ability of the patient to write legibly, if at all, irrespective of the aid of vision.

TESTS TO DETERMINE THE IRRITABILITY OF THE MUSCLES.

In some forms of cerebral and spinal disease it becomes necessary to test the so-called "irritability" of the muscles. Two forms of tests are employed for this purpose, viz., mechanical and electric.

Mechanical tests enable us to decide (1) as to the existence of *diminished* or *increased tension* of the muscles; (2) the *presence of twitchings* of individual muscular fibres in certain regions (as if a live animal were imprisoned beneath the skin); (3) the *presence of tremor;* and (4) the state of muscular rigidity and permanent shortening known as "*contracture.*"

Electric currents are employed as aids in diagnosis, chiefly in deciding the question of the existence of *degenerative changes* in the muscles. The increase or decrease of such changes, when they have been found to exist, can also be scientifically determined by the employment of electric tests from time to time.

MECHANICAL IRRITABILITY OF THE MUSCLES.

When the muscles are subjected either to manipulation, a light tapping with the tip of the finger, or a stroke with a percussion hammer, either an abnormal exaggeration or a diminution of the mechanical excitability of the part struck is sometimes detected in connection with disease or injury of the brain, the spinal cord, or of the nerves themselves.

The following clinical deductions are offered as a summary of these tests:—

1. *Motor paralysis* usually decreases the mechanical excitability of the muscles affected.

2. When the "*galvanic excitability*" is markedly increased (reaction of degeneration) the *mechanical excitability* of the muscles is also increased.

3. A *marked increase* in the "*deep*" or "*tendon reflexes*" is likewise associated with an increase in the mechanical excitability of muscles. This is particularly characteristic of sclerosis or hardening of the lateral columns of the spinal cord.

4. *Atrophy*, or *wasting of the muscles*, as the result of disease-processes, such as functional paralysis, poliomyclitis, etc., usually tends to diminish the tonicity of the affected muscles (state of flaccidity). An exception to this rule exists for a time in amyotrophic lateral sclerosis and other conditions where marked muscular rigidity precedes the atrophy.

5. Any disease that tends to cause *irritation of the motor nerve-fibres*, or to arrest the *control of the brain over the spinal segments*, is liable to be associated with rigidity of the muscles. Twitchings, muscular cramps, tremors, spasms, and contractures, may be associated with this increase of muscular tension.

The distribution of the muscular rigidity differs if the exciting cause be confined to the spinal coverings or the substance of the cord itself. In the former case the flexors are chiefly involved. Hardening or sclerosis of the lateral columns of the cord usually causes the lower limbs to be firmly extended and closely approximated to each other.

6. The disease known as "progressive muscular atrophy" is the one most commonly associated with contractions of separate fibres or bundles of fibres in the muscles,—the so-called "*fibrillary twitchings.*" These twitchings are not confined, however, to this condition. Hypochondriacs and certain functional diseases of the spinal cord may also be associated with them. A slow destructive process affecting the motor nerve-cells, or the motor nerves themselves, may also cause them.

7. A permanent shortening of muscles (state of *contracture*) is a frequent sequel to extensive atrophy or wasting of the muscular fibres. It may result also from the prolonged and unrestrained action of certain muscles whose antagonists are lacking in muscular power, as in the case of infantile paralysis, lateral sclerosis, etc.

TESTS EMPLOYED IN THE DIAGNOSIS OF LESIONS OF THE WHITE SUBSTANCE OF THE CEREBRAL HEMISPHERES.

Lesions of the centrum ovale have always been regarded as peculiarly difficult of detection and localization during life. In quite a large proportion of cases where extensive disease of this portion of the brain has been discovered after death, the presence of the lesion has been either unsuspected during life, or, if suspected, imperfectly localized. The fibres which assist to form the white substance of the brain comprise the commissural, associating, and peduncular tracts. These have been described already on page 17. Dr. M. A. Starr has lately written a very interesting and lucid article upon lesions of the centrum ovale (*Med. Record*, Feb., 1886). He explains how a severance of the "commissural" and "associating tracts" of the cerebral hemisphere may be

recognized during life by a careful examination of the mental faculties of the patient and by testing the ability of the patient to perform identical and simultaneous bilateral movements of the face and the extremities.

The tests designated by this author may be summarized as follows :—

To Test the Commissural Fibres.—If the two hands are moved in unison, during attempts to draw a circle or write one's name with each hand simultaneously, the right hand should move to the right to exactly the same extent as the left hand does to the left. Therefore, although the writing of the right hand is legible, that of the left hand will be backwards and can be easily read only by the aid of a mirror ("mirror-writing" test). Whenever simultaneous bilateral movements are found to be defective, the "commissural" fibres (Fig. 6) will probably be found to be congenitally imperfect or impaired by a morbid lesion. The history of the patient would decide which of these two probably existed in any given case. On this principle, bilateral movements of the upper and lower limbs can be tested in a variety of ways.

The Tests for the "Associating-tracts" are somewhat more complex—although they are by no means difficult to understand. The following suggestions are pertinent to this field :—

1. To test the *connection between the hearing centres and Broca's speech centre* (temporo-frontal tract). Request the patient to repeat promptly words dictated to him. Notice also if any impairment of spoken language exists during a continued conversation.
2. To test the tract between the *hearing and sight centres* (occipito-temporal tract). Ascertain if the patient can read intelligibly to himself and afterwards tell correctly what he has read.
3. To test the "*occipito-temporo-frontal tract.*" Ask the patient to read aloud some selected paragraphs.
4. To test the tract which unites the *sight centres with the motor centres* (occipito-central tract). Ask the patient to write what a selected paragraph contains.
5. To test the tract which unites the *hearing centres with the motor centres* (temporo-central tract). Request the patient to write from dictation.
6. To further test the tract uniting the *sight and hearing centres with the speech centres.* Ask the patient to name the color of different objects placed in his field of vision; also their form and general appearance.
7. To test the tract connecting the *frontal convolutions and the motor centres* (fronto-central tract). Request the patient to write what he speaks to himself—preferably in an audible whisper, so that the observer can detect an error.
8. To test the tract connecting the *smell and taste centres with the hearing and speech centres* (the hippocampo-temporo-frontal tract). Request the patient to speak the names of particular odors or tastes, of which he may be made cognizant during the interview by the physician or an attendant.
9. To test the connections between the *smell, taste and hearing centres* with the *motor centres* (the hippocampo-temporo-central tract). Request the patient to write the names of odors or taste-impressions, of which he may be made cognizant at the time by the observers.

After such tests, as those described, have been carefully made, all forms of impairment of the various tracts observed may be contrasted, and thus be made a basis for a diagnosis of a lesion of the white substance of the hemispheres. Some knowledge of the anatomical relations of the various associating and commissural tracts is of course necessary in order to form the proper deductions respecting an individual case.

It should be remembered :—

1. That the "*occipito-temporal*" *tract* lies in close relationship with the fibres of vision; hence, the symptoms of its destruction are liable to accompany *homonymous hemianopsia* of the same side as the affected cerebral hemisphere.

2. That the "*occipito-central*" *tract* lies on a higher plane than the occipito-temporal; hence this bundle of fibres may escape a lesion which involves the preceding tract; and the patient may retain the power of writing selected paragraphs, even if he cannot read them correctly. Such a case as that reported by Charcot (page 8) illustrates this point in diagnosis.

3. That the "*parieto-temporal*" *tract* is probably designed to allow of the association between memories of sensations of touch, pain and temperature, with the memories of those sounds by which we express such sensations in words. Patients in health can thus announce to others whether an object is hard or soft, cold or warm, rough or smooth, etc.; and a lesion of this tract may interfere seriously with the proper expression in words of the patient's real impressions gained by touch.

4. A perfect connection between the hearing and speech centres (temporo-frontal tract) is absolutely essential to correct speech; because we are enabled to produce any desired sounds only by recalling to memory and imitating similar sounds whose meaning has been gradually acquired by the ear and recorded in the cells of the cortical area occupied by the centres of hearing.

THE PRINCIPLES OF ELECTRO-DIAGNOSIS.*

The various electric tests that are employed as aids in the diagnosis of nervous affections are too complex to be fully described and explained without entering somewhat into the domain of physics and physiology. Erb† has lately written an excellent work upon the subject, and most of the later treatises upon physiology will afford general information respecting the reactions of healthy muscle to the faradaic and galvanic currents. The few practical hints which are given here are offered with an apology for their incompleteness, although it is hoped that they will assist you in your studies in this field.

* Portions of this lecture have already been published.

† "Handbook of Electro-Therapeutics," New York, 1883.

Having first moistened the electrodes and connected them with the battery in action, it is customary to hold them both in one hand (close together, but not in contact), and apply them to the ball of the thumb of the opposite hand or the cheek to see if the current is passing properly. If the current to be employed is a *very weak one*, touch the electrodes to the tip of the tongue before it is used upon the patient. The use of a reliable milliampère-meter will prove of value in determining the existence as well as the strength of a current.

Next, sponge the part of the patient's body to be tested with a *weak solution of table-salt in warm water*, in order to render the skin a good conductor of the electric currents. If the wire-brush is to be used, this step is omitted.

The "*polar method*" is the one commonly used. Apply one electrode of large size, either over the breast-bone of the patient (at about its centre) or over the back of the neck. The breast-bone is the preferable point on account of the absence of muscles in the median line.* The other electrode (of small size) is placed over some special nerve-trunk or the muscle to be tested; in case muscle is to be tested, the electrode is placed usually at the point where the motor nerve enters its substance,—the so-called "motor-point" of the muscle. In this way the action of the two poles can be readily distinguished.

Use both the *continuous or galvanic current* and the *interrupted or faradaic current* in testing muscular reactions. The former is of the greatest value in diagnosis.

In *studying the muscular reactions to the different currents* employed, remember (1) that the negative pole is called the cathode (C),† and the positive pole the anode (A); (2) that muscular contractions occur both when the current is altered in strength and when the circuit is closed or opened; (3) that the faradaic current produces an apparently continuous muscular contraction, because its interruptions are so very rapid; (4) that very weak currents do not produce contractions; (5) that alterations in the strength of the current cause proportionate variations in the contractions; (6) that the contractions are short, sharp, and sudden in health; (7) that the effects of applying the electrode over the substance of the muscle and over its motor-point are identical in health, but not in some diseased conditions; (8) that the galvanic current will not usually produce muscular contractions while it is constant, but only when its strength is modified or when the circuit is closed or broken; (9) that the

* This is known as the "indifferent point," when *polar effects* are being studied at the other electrode.

† German authors employ different symbols from those given. These are as follows: C. C. C. = Ka S. Z., C. O. C. = Ka O. Z., A. C. C. = An S. Z., A. O. C. = An O. Z. The symbols Ka = cathode, An = anode, S = closure (*Schliessung*), O, = opening (*Oeffnung*), Z = contraction (*Zuckung*).

direction of the current can be changed, without altering the position of the electrodes, by a simple apparatus that changes the cathode into the anode, and *vice versa* (the *commutator*).

The current passes always from the anode to the cathode. Hence, when the positive pole is placed on the breast or neck, and the other on the muscle to be tested, we have a *descending current.* An *ascending current* exists if the cathode is on the same distant or neutral point.

An "automatic interrupter" on an "interrupting electrode" is necessary in employing the galvanic current in testing muscular reactions.

The *descending current* (cathode over the nerve or muscle) when closed and again broken can thus give us:—

1. The cathodal closure contraction:—
   C. C. C. or Ka S. Z. of the Germans.
2. The cathodal opening contraction:—
   C. O. C. or Ka O. Z. of the Germans.

The *ascending current* (the poles being now reversed) when closed and again broken can give us:—

1. The anodal closure contraction:—
   An C. C. or An S. Z. of the Germans.
2. The anodal opening contraction:—
   An O. C. or An O. Z. of the Germans.

These four forms of contraction require currents of different strengths to produce them. They are, therefore, induced by *gradually increasing the number of cells* employed. The following order is the only one commonly observed in healthy muscle:—

1.....C. C. C. = Ka S. Z.
2.....An C. C. = An S. Z.
3.....An O. C. = An O. Z.
4.....C. O. C. = Ka O. Z.

It will be observed that the *cathodal contractions* appear first and last in health, while the *anodal contractions* follow each other; also, that the *closure contractions* precede the *opening contractions* of both the cathode and anode. When a nerve-trunk is stimulated by electric currents the formula of the normal muscular contractions is altered. This will be spoken of hereafter.

Again, as the strength of the current is gradually increased, the contractions which have successively appeared *become intensified proportionately* (as is shown below), and *new reactions* are added:—

First stage (*moderate current*), C. C. C.
Second stage (*stronger current*), C.′ C.′ C.′ and An C. C.

Third stage (*still stronger current*), C.″ C.″ C.″ and An′ C.′ C.′ and A. O. C.

Fourth stage (*very strong current*), C.‴ C.‴ C.‴ and An″ C.″ C.″ and An′ O.′ C.′ and C. O. C.

C.‴ C.‴ C.‴ is called "*cathodal tetanus*," because the contraction is very violent. Sometimes the anodal contractions both occur with the same intensity of current, thus merging the second and third stages into one. Again An O. C. may in some cases appear before An C. C.

Disease of the nerve-centres or of the nerves themselves may cause modifications of the normal formula of muscular contractions. This constitutes the key-note to the value of electric currents in diagnosis. Mechanical devices may be employed to trace the muscular contractions, as the sphygmograph does the pulse. Fig. 58.

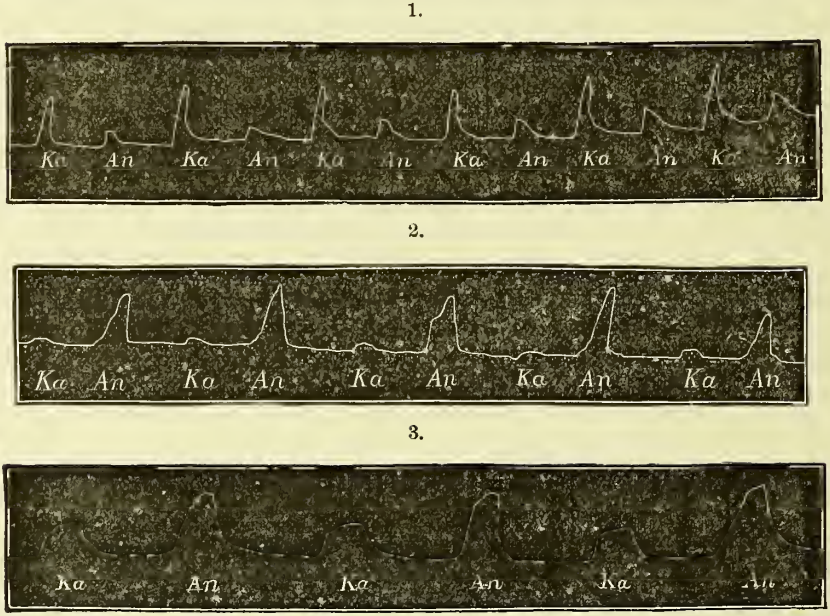


Fig. 58.—Curves of Closure Contractions in Direct (Unipolar) Stimulation of the Muscles in the Distribution of the Peroneal Nerve in the Leg. (Erb). Ka = C. C. C.; An = A. C. C. 1, Curve of health, thirty-three elements; C. C. C. is greater than A. C. C.; 2, case of chronic anterior poliomyelitis, showing reaction of degeneration with thirty-three elements; 3, same case, with forty elements. In 2 and 3, the excess of A. C. C. over C. C. C. is apparent; 3, in the slow character of the contractions is very marked.

If the destructive process is within the brain or spinal cord, and *situated above the "trophic centres"* of the nerves supplying the paralyzed muscles, the electrical reactions of the paralyzed muscles will be normal in respect to the sequence and character of the muscular contractions.

Sometimes, however, a *much stronger current* (galvanic or faradaic) is required to produce them over the healthy muscles. This fact is due to atrophic changes in the muscles. It may be of decided value in diagnosis.

When disease processes in the brain or spinal cord cause *destruction of the trophic centres* of the nerves that supply the paralyzed muscles, or when the *cerebro-spinal nerves themselves are seriously injured*, we encounter what Erb has described as the "REACTION OF DEGENERATION." This will require some explanation.

1. *Every nerve degenerates when separated from its trophic centre;* hence, the electric excitability of the *nerve*, both to the faradaic and galvanic currents, gradually diminishes and ceases entirely at the end of about two weeks.

2. The *faradaic current ceases to cause muscular contractions* when applied directly over the substance of the muscle. This is explained by the fact that the so-called "nerve-plates" within the substance of the muscle are degenerated, and currents of momentary duration fail to affect the muscular fibres.

3. The muscular contractions produced by the *galvanic current are diminished for about ten days.* Subsequently the excitability of the muscles *to slowly interrupted galvanic currents becomes increased*, so that very weak currents may excite contractions. This may disappear in five or six months.

4. The *polar reactions become altered in their sequence.* The anode contractions appear before those of the cathode, as shown below:—

1.....	A. C. C.	instead of	C. C. C.	as in	health.
2.....	C. C. C.	"	A. C. C.	"	"
3.....	C. O. C.	"	A. O. C.	"	"
4.....	A. O. C.	"	C. O. C.	"	"

5. The *character of the muscular contractions becomes altered.* In health, they are sharp, short, and sudden. When degeneration occurs, they are slow to appear; they are prolonged and continue even during the passage of the current; and, finally, they assume the character of "tetanic" contractions, irrespective of the strength of the current employed.

Finally, in unilateral paralysis the electrical reactions of the muscles of the paralyzed side should be contrasted with those of homologous muscles of the unaffected side. When both sides are impaired, the standard of comparison should be that of a healthy subject of about the same size, weight, and muscular development.

Now let us suppose that we suspect disease in, and wish to test the reaction of some special nerve,—the musculo-spiral, for example. We

place the positive pole (An) of a galvanic battery over the breast-bone with a large flat electrode attached, and the negative pole (Ka) over the nerve (where it winds around the humerus below the deltoid muscle) with an "interrupting" small electrode attached to the negative rheophore. We then put into circuit a few cells at a time and press the button of the interrupting electrode at intervals till we get a contraction of muscles. When the current is sufficiently strong to excite the nerve-trunk, contraction of the extensor muscles of the forearm becomes apparent (the *cathodal closure contraction*). Thus we ascertain the number of cells of the battery in use, or preferably the number of milliampères required to produce C. C. C. (Ka S. Z. of the Germans). Now add a few more cells, and reverse the poles by means of the commutator. When the circuit is *broken*, by releasing the button of the interrupting electrode, we get the anodal opening contraction (A. O. C., or An O. Z.), and, with a few more cells, the anodal closure contraction (A. C. C., or An S. Z). Again reverse the current, and add a few more cells. Now, on pressing the button of the interrupting electrode, we get a very intense cathodal closure contraction (C‴. C‴. C‴., or Ka S. Z‴.), and, on releasing it, the cathodal opening contraction (C. O. C., or Ka O. Z.) is developed, thus completing the chain of polar nerve reactions.

You should bear in mind that the *polar nerve-reactions differ in their normal sequence from those of the muscles* when the electrode is placed over the "motor point" of the muscle tested.

NORMAL NERVE-REACTION.

C. C. C.>A. O. C.>A. C. C.>C. O. C.

NORMAL MUSCLE-REACTION.

C. C. C.>A. C. C.>A. O. C.>C. O. C.

The *final contraction* (C. O. C.) of each of these series is seldom seen, because the current required to produce it is too painful to be endured. *Fewer cells are required to cause muscular formulæ than those of a nerve-trunk.*

In recording the results of an electrical examination of nerve-trunks and muscles it is best to arrange the record-page so that the two sides of the body may be easily contrasted. The number of galvanic cells employed or the number of milliampères of current (as shown by a galvanometer) should also be specified, and the faradaic reaction of homologous nerves or muscles should be stated for the purpose of comparison and for clinical deduction. We may follow with advantage some such plan as the following:—

NAME..... DATE..... AGE.....

HISTORY OF CASE. See page .... of CASE-BOOK.

FARADAIC TESTS.

	Right side.	Left side.	Extent of secondary coil employed. (In centimetres.)	Nerve tested.
Nerve reactions.....				
Muscle reactions.....				Muscle tested.

GALVANIC TESTS.

	Right side.	Contraction produced.	Left side.	Nerve or muscle tested.
	Cells or milliampères.		Cells or milliampères.	
Nerve-reactions.....		C. C. C. A. O. C. A. C. C C. O. C.		nerve.
Muscle-reactions.....		C. C. C. A. C. C. A. O. C. C. O. C.		muscle.

Slips of this character may be printed and kept on hand. They can be pasted into the case-book of the physician when filled out. The tests made at different dates can thus be compared with each other and the progress of each case determined.

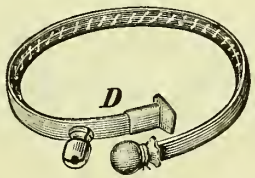


FIG. 59.—THE AUTHOR'S SPRING ELECTRODE.—*D*, the binding-post for attaching the rheophore which connects it with the battery, or with the diagnostic key-board when that instrument is employed. The motor point of the electrode is represented as enveloped in chamois-skin. It must be thoroughly dampened in salt-and-water before it is applied to the nerve or muscle to be tested. The other end of the electrode is designed to prevent slipping of the instrument after its proper adjustment.

For the purpose of demonstrating the special action of individual muscles and nerves before classes of students, as well as the study of muscle- and nerve-reactions in disease, I have devised small electrodes which may be made stationary upon any desired part of the head, limbs, or trunk, by means of straps, strips of adhesive-plaster, or insulated springs. By means of these I have been enabled to make many points clear to a large audience which would be extremely difficult to show by any

other method. Furthermore, it is often desirable to refer from time to time during an examination of a patient to the effects of currents of known intensity upon certain nerves and muscles for the sake of accurate comparison, etc. Small electrodes of the type described may be accurately placed upon a patient and allowed to remain upon the spot selected during the entire examination. To each of these a separate rheophore may be attached, and, by a simple device of my own, each may be controlled by touching a key upon a board, without movement of the operator. I can thus observe simultaneously the reactions of corresponding muscles or nerves upon the two sides, those of the leg and arm of the same side, and any other comparisons which may be required in diagnosis. The "motor-points" of the body are not always exactly where charts depict them; hence it is sometimes necessary to hunt for them within a radius of an inch or two of the normal point. When they are found with exactness, a small electrode may be fastened over the spot (with moistened absorbent cotton beneath it) and allowed to remain stationary during the

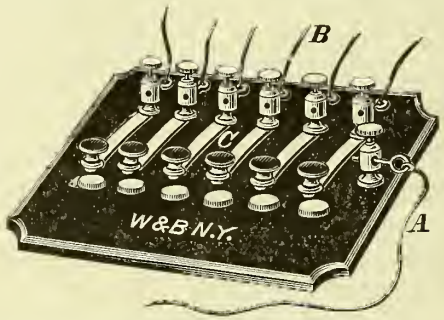


FIG. 60.—THE AUTHOR'S DIAGNOSTIC KEY-BOARD.—*A*, the rheophore which connects it with one of the binding-posts of a galvanic battery; *B*, rheophores connecting its binding-posts with spring electrodes previously placed upon the body of the patient so as to influence the nerves or muscles to be tested; *C*, buttons and springs which make a circuit to the body of the patient when the knob on the spring is pressed downward so as to impinge upon the button. The number of rheophores which may be employed depends upon the necessities of the case; the cut shows an instrument capable of six.

entire sitting. Whenever it becomes necessary to refer to the reactions of that point, it can be called into action by touching the key connected with it by its individual rheophore. The cuts introduced show the arrangement of my device for this purpose. I have given a more complete description of the advantages of this method over others previously employed, in the *New York Medical Journal* of May 9, 1885.

Now, from such a table of record it is apparent that the *faradaic current should first be employed* upon the patient (the poles of the secondary coil being used). The extent of the overlap of this coil (in centimetres) necessary to produce muscular contractions when the nerve- and muscle-reactions are being separately tested should be recorded. In case no muscular contractions ensue, the extent of the overlap which produces

an *unbearably painful current* should be ascertained and noted. This may be compared with that necessary to produce contractions upon the healthy side.

The next step in the examination consists in *changing the rheophores to the binding-posts of a galvanic battery.* We can now ascertain the number of cells or milliampères (which is preferable) required to produce the different varieties of contractions (enumerated in the table designed for record) of muscles in homologous regions of the right and left sides. Each nerve which is impaired should be tested first; and the muscles

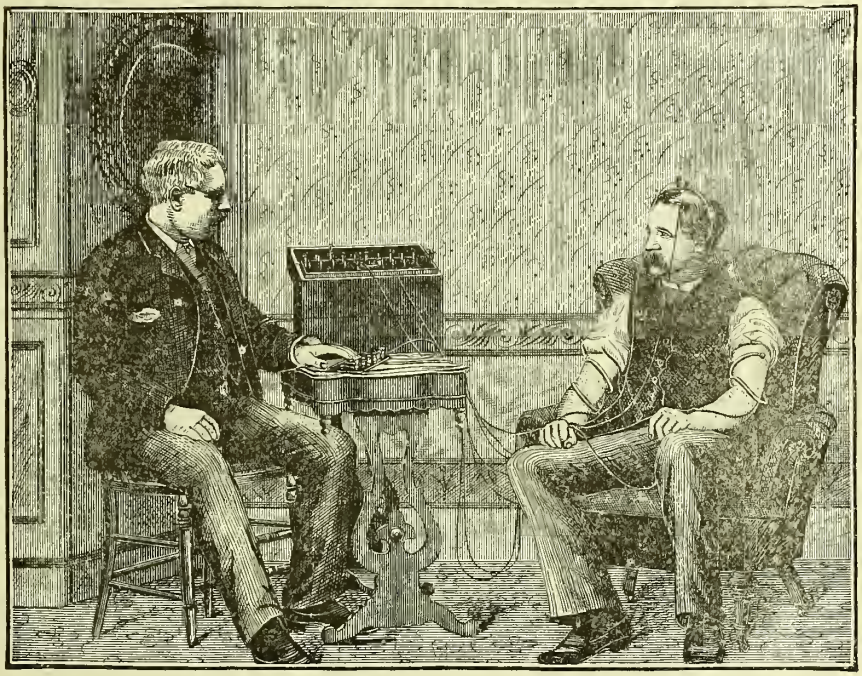


Fig. 61.—The Author's Diagnostic Key-board as Applied in Actual Use.—The spring electrodes are represented in the cut (for the purpose of illustration) as applied to the facial, ulnar, and musculo-spiral nerves of each side. If he so chooses, the operator can have his case-book on a stand at his right, for recording his observations as they are made.

supplied by it should be tested afterward. The strength of the current employed should be ascertained by throwing a galvanometer into the circuit (when extreme accuracy is desired); by so doing, a comparison of the nerve- and muscle-reactions of the two sides can be based upon conditions which are exactly alike.

When we have completed the steps indicated by the chart prepared for the assistance of the practitioner (page 191) we are in possession of certain facts which may be of great practical value as regards both diagnosis and prognosis:—

1. Suppose a case of localized paralysis is examined, and the faradaic and galvanic reactions of both a nerve and its muscles are normal and exactly alike on the two sides. We have reason then to believe that the exciting cause is either hysteria, a lesion of a higher spinal segment than that from which the nerve arises, or a lesion within the brain, provided the possibility of deception on the part of the patient respecting his paralytic condition can be excluded.

2. If the *nerve-reactions* of the affected side to both currents *are exaggerated* (*i.e.*, if the contractions occur in their proper sequence, but under a weaker current than in health), the *probability of an existing central lesion is heightened*, although hysteria may possibly still exist as the exciting cause of the paralysis.

3. If the faradaic current applied through the nerve *fails to produce contractions* of the affected muscles as readily as upon the healthy side (*i.e.*, if a stronger current is demanded to call any one of the paralyzed muscles into action indirectly through the nerve which supplies it,) then we know that the *nerve filaments within the spinal cord* or those of the *trunk of the nerve itself* are affected by a lesion which *has impaired but not entirely destroyed their usefulness.*

4. If *no current* from a faradaic machine (which can be endured by the patient) *causes muscular contractions*, we know positively that the *motor cells of the anterior horns* of that spinal segment which controls the paralyzed muscles are impaired, or that the *nerve itself has been severed* from its connection with the spinal cord, *or is undergoing degeneration.*

5. When the MUSCLE-REACTIONS to the faradaic current have been tested, the previous deductions (based on the nerve reactions) still hold good. The electrode should, however, be placed over the "motor point" of each muscle thus tested. These are shown in plates at the end of this volume.

6. If the *formulæ obtained by the galvanic current are normal*, all questions regarding the existence of degenerative changes in the nerve- or the muscle-plates can be excluded. When the *normal order is altered*, degenerative changes in the nerve- or the motor-cells of the spinal cord are present.

7. The history of a case in which *motility is impaired* is never complete without a record of an electrical examination of the nerve- and muscle-reactions to both the faradaic and galvanic current. When doubt exists respecting the existence of a cerebral lesion or hysteria, the facts obtained by other methods of examination (fully described by me in the preceding pages) will clear up all doubts.

8. Patients afflicted with *paralysis from a cerebral lesion* generally exhibit normal electro-nerve and electro-muscular reactions in the paralyzed parts. In some instances the reactions may even be exaggerated.

9. *Hysterical patients* afflicted with paralysis may exhibit either normal or exaggerated electro-muscular reactions to faradism or galvanism. The sensitiveness of the muscles to faradism is generally decreased; in some cases it may be totally wanting (Duchenne).

10. In *rheumatic paralysis* the electro-muscular contractility is, as a rule, markedly increased; this may be shown by a comparison of the reactions of the two sides of the body. In exceptional cases this is not found to be so, as I have seen the reactions follow only the strongest currents.

11. In *peripheral paralysis* the faradaic and galvanic reactions are altered after ten days have elapsed. The muscular contractility to the faradaic current is lost early to a greater or less extent; and the formula of degenerative changes is developed later by the employment of the galvanic current.

12. A decreased musculo-excitability to the faradaic current in the musculo-spiral nerve and the extensor muscles of the forearm on both sides—the flexors being normal and the lower extremities being unaffected—tells us of the approach of *lead-poisoning* before the actual symptoms are well marked.

13. In *progressive muscular atrophy* a response to the faradaic current can be obtained as long as any fibres in the muscle tested remain free from fatty metamorphosis.

14. *No alterations* in the electro-contractility of muscles is observed in any of the diseases confined to the *posterior part of the spinal cord.*

DETECTION OF FEIGNED DISEASES.

In addition to the uses of electricity for the purpose of determining the presence or absence of nerve- and muscle-degeneration, and the discrimination between cerebral and spinal paralysis and the various types of peripheral palsies, some other useful purposes in diagnosis have been published from time to time.

It is stated that muscular contractions produced by the faradaic current cannot be maintained over four hours in a dead subject.

Malingerers are not uncommon among the applicants for charitable aid, and they are occasionally encountered among the higher walks of life. Epilepsy and paralysis are the most common diseases which are feigned.

Feigned epilepsy can be distinguished by the application of a strong faradaic current to the forehead or tibia by means of a wire brush. The intense pain so produced will not be appreciated by a true epileptic, but will bring the fit to a sudden close, if assumed in order to create sympathy or aid.

Feigned motor paralysis is usually exhibited in some of the vari-

ous forms of peripheral paralysis. Few malingerers know enough to simulate hemiplegia or paraplegia without detection. If two weeks have elapsed since the attack, the presence of normal electrical reactions of nerve and muscle in the affected limb is strong ground for suspicion, provided a history of some cerebral lesion or of hysteria cannot be elicited. There are various other tests which a skilled anatomist can employ in each case that will help to clear up all doubts upon this subject.

Feigned anæsthesia may be told by the use of the faradaic current with the wire brush attached to the negative rheophore. The opposed limbs will quickly show how much actual anæsthesia exists.

DETECTION OF BULLETS OR BURIED METAL.

An ingenious application of electricity to surgery has been made which has utility in diagnosis.

The so-called "*electric probe*" consists of two wires which are perfectly insulated from each other by rubber or ivory. These wires terminate in metal tips which project slightly beyond the insulating medium, and at the other end of the wires a galvanic cell and an electro-magnet acting upon a bell are attached. When the probe is pushed into the tract made by a metallic missile, and the tips are brought into contact with it, the circuit is completed and the bell rings. The animal tissues are not sufficiently good conductors of electricity to form a circuit; hence the bell will not ring until the metal is touched by the tips of the probe.

Recent experimentation with the so-called "*induction balance*" goes to prove this instrument of great value in some cases where buried metal is suspected to exist beyond the reach of a probe.

ELECTRO-DIAGNOSIS OF AURAL DISEASES.

We owe to Brenner, of St. Petersburg, the first suggestion of this use for electric currents in diagnosis. From a somewhat limited experience in its use, I am led to believe that its utility cannot longer be called into question. Brenner's formula seems, furthermore, to be in accord with all that has been proved in respect to nerve-trunks in health, in all parts of the body. The formula is simply that of the galvanic reaction of the auditory nerve in health.

1. In place of the C. C. C. observed in muscle, we get, when a galvanic current is sent through the auditory nerve, a *ringing noise* when the *cathodal closure* occurs. (C. C. S.)

2. The *cathodal opening* produces no effect.

3. The *anodal opening produces a ringing noise* when a current of high intensity is employed.

4. After the cathodal closure (*cathodal duration*—C. D.), the ringing noise produced at the closure *gradually diminishes.*

The formula which is indicative of health when a pole is connected with each ear may be expressed in symbols, as follows:—

	Right Ear (Anode).	Left Ear (Cathode).
C. (closure)	.....	S. (loud)
O. (opening)	S. (weak)	.....
D. (duration).....	.....	S. >

Now, it is maintained by Brenner that any deviation from the normal reactions of the auditory nerve (shown in the preceding formula) indicates disease of the acoustic mechanism. The variations produced by the different diseased conditions encountered cannot be given here from want of space.

In applying galvanic currents to the ear, it is best to place a medium-sized electrode over the entire tragus or to fill the external auditory canal of the ear to be tested with tepid water containing a little salt, and then to introduce an electrode of metal inclosed in an aural speculum of hard rubber into the ear until the metal touches the water. If each ear is to be tested separately, the other electrode should be placed at an indifferent point, preferably the mastoid region of the same side or the middle of the sternum.

Regarding this test, it is well to state that repeated sittings are often necessary. The patient has to become accustomed to the disagreeable effects of the current. It is desirable that you begin with very weak currents and increase the strength very gradually. As the reaction of *cathodal closure* (C. C.) is the most important, it can be intensified by previously allowing cathodal duration (C. D.) to act, or by rapidly foling C. C. by A. O.

EXAMINATION OF THE EYE, NOSE, AND MOUTH BY ELECTRICITY.

Special effects are produced upon the organs of sight, smell, and taste by electric currents. Sparks or vivid flashes of light follow electric stimulation of the eye; and subjective odors and tastes are also produced when the olfactory and gustatory nerves are excited by this agent. The "polar method" of examination is employed when any of the special senses are thus tested. I would caution the reader against employing too strong currents about any of the organs of special sense. The examination of the optic, olfactory, and gustatory nerves requires experience and should not be attempted by novices.

When examining the *reactions of the eye*, the small electrode is placed upon the closed eyelid, temple or forehead. The large electrode is placed upon the back of the neck. The room should be darkened and the patient should keep both eyes closed.

When *testing the sense of taste*, the poles should be in contact with the cheeks, and the sensations of taste experienced upon both sides by the patient should be ascertained. A fine electrode can also be placed upon the tongue, the pharynx, or the inside of the cheek, in case localized polar reactions are to be determined. A double electrode, with two metal tips which are not in contact, may be employed for this purpose (Neumann).

ELECTRICAL EXAMINATION OF THE SENSIBILITY OF THE SKIN.

The electrode devised by Erb is, to my mind, the best for this purpose. It consists of four hundred varnished wires in a tube of hard rubber. The ends of these wires make a perfectly smooth surface. This electrode is connected with the secondary coil of a faradaic machine and

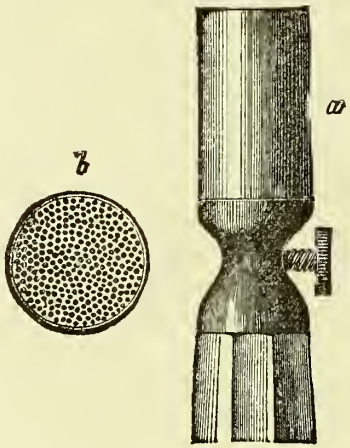


FIG. 62.—ERB'S ELECTRODE FOR THE EXAMINATION OF FARADO-CUTANEOUS SENSIBILITY.—*a*, A hard-rubber tube; *b*, the free surface of the electrode.

is then pressed upon the area of the body to be tested—the other pole being at the sternum. The *minimum* of the overlap of the secondary coil which can be felt, and the *maximum* which can be endured, are both recorded. Homologous parts of each side should be compared with each other.

Regarding this test Erb wisely remarks: "The skin, regarded as a sensory organ, cannot be tested with irritants other than those adequate to it,—viz., touch, pressure, various temperatures, and the higher grades of those irritants which produce pain. It may be disputed whether electricity should be included among these 'adequate' irritants of the skin. The electric sensation is a specific, distinct quality of tegumentary sensibility, whose careful examination, however, is of value in many morbid conditions."

TESTS TO DETERMINE THE CONDITION OF THE SENSORY NERVES.

Before completing a diagnosis of some forms of nervous disease, it is necessary to investigate the following functions: 1, The condition of the sensory nerves of the skin in respect to the sense of touch; 2, the appreciation by these nerves of varying degrees of temperature; 3, the appreciation by the patient of painful impressions transmitted to the brain by the nerves; and 4, the condition of the special senses of sight, smell, hearing, and taste.

TESTS FOR TACTILE SENSIBILITY.

In this series of tests, as also in those employed to detect abnormalities of appreciation of different degrees of temperature, the following precautions must be taken against error in the results obtained:—

1. The *nature of the tests* to be employed must be clearly explained to the patient, as well as the importance of accuracy in his decision respecting the sensations perceived. This insures his intelligent coöperation, and makes the patient more earnest in his endeavors to answer correctly.

Fig. 63.—Beard's Piesmeter.—This instrument consists of a spring in a tube that resists pressure made upon the piston. A scale indicates the amount of pressure upon the spring. It is employed to determine the degree of sensitiveness to pressure in different parts of the body The forehead, tongue, and cheek are the most sensitive to pressure; the least so are the backs of the thighs and legs.

2. It is preferable that the *patient be blindfolded*, in order to avoid any information respecting the tests used reaching him by sight.

3. To make the patient keenly alert to avoid errors of statement, it is well to employ *blank experiments* from time to time. Thus, when the skin has not been touched with any instrument or foreign substance, it is well to ask "where the object is now felt," "how many points are now in contact with the skin," etc.

Having explained the objects of the tests about to be employed and then blindfolded the patient, the tactile sensibility of the skin should be first determined by the following methods:—

(1) *Consciousness of simple contact impressions.* When the skin is brushed by a hair or a fine feather, notice, first, if the patient perceives the contact immediately, and, secondly, if he can describe the sensation correctly.

(2) *The ability to locate contact impressions.* With tests of decreasing delicacy (the touch of a hair being the most delicate, and painful impressions the least so), notice to what extent the patient is able to

correctly designate the point of contact of the body employed with the skin of different localities.

(3) *The degree of sensibility of different regions.* This has to be investigated with great care in some cases. Several methods are employed to determine it with accuracy. These are as follows:—

(*a*) Objects of *different shapes* may be laid upon the skin and the patient requested to describe their form and character. Coins, keys, and weights may be employed for this purpose, as they are always to be had. This test should be used over many parts of the body, and the results obtained compared with those of similar experiments made by the physician upon himself or some healthy person.

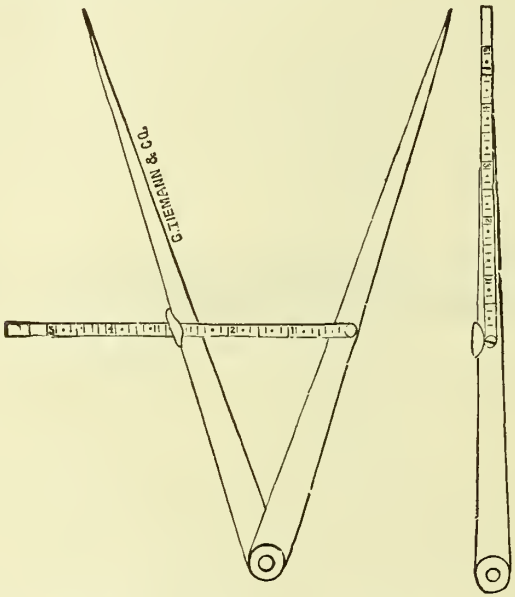


Fig. 64.—Hammond's Æsthesiometer.—When closed it can be conveniently carried in the pocket.

(*b*) The *appreciation of pressure*, as suggested by Weber, may be tested by placing weights of varying sizes upon the skin of some part, that has previously been supported in order to avoid the so-called "muscular sense" being a factor in the patient's decision. Dr. Beard has devised an instrument for this test that answers all purposes very well.

(*c*) Again, the various forms of æsthesiometers are used to detect the minimum distance which can exist between two points of simple contact with the skin without destroying the distinct perception of both points by the patient. This distance varies in health between extremely wide limits, because some regions are abundantly supplied with sensory

nerves and tactile corpuscles, while others are not. For this reason, the following measures* can be used as the healthy standard for comparison in any given case. They are given in inches, lines, and millimètres so as to meet the requirements of any scale:—

1.	Point of tongue	$\frac{1}{24}$	inch	= $\frac{1}{2}$	line	= 1.1	m
2.	Palmar surface of finger tips .	$\frac{1}{12}$	"	= 1	"	= 2.2	"
3.	Mucous surface of lips	$\frac{1}{6}$	"	= 2	lines	= 4.2	"
4.	Palm of hand and tip of nose .	$\frac{1}{4}$	"	= 3	"	= 6.3	"
5.	White part of lips	$\frac{1}{3}$	"	= 4	"	= 8.4	"
6.	Lower part of forehead . . .	$\frac{5}{6}$	"	= 10	"	= 21.1	"
7.	Back of hand	$1\frac{1}{6}$	"	= 14	"	= 29.2	"
8.	Dorsum of foot	$1\frac{1}{2}$	"	= 18	"	= 37.5	"
9.	Forearm	$1\frac{3}{5}$	"	= 19	"	= 39.6	"
10.	Sternum	$1\frac{4}{5}$	"	= 21	"	= 44.1	"
11.	Middle of thigh	$2\frac{1}{2}$	inches	= 30	"	= 62.5	"
12.	Back	$2\frac{3}{5}$	"	= 31	"	= 66.0	"

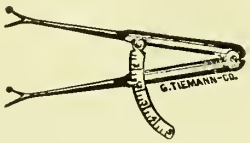


FIG. 65.—CARROLL'S ÆSTHESIOMETER.—The instrument has two points upon each leg of the compass, one blunt and the other sharp. It is a convenient instrument to determine the condition of the sensory nerves in respect to contact sensations and those of pain. This is accomplished by simply substituting the blunt for the sharp points, or *vice versa*.

Various forms of æsthesiometers have been devised, but a simple pair of compasses, such as are used by artists, will answer all purposes.

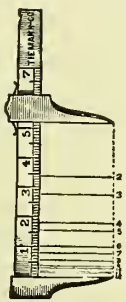


FIG. 66.—SIEVEKING'S ÆSTHESIOMETER.—A modification of the ordinary beam compass employed by carpenters, but graded in inches and tenths of an inch. Its points are not sharp.

The distance between the points can be ascertained by a rule graded in inches, lines, or millimètres. The *points should not be sharp*, as they will cause pain if so, and thus defeat the object of this test.

* More complete tables than the one offered may be found in many of the later works on physiology and nervous diseases.

The suggestions previously made respecting the definite instructions to the patient, the use of blank experiments, and the employment of a bandage over the patient's eyes, apply to this test as well as to those previously described.

The following rules must be observed in case the æsthesiometer is to be used:—

1. The *two points of the instrument must be made to touch the skin simultaneously;* otherwise the patient will detect the two points of contact more readily than if both meet the skin at the same moment.

2. The *contact should be a gentle one;* otherwise the impression upon the skin becomes a painful sensation.

3. The *relative position of the two points should always bear the same relation to the axis of the limb or median line of the body*, because the sensibility of a part is affected differently when the points are directed transversely or longitudinally. This is essential to the accurate comparison of the sensibility of different regions of the body, or of corresponding regions of either side.

4. The table which has been previously given should be employed as a standard of comparison *only when the sensory functions of the skin are impaired upon both sides.* When the derangement is one-sided, the *healthy side* will be the safest guide for comparison.

ABNORMAL CONDITIONS OF SENSATION.

We are now prepared to consider the significance of the disorders of cutaneous sensibility, viz., anæsthesia or loss of sensibility; hyperæsthesia, or increased sensibility; the existence of pain; and the lack of appreciation of varying degrees of temperature.

Anæsthesia.—Certain regions of the body may be deprived of cutaneous sensibility (either totally or partially) (1) by diseased conditions of the brain or spinal cord, and (2) by any abnormal state of the nerves themselves that tends to impair or destroy their ability to conduct sensations to the nerve centres.

In the latter case, the loss of sensation is liable to be associated with an impairment also of motion, because the cerebro-spinal nerves are composed, as a rule, of both motor and sensory fibres. The fact that sympathetic nerve fibres are also present in the majority of nerves, helps us to explain certain disorders in the nutrition of the skin that sometimes accompany motor or sensory paralysis dependent on injury or destruction of some individual nerve.

The regions of the spinal cord and brain that are *functionally associated with sensation*, have been already touched upon in Section I. It may be stated in a general way that the nerve fibres that conduct sensory impressions from the peripheral parts of the body to the brain travel

chiefly through the *posterior columns* of the spinal cord and *its gray matter* in order to reach the brain—the seat of intelligent perception of such sensations. Within the substance of the brain itself, these fibres pass through the outer part of the *formatio reticularis* and the *posterior part of the so-called "internal capsule"* of that organ. We are justified, I think, in drawing the following clinical deductions as regards the existence of cutaneous anæsthesia:—

1. Lesions of the *cerebral hemispheres* produce anæsthesia when they involve the *posterior one-third* of the internal capsule.

If the sensory cranial nerves are affected by such a lesion, the loss of sensation is commonly on the same side as the lesion, except in case of the optic nerve (the condition known as hemianopsia). The anæsthesia of parts below the head, if due to cerebral causes, is confined to the side opposite to the hemisphere in which the lesion exists.

2. Anæsthesia from lesions of *one lateral half* of the substance of the spinal cord exists, as a rule, on the side opposite to the spinal lesion.

3. Lesions which involve *both lateral halves* of the spinal cord create anæsthesia on both sides of the body, provided the destructive process affects the so-called "sensory tract" of the cord, viz., the posterior columns, or the gray matter around its central canal.

4. Anæsthesia may exist *on the same side as a spinal lesion*, provided the posterior roots of the spinal nerves be pressed upon or destroyed by it, or in case the sensory nerves be affected by the spinal lesion before they cross to the opposite side of the cord.

5. Anæsthesia, unlike motor paralysis, is not necessarily present in parts of the body supplied by those nerves that are given off from the cord below the seat of the lesion. Anæsthesia is often associated with a condition of increased sensibility or "hyperæsthesia" of parts below the seat of the spinal lesion, and on the side opposite to it.

6. Anæsthesia may often co-exist with other sensory symptoms, such as pain, incoördination of movement, the peculiar sensation known as "formication," numbness, tingling, and other subjective sensations.

7. Anæsthesia of spinal origin is *generally bilateral and symmetrical*, because lesions of the cord commonly affect both lateral halves.

8. Tactile sensibility may be destroyed by spinal lesions, and yet the sensibility to pain and temperature may occasionally be retained.

9. Unilateral anæsthesia of the face and of the opposed arm and leg indicates a unilateral *lesion of the formatio reticularis.*

In rare cases, sensibility to temperature may be lost, and the sensibility to pain and touch may be normal. It is not extremely infrequent for the neurologist to record an absence of sensibility to pain, when tactile sensibility remains unaffected, and accurate perceptions of tem-

perature are still experienced by the patient. These subjects can detect a needle thrust into the muscles from a simple sensation of touch. These clinical facts seem to confirm the view that has been advanced by late physiologists,* viz., that the paths of conduction of sensations of touch, pain, and temperature probably lie in different parts of the spinal cord.

Hyperæsthesia.—The skin may be rendered extremely sensitive in certain diseased conditions. This abnormal state of the nerves is termed "hyperæsthesia" in contradistinction to "anæsthesia" or a loss of sensation.

When the "sensory tracts" of the spinal cord are involved by a localized lesion, the *parts below the regions that are rendered anæsthetic* by the cutting of the sensory nerves are sometimes affected with *hyperæsthesia.* The cause of this is not yet definitely known.

A narrow band of hyperæsthesia is also developed, as a rule, at the upper level of the spinal lesion. If in the dorsal region, this zone of hyperæsthesia generally encircles the body. When in the lumbar region, it is more or less vertical over the limbs in accordance with the particular spinal segment which happens to be affected.

Hyperæsthesia probably indicates, according to our present knowledge, some *irritation of the nerve fibres* distributed to the regions so affected. The cut introduced is admirably adapted to illustrate the effects of a one-sided spinal lesion upon the sensory functions of the skin.

In the disease known as locomotor ataxia, after a paroxysm of "stabbing pains" has subsided, the seat of previous pain becomes markedly sensitive to the touch, while the rest of the body is not similarly affected.

Hyperæsthesia may be of service in diagnosis. It may afford valuable information respecting the spinal segments that are *irritated* by some destructive process within adjacent regions of the spinal cord. Again,

* The *lateral* columns (Fig. 34) and the *posterior* columns are probably concerned (as well as the gray substance of the cord in the region of its central canal) in the transmission of sensory impressions to the brain.

Woroschiloff, Ludwig, and Ott have apparently demonstrated by careful and apparently conclusive experiments that, in the lower animals, the *lateral columns* in the dorsal region of the spinal cord are physiologically associated with the transmission of sensations from the legs. Whether this is true of man is not yet determined, although Gower's reported case of a crushed cord in man gave evidence of ascending degeneration, both in the postero-internal columns (Goll's columns) and also in the lateral columns in front of the "crossed pyramidal tract" (Fig. 29). Unfortunately, this case stands alone as yet.

This view is directly opposed to the older one that has been generally accepted by standard authors, viz., that sensations of pain travel along the gray matter of the cord, and those of touch, and perhaps of temperature, pass up the posterior columns of the spinal cord.

The late researches of Starr seem to prove that impressions of *muscular sense* from the upper limbs are transmitted by Burdach's column, and from the lower limbs by Goll's column of the corresponding side of the cord.

if limited to the area of distribution of some individual nerve, it may point most suggestively toward the existence of some local cause of irritation of that nerve itself. Finally, Valleix has pointed out the situation of certain regions in the course of the more important nerves of the body where extreme sensitiveness to pressure or touch exists in con-

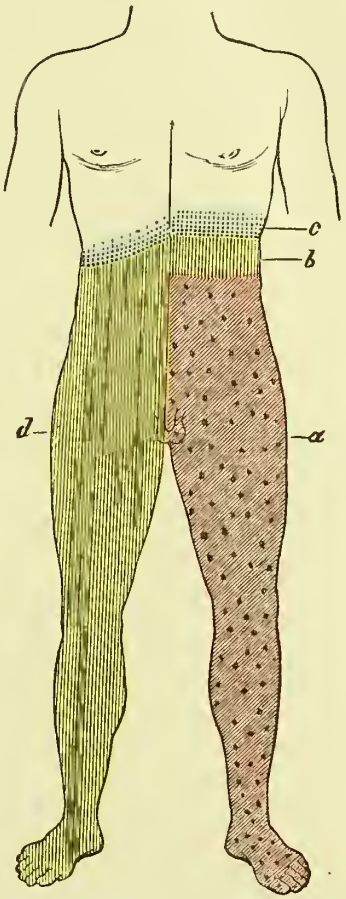


FIG. 67.—DIAGRAMMATIC REPRESENTATION OF THE SKIN SYMPTOMS IN UNILATERAL LESION OF THE DORSAL PORTION OF THE SPINAL CORD ON THE LEFT SIDE. (After Erb.) The diagonal shading (*a*) signifies motor and vaso-motor paralysis; the vertical shading (*d* and *b*) signifies anæsthesia of the skin; the dotted shading (*c*) indicates the hyperæsthesia of the skin.

nection with neuralgic attacks. These are known as the "puncta dolorosa." They have been separately described by the author in his work entitled "The Applied Anatomy of the Nervous System."

Hyperæsthesia may be functional or organic. If *functional*, it is often due to some form of general spinal irritation; if *organic*, it is com-

monly associated with more or less anæsthesia. We meet the organic variety chiefly in connection with spinal meningitis, compression of the sensory nerve roots, and locomotor ataxia.

Delayed Sensation.—To the beginner in medicine as well as to the laity, nothing strikes the intelligence so forcibly as this symptom when well marked. Imagine a patient stuck with a pin, when unaware of its occurrence, and an interval of time (varying from one to thirty seconds) to elapse without any consciousness of the wound. Imagine the patient then suddenly becoming conscious of the injury with all the evidences of pain that should have occurred without any perceptible interval of time in a healthy subject. This is delayed sensation. It occurs chiefly in connection with the disease known as "locomotor ataxia."

This symptom is to be interpreted as an evidence of imperfect conduction of sensation to the brain by means of the sensory nerves and the so-called "sensory tracts" of the spinal cord. The sensation is not arrested "in toto;" it is simply delayed. Complete abolition of sensation or "anæsthesia" is liable to be developed later—when the nerves or sensory tracts are so extensively involved as to be no longer able to perform the functions.

SENSIBILITY TO TEMPERATURE.

In testing this variety of sensibility, the precautionary steps previously mentioned in connection with sensory disturbances must be carefully observed.

Test-tubes holding water of different degrees of temperature are then applied to the different regions of the body which have given previous evidences of sensory disturbances, and the patient's ability to discriminate between them with accuracy should be noted. The temperature of the test-tubes should be greater or less than that of the skin ($98\frac{1}{2}^{\circ}$) and of a uniform size. This prevents the confusion of simple "tactile" sensations with those of temperature. Breathing upon the surface of the patient answers as a rough test for the appreciation of heat.

SENSIBILITY TO PAIN.

The tests for this variety of sensibility comprise (1) pinching or pricking of the skin; (2) the application of extreme heat to the skin; and (3) the use of a powerful faradaic current upon the skin with dry electrodes. The patient *should never be prepared for this test*, as he may fail to give external evidences of pain from an assumed fortitude. Sensitiveness to pain and temperature may sometimes be affected when tactile sensations are not impaired.

THE SPECIAL SENSES.

These comprise smell, sight, hearing, taste, and touch. The latter has already been discussed, and the tests employed to detect abnormalities of the eye or its muscles have been quite fully described.

Smell.—The abolition of smell, or "*anosmia*," is to be detected by the following methods: (1) Use the same test upon the nostrils alternately; (2) avoid all irritating substances, such as ammonia, acetic acid, snuff, etc.; (3) employ both agreeable and disagreeable odors (cologne, camphor, musk, etc., on the one hand, and valerian, turpentine, asafœtida, sulphuretted hydrogen, etc., on the other); (4) employ odoriferous sub-

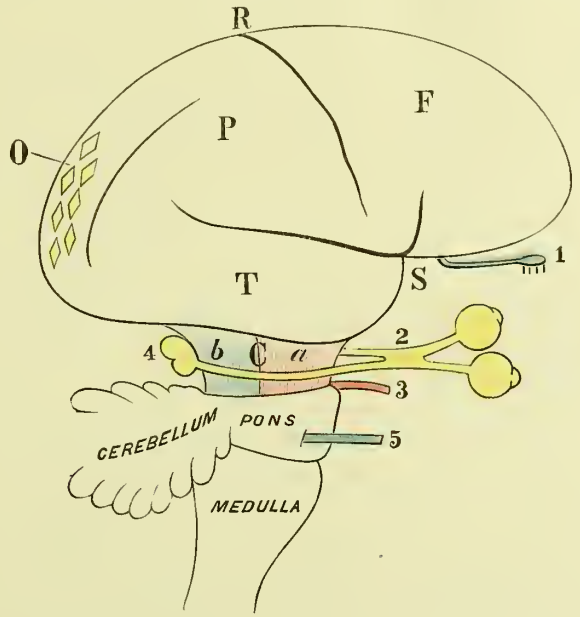


Fig. 68.—A Diagram Designed by the Author to Show Some of the Relations of the Optic and Olfactory Nerve Fibres to Surrounding Parts. F, Frontal lobes of cerebrum; P, parietal lobe; T, temporo sphenoidal lobe; S, fissure of Sylvius; R, fissure of Rolando; O, occipital lobe; C, cerebellum; M, medulla oblongata; 1, corpora quadrigemina; 2, optic tracts; 3, optic chiasm; 4, optic nerves; 5, olfactory nerve; 6, motor-oculi nerve; 7, trigeminus nerve; *a*, basis cruris; *b*, tegmentum cruris. The diamonds in the occipital lobe represent the *cortical visual centres of Munk*. The cerebellum and pons Varolii are shown as if separated from the cerebrum, in order to make the relations of the crus to the optic tracts apparent.

stances on the tongue (coffee, wines, cheese, etc.), so that the nose may perceive them by means of the throat, rather as imaginary taste perceptions than as true olfactory impressions.

The abnormal acuteness of smell, or "*hyperosmia*," may indicate brain disease that creates irritation of the olfactory nerve. Nauseating

odors to the healthy subject may become agreeable to such patients. Pleasant odors, such as those of flowers, may cause nausea, headache, or possibly convulsions.

Anosmia has been observed to accompany a congenital defect in the olfactory nerve, Bell's paralysis, tumors at the base of the brain, absence of the pituitary body, syphilitic disease of the nose, hysteria, insanity, paralysis of the fifth cranial nerve, meningitis, typhoid fever, injuries to the nose or skull, and nasal catarrh.

Hyperosmia is commonly met with during convalescence from some exhausting disease, and in connection with hysteria, insanity, meningitis, tumors of the frontal lobes, softening of the brain, epilepsy, and adhesions of the olfactory bulbs to the dura mater.

Sight.—In connection with vision, in addition to errors of refraction and accommodation, and the condition known as "ocular insufficiency" (which have been already discussed at some length), the neurologist is chiefly called upon to detect the following conditions: (1) Paralysis of the eye muscles; (2) the Robertson pupil; (3) the condition known as "hemianopsia," or, less correctly, "hemiopia;" (4) the condition of the retina known as "choked disk;" (5) the conditions known as "amblyopia" and "amaurosis."

Paralysis of the Eye Muscles.—The attitudes assumed by the patient as a result of defective power in some of its muscles have been discussed in the second portion of this chapter.

Hemianopsia.—This condition is characterized by a *blindness of one lateral half of each eye;* the unaffected half of each eye retains its power of sight. The forms of this condition that are observed, and the tests employed to detect it, have been referred to already.

Choked disk.—This condition is also known as "neuro-retinitis," because the optic nerve and retina both participate in the changes that ensue. It has been discussed already (page 150).

Robertson's Pupil.—This condition is characterized by *extremely small* pupils that contract for the focusing of vision upon near objects (within a radius of twenty feet), but do not respond to varying degrees of light. The tests employed to determine this point have been previously mentioned in the second section of this chapter.

Amblyopia and Amaurosis.—These terms are commonly used to cover all the various conditions of blindness where no organic changes in the eye itself can be seen to account for them. The term "amblyopia" is frequently used to denote a mild degree of "amaurosis."

The more common causes of these two conditions comprise (1) poisons, such as lead, tobacco, and urea; (2) exposure to a prolonged glare, as in snow-blindness; (3) concussion of the eye; (4) irritation of the fifth cranial nerve, as in severe neuralgia; (5) certain brain diseases.

The latter are of special interest in this connection. Several diagrams incorporated in this work may prove of aid in explaining certain anatomical points that bear directly upon the subject.

The following diagram (Fig. 69) shows that the optic nerve fibres eventually pass to those regions of the gray matter on the surface of the brain (the cerebral cortex) that are associated with the intelligent perception of the images focused upon the retina. But it will be also observed that the optic nerve fibres (*a* and *b*) first pass through certain collections of gray matter or "centres" within the optic thalami and the corpora quadrigemina before they radiate to the so-called "visual area" of the convolutions.

Let us now compare this diagram with another (Fig. 21), which will make some of these statements more intelligible to the general reader.

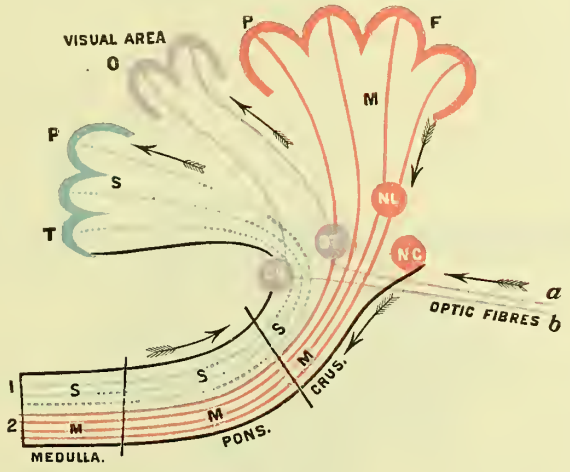


FIG. 69.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE GENERAL COURSE OF FIBRES IN THE "SENSORY" AND "MOTOR TRACTS" AND THEIR RELATION TO CERTAIN FASCICULI OF THE OPTIC NERVE TRACTS. (Modified from Seguin.) S, Sensory tract in posterior region of mesocephalon, extending to O and T, occipital and temporal lobes of hemispheres: M, motor tract in basis cruris, extending to P and F, parietal and (part of) frontal lobes of hemispheres; C Q, corpus quadrigeminum; O T, optic thalamus; N L, nucleus lenticularis; N C, nucleus caudatus; 1, the fibres forming the "tegmentum cruris" (Meynert); 2, the fibres forming the "basis cruris" (Meynert); *a*, fibres of the optic nerve which become associated with the "optic centre" in the optic thalamus, and are subsequently prolonged to the "visual area" of the convolutions of the cerebrum; *b*, optic fibres which join the cells of the "corpora quadrigemina," and are then prolonged to the visual area of the cerebral cortex.

It will help to explain why it is that pressure upon the optic tracts, as they are called, causes hemianopsia or blindness of one lateral half of each retina.

TASTE.—This special sense is presided over by the gustatory branch of the fifth cranial nerve, the glosso-pharyngeal nerve, and the chorda tympani branch of the facial nerve. Taste may be affected, therefore, by any diseased condition that can cause either irritation or destruction of

the fibres of these nerves. Certain functional diseases, in contradistinction to organic lesions of the brain, may also cause modifications of taste.

An abnormal sensitiveness of taste is known as "*hyperguesia.*" It may be developed in connection with hysteria; with melancholia and some other types of insanity; and with facial paralysis of rheumatic origin. Such subjects can often detect extremely small quantities of sapid substances in solution, which in health would be unperceived. They may perceive gustatory sensations when the electric current is applied over the spine in the region of the neck or upper dorsal vertebræ. They may develop a loathing of certain dishes which have previously been their delight, from some imaginary taste of a disagreeable character. Again, this condition may express itself in an unnatural enjoyment of food. Finally, sweetish, sapid, or sour tastes within the mouth may be constantly present.

A loss of the sense of taste is known as "*aguesia.*" It may be complete or partial. Some regions of the tongue may be affected, and others retain the sense of taste. In some instances, the tongue may be sensible to certain substances, and insensible to others. It may be associated

Fig. 70.—Seguin's Surface Thermometer.

with a sense of burning and bitterness within the mouth, as in a case reported by Böttcher, where a tumor at the base of the brain was its exciting cause.

This abnormal state has been observed to follow the development of tumors of the brain or its coverings; paralysis of the fifth cranial nerve; sclerosis of the medulla oblongata; injuries to the glosso-pharyngeal nerves; atrophy of the nerves associated with taste; and ear disease causing pressure upon the chorda tympani branch of the facial nerve.

Hearing.—The mechanism of the ear is so complicated that defects in hearing are commonly due to some abnormal condition of the apparatus itself, rather than of the nerve of hearing or the brain. Perhaps the most reliable test to determine the presence of the latter condition is the employment of the tuning-fork. If this instrument be set in vibration and applied to the teeth, or the bones of the skull, the transmission of the sound-waves through the bones will enable them to reach the nerve filaments of the internal ear, and afford the patient perceptions of sound. If the patient is unable to perceive sound when thus conducted to the nerve filaments, it is strongly suggestive of some diseased condition within the cavity of the skull.

CEREBRAL THERMOMETRY.

Within a few years much attention has been given to the temperature of limited portions of the skull in health and disease (Broca, Hammond, Seguin, Amidon, Gray, and others). Many forms of instruments may be employed for this purpose. Probably the simplest and least expensive is the surface thermometer devised by Seguin, which has a large flattened bulb well adapted to insure close contact with the scalp. Any number of such thermometers may be fastened to a shaven scalp by means of perforated straps (Gray) or an India-rubber cap similarly perforated. The effect of the temperature of the air upon the mercury may be avoided by coating the parts not in contact with the scalp with shellac.

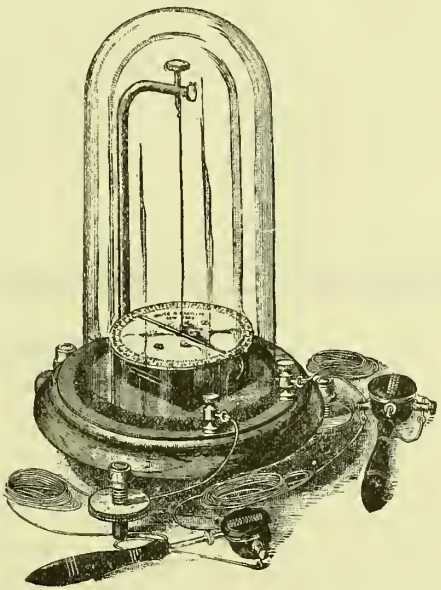


FIG. 71.—THERMO-ELECTRIC DIFFERENTIAL CALORIMETER.—Connect the two thermostats as shown in figure, viz.: connect by means of one of the metal tipped cords one binding-post of each of the thermo-piles to the two binding-posts on base of the galvanometer. Then connect the two remaining posts, one on each of the thermo-piles with each other. After so doing, place the thumb on the face of one of the thermo-piles and observe the direction of the deflection of the galvanometer needle, then place thumb on face of the other thermo-pile, leaving the first uncovered, and, if the deflection is in the opposite direction to that first obtained, the instruments are properly connected. If, however, the second deflection is in same direction as obtained by pressing thumb on first thermo-pile, disconnect the two cords from either thermo-pile and interchange them, viz.: take cord from right-hand post and place in left, and cord from left post and place in right-hand post: the deflections will then be as first alluded to, one pile turning needle in one direction and the other in the opposite direction.

More delicate tests of temperature may be obtained by the thermo-electric calorimeter devised by Lombard. One or two minutes is only required by this instrument to detect variations in the temperature of homologous regions of the scalp, but it is expensive and only available for use in the office.

It is essential that two thermometers at least be employed when the thermometry of the scalp is being tested, in order that the temperature of homologous parts of the two hemispheres may be simultaneously taken, thus insuring the same conditions of vascular supply. A comparison should always be made between the results so obtained, before any clinical deductions can be drawn from them.

The temperature of the scalp seems to be somewhat below the normal standard of health (98.5° Fahr.) in all of its parts.

Amidon has shown that willed muscular movements if continued for some time are associated with an increase of heat over the cortical centres which are called into action. He has thus confirmed some of the deductions obtained by physiological experiments upon animals. Gray and Mills report the diagnosis of a tumor of the brain by the detection of a localized elevation of temperature over the area involved. The difference between the healthy and unhealthy side was about one degree and a half. Hamilton reports a case where a difference of three degrees existed, and persisted at repeated examinations; the case was living at the date of this statement, so that the diagnosis of tumor had not been posi-

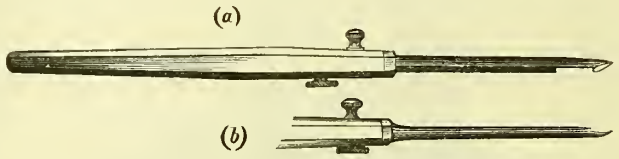


FIG. 72.—DUCHENNE'S TROCHAR. *a*, Open; *b*, closed.

tively verified. From my own experience, I am led to believe that an unilateral deviation of one and a half or two degrees above or below the normal point, within a circumscribed area of the scalp, which is persistent and unattended with as marked a rise or fall in temperature in adjacent areas, must be regarded as a valuable diagnostic symptom of disease within that area. If it be a cerebral tumor, I should expect to find by the ophthalmoscope the characteristic evidences of neuro-retinitis, known as the "choked disk."

Sometimes it is very important to decide as to the existence of organic changes in the muscular tissue of different parts of the body. By means of this very ingenious and useful instrument we are enabled to extract with little pain, and no danger, small pieces of any muscle which can be examined microscopically at your leisure. This instrument is introduced (with the slide open) into the substance of the muscle; subsequently the slide is closed and the instrument is then withdrawn. A small piece of the muscle will be found to have been removed and retained within the instrument.

In closing this chapter, the author feels that he has perhaps overtaxed the patience of his readers. If he has erred in this direction, it is because he has endeavored to cover a large field within the limited compass of a single chapter, and to so interpret the symptoms of nervous diseases as to bring them within the grasp of the general practitioner of medicine. It must not be inferred that all of the tests described are of necessity demanded in each individual case that is brought to the notice of the neurologist. As Gower happily remarks, "To know our enemy is, if not 'half the battle,' at least an important part of it." When once the symptoms of nervous diseases have been thoroughly mastered, the special lines of investigation demanded in each case become as clearly defined as does the course of his vessel to the mariner, to reach the harbor for which he steers. This chapter gives but the rough outlines of a chart in which the short cuts to valuable information in diagnosis are imperfectly jotted. Arduous and persistent labor on the part of each of its followers can alone fill in its details and render it complete.

SECTION III.

DISEASES OF THE BRAIN AND ITS ENVELOPES.

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Diseases of the brain are, perhaps, in many cases, the most difficult to recognize with certainty during life of any that the physician encounters. The results of autopsies go to prove that serious errors in diagnosis are too often made in connection with cerebral affections. In occasional instances, such errors are, perhaps, unavoidable; but many that are constantly made might unquestionably have been avoided had more study been given to the peculiar features of each disease, and greater care exercised in the clinical examination of the patient.

In the two preceding sections we have discussed at considerable length the anatomical and physiological bases of cerebral localization; and also the various tests which may be demanded from time to time during the clinical examination of a patient. We are now prepared to intelligently discuss separate diseases of the nervous system from the clinical aspect.

Too great stress cannot be laid upon the necessity of a thorough familiarity with the two preliminary sections of this work. Constant reference will be made by me to many facts and deductions incorporated and interpreted in those chapters.

The following table may aid the reader in classifying the more important diseases of the brain and its envelopes:—

Abnormal Conditions of the Brain and its Envelopes.

1. Congenital Defects or Malformations of the Brain and Cranium.	Double-head.	
	Fusion of two heads.	
	Absence of brain or head.	
	Cyclocephalic deformity.	
	Abnormalities of the cerebral envelopes.	
	Incomplete development of special ganglia.	
	Hydrocephalus.	
	Encephalocele.	
2. Diseases affecting the Vessels of the Brain.	Aneurisms.	
	Atheroma.	
	Arterial thrombosis,	Affecting sinuses and large vessels. / Capillary variety.
	Venous thrombosis.	
	Embolism.	
	Aphasia.	
	Cerebral hyperæmia,	Active variety. / Passive variety.
	Cerebral anæmia,	Localized variety. / General variety.
	Apoplexy—Cerebral hemorrhage.	

ABNORMAL CONDITIONS OF THE BRAIN AND ITS ENVELOPES (*continued*).

3. INFLAMMATORY CONDITIONS OF THE BRAIN.	Pachymeningitis,	Suppurative variety. Non-suppur. variety.
	Meningitis,	Acute. Sub-acute. Chronic.
	Hydrocephalus (tubercular meningitis).	Acute. Chronic.
	Encephalitis.	
4. STRUCTURAL CHANGES OF THE BRAIN-TISSUES.	Softening,	Red. White. Yellow.
	Abscess,	Acute. Chronic.
	Sclerosis,	General variety. Multiple variety. Miliary variety.
	Atrophy,	Infantile variety. Senile variety.
5. CEREBRAL TUMORS.	Carcinoma. Lipoma. Glioma. Myxoma. Papilloma. Melanoma. Sarcoma. Gummata. Psammoma. Cholesteatoma. Tubercle. Fibrous. Fibro-plastic. Parasitic.	

CONGENITAL MALFORMATIONS OR DEFECTS OF DEVELOPMENT.*

Defects in development of the central nervous system (which are occasionally observed in connection with congenital malformations) are being studied to-day with great interest, both by anatomists and physiologists. Sections of such brains, when properly prepared and stained, are often very instructive, because they shed more or less light upon the course and connections of tracts of fibres whose functions are in many cases more or less imperfectly understood.

The following paragraphs relate to those defects which may be observed in the human brain as a result of imperfect development:—

Two heads have been repeatedly met with upon the same body. They may be either distinct or joined laterally or anteriorly. They

* Portions of this chapter have been incorporated by the author in the third edition of his work, entitled, "A Practical Treatise on Surgical Diagnosis," N. Y., 1884.

may assume different sizes and shapes, and may exhibit different degrees of development.

An entire absence of the brain at birth, and, in some instances, of the spinal cord in part, has not infrequently been recorded.

The cerebrum, cerebellum, pons Varolii, medulla oblongata, and even a part of the spinal cord, may be occasionally wanting, and yet the cranial nerves may be perfectly developed.

In rare instances, the base of the skull has been exposed to view. In others, the upper cranial bones have been wanting, and the integumentary covering of the head has been found to be distended by a fluid accumulation beneath it. In some cases reported, only parts of the brain have been absent, the remaining ganglia being normally developed.

The condition termed "*Cyclocephalous*"—due to a fusion of the two orbits into one cavity—is sometimes encountered.

The meninges may be occasionally found to be incomplete. The falx and tentorium may be wholly or partially absent, or may be perforated with holes. The dura is sometimes wanting at the base of the skull.

The corpus callosum, fornix, and septum lucidum have been found wanting in the brains of some idiots. The optic nerves have sometimes no commissure.

The whole brain may be occasionally so small as to constitute the "*microcephalous state.*" Again, only certain convolutions may exhibit arrested development, and a cyst filled with serum is then found to commonly spring from the meninges and fill the space thus left vacant.

The two hemispheres of the cerebrum may present extreme variation in point of size and weight. The thalami and corpora striata may occasionally exhibit atrophy.

The brain may occasionally protrude from the cavity of the cranium at the various sutures or fontanelles,—constituting "*encephalocele.*"

Finally, the "*hydrocephalic condition*" may exist (characterized by excessive fluid outside of or within the ventricles of the brain). It usually tends to increase after birth.

The various types of congenital malformations of the nerve centres which are encountered seem, as a rule, to depend either upon some violence to the uterus or some mental shock to the mother during pregnancy.

DISEASED CONDITIONS OF THE CEREBRAL VESSELS.

Under this heading may be included the following conditions: Aneurismal dilatations; atheromatous and calcareous changes; rupture or spontaneous perforation of blood-vessels; thrombosis of the arteries

or sinuses of the brain or its coverings; embolism; fibroid degeneration; hyperæmia; anæmia; and to inflammatory changes in the coats of blood-vessels.

ANEURISMAL DILATATIONS.

The vessels within the skull most frequently affected are the internal carotid, basilar, and middle cerebral. Within the cavernous sinus, large aneurismal tumors are not uncommon. It must not be supposed, however, that the smaller vessels of the brain are exempt. Miliary aneurisms, which sometimes give to an artery and its branches an appearance resembling a bunch of grapes, frequently affect the vessels that form the circle of Willis, and even those of the pia-mater, within

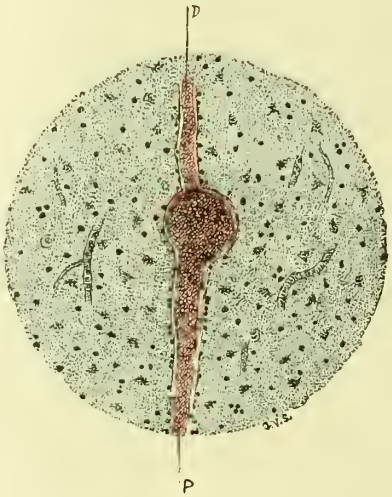


Fig. 73.—A Miliary Aneurism of the Cerebral Cortex—Anterior to Roland's Fissure (350 diameters). This beautiful drawing was kindly made from a microscopic slide by my friend Dr. G. Van Schaick, of New York. (*P*, proximal end; *D*, distal end.)

the substance of the brain and in the ventricles. The small vessels which nourish the corpora striata and the optic thalami are sometimes affected.

Miliary aneurisms of the brain frequently coexist with aneurismal tumors of larger vessels outside of the cranium; but they seem to exhibit an independence of atheroma which is quite remarkable.

Morbid Anatomy.—Those who have devoted special attention to this subject—Charcot, Zenker, Bouchard, Meynert, Hammond, and others—differ regarding the cause of these dilatations. Some regard them as due to a "sclerosis of the tunica intima" of the arterioles. Others believe that small "dissecting aneurisms" (this term covers all those types of

aneurism where the blood escapes *between the coats of an artery* for a greater or less distance) first form, because of a rupture of the inner coat. When the external coat of such an aneurism ruptures, a cerebral hemorrhage ensues. The fact that miliary aneurisms affect all ages (since even children are not exempt) seems to me to point to an "aneurismal diathesis" as an exciting cause in some instances. If such be the case, the arterial coats would exhibit under a microscopic lens marked congenital defects in their construction.

Etiology.—Among the other exciting causes of cerebral aneurism may be mentioned the cachexia of cancer; tuberculosis; uræmic poisoning; chronic alcoholism; lead poisoning; leucocythæmia; rheumatism; gout; syphilis; and general paralysis. The remarkable tendency of alcohol to excite aneurismal tendencies (not only in the brain and retina, but in other parts as well) is adduced by some authors as an argument in favor of the view that arterio-sclerosis precedes and causes the alterations in the calibre of the vessels.

In case cerebral aneurisms be of large size, atheromatous or calcareous changes within the arterial coats are seldom absent.

Symptoms.—Miliary cerebral aneurisms give rise not infrequently to headache and vertigo. Attacks of paralysis which follow one another rapidly, and from which the patient quickly recovers, may be regarded as almost a positive proof of their existence.

These small aneurisms produced their motor effects only by rupture. Minute extravasations into the brain-tissue commonly occur at first; but later on large apoplectic clots may be formed. The discussion of the symptomatology of cerebral hemorrhage will occupy subsequent pages of this volume. A previous section of this work also covers many suggestions of an anatomical and clinical nature that bear upon cerebral localization.

ATHEROMA.

The cerebral vessels may participate in a peculiar form of degenerative change, termed atheroma. When this condition is developed, vessels in other parts of the body are usually affected simultaneously.

Morbid Anatomy.—The changes observed in the cerebral vessels, when atheromatous, seem to be produced slowly.

The extent to which it affects the blood-vessels admits of large variation. In some instances every vessel named by anatomists is thus diseased, while, in others, only certain vessels, and even parts of vessels, are found to be affected. In extensively developed atheroma, a symmetrical condition is usually present on the two sides. This fact is of importance in some cases, since a guide to diagnosis may be thus afforded. Cases, which have often been reported, of parallel and contemporaneous popli-

teal aneurisms in the same person illustrate well the tendency toward a symmetrical development in the limbs.

Atheroma develops more often in the lower limbs than in the upper, and the extent of its progress seems to be greater when situated below the diaphragm than when above it.

It is a direct result of an existing chronic endarteritis, the lining membrane of the vessels being invariably involved to a greater or less degree. It is most frequently found in the arteries, although the veins may develop an atheromatous condition when exposed to any source of prolonged irritation. It is also developed in the male sex in far greater proportion than in females, and is apparently influenced to some extent by climate.

As a result of this condition the affected vessel becomes impaired in its contractile power, loses its natural tone, and, in consequence of its inability to sustain its accustomed internal pressure, undergoes, in many cases, dilatation at the seat of the disease.

Etiology.—This condition may be produced by age, chronic alcoholism, gout, rheumatism, lead poisoning, syphilis, chronic diseases of the kidney, exposure, or traumatism.

Symptoms.—When the condition of atheroma is once developed, rings of ossification are often perceptible along the course of the superficial vessels. An abnormal tortuosity of the artery is not infrequently present, if the atheromatous condition is diffused for some distance.

The existence of atheromatous changes is not always to be detected, however, even in the superficial vessels of the extremities, by the sense of touch. *Diminished arterial volume*, and an *impaired nutrition* to tissues when an excessive arterial supply is demanded (as occurs in inflammatory processes) are frequently points of value in the diagnosis of an atheromatous condition. Atheroma has especial medical importance on account of a tendency which it creates toward rupture of the affected vessels, either from an ulcerative destruction of their coats or from the rigid and brittle condition of the walls of the vessel produced by the calcareous deposits.

The dangers which result from atheromatous changes within the coats of the cerebral vessels render the detection of these changes important, even if the disease be unassociated with marked external evidences of its existence.

It must be remembered that the process of repair cannot be perfected in an artery whose lining membrane is tough or osseous, or in a state of fatty degeneration, whose middle coat has atrophied, and whose contractility is destroyed.

One important point in diagnosis in this connection has lately been

brought to professional notice, viz., that *bi-temporal hemianopsia* indicates in most cases an atheromatous change in the vessels composing the circle of Willis. This subject has been discussed lately, under the head of "Lesions affecting the Optic Nerve."

CEREBRAL THROMBOSIS.

A coagulum of blood may form in either the sinuses, veins, or arteries of the brain. Some changes in the coats of the vessels, however, usually precede and cause the development of such a coagulum. The vessels are therefore occluded gradually. This is in marked contrast to embolism, which causes a sudden occlusion of a vessel whose coats may be perfectly healthy.

Morbid Anatomy.—Thrombosis is much less common in arteries than in venous channels; but it is not uncommon in the internal carotid, the vertebral, the basilar, and the middle cerebral arteries. In the superior longitudinal and lateral sinuses thrombosis is frequently found; chiefly in connection with pachymeningitis. Old age seems to predispose to the development of cerebral thrombosis; and males are more commonly affected than females. Cerebral thrombosis in children may develop in connection with exhausting diarrhœa.

The effect of thrombosis of arteries or veins within the cavity of the cranium is to render the nutrition of surrounding parts more or less imperfect, and thus to impair the function of those parts. If it be of pyæmic origin the thrombosis may cause *suppuration* of the adjacent structures; and, by its disintegration, other vessels more or less distant from the seat of the original thrombus may become plugged by the detritus. Metastatic abscesses in the viscera are produced in this way.

Etiology.—The causes which chiefly tend to produce thrombosis include (1) *atheroma*, which produces a roughened condition of the internal coat of the blood-vessels; (2) *hyperinosis*, or that condition of the blood in which a marked excess of fibrine is present, as in some acute diseases, of which inflammatory rheumatism stands foremost; (3) *pressure upon some large vessel or sinus*, so that the circulation within it is rendered extremely slow; (4) *chronic interstitial nephritis;* (5) *syphilis;* and (6) *pyæmia*, which seems to be associated with a special tendency toward spontaneous coagulation of blood within the vessels, when the rapidity of the current is slowed.

Symptoms.—The symptoms produced by cerebral thrombosis must, of necessity, be modified by the situation of the clot and the vessel occluded by it. A knowledge of the *functions of different portions of the brain* can alone decide questions which may arise in any indi-

vidual case respecting the situation and the probable termination of the lesion. Either coma or paralysis (in any of its forms) is apt to be one of the results. It is liable to be confounded chiefly with cerebral embolism or cerebral hemorrhage. Cerebral softening, which may be one of its sequelæ, will be treated of as a separate affection in this volume.

It must be remembered that syphilis and chronic nephritis are among the most frequent of the causes of arterial thrombosis. This fact is explained by the development of an inflammatory condition of the internal coat of the vessel,—the so-called "*endarteritis obliterans.*" For this reason the history of the patient may prove an important factor in the differentiation between cerebral thrombosis and some other conditions of the brain which might be mistaken for it.

Cases where cerebral thrombosis has existed during life and yet been unrecognized by many medical men of note confirm the statement that an accurate diagnosis of this condition is a matter of extreme uncertainty in some instances.

The symptoms of the conditions are modified in every case (1) by the seat of the blood-clot, and (2) by the amount of interference with the circulation that ensues in surrounding parts. Furthermore, serious effusion may occur (in some cases) from neighboring vessels, provided the circulation in adjacent parts is rendered imperfect. This may tend to mask the symptoms of the primary condition.

To enter into detail respecting all the phenomena that may be encountered in connection with cerebral thrombosis would necessitate the discussion of the entire subject of cerebral localization.* Hints may be given, however, that may shed some light upon cases of this character. None of these suggestions are to be considered, however, as pathognomonic of this condition.

Thrombosis of the *lateral sinuses* may be attended, according to the observations of Gerhardt, with a difference in the size of the external jugular veins.

If thrombosis occurs in the *transverse sinus* a circumscribed œdema of a painful character may develop behind the ear, provided the veins leading to the sigmoid fossa are implicated.

Epistaxis may occur when the *superior longitudinal sinus* is thus plugged. A complicating œdema of the forehead and the development of exophthalmus has been reported from the same cause.

Suppuration of the ear not unfrequently occurs when the *petrosal* or *cavernous sinuses* are implicated.

A thrombus of one of the larger arteries of the brain may produce sudden anæmia of the parts nourished by its branches, and thus induce

* See closing pages of Section I.

paralysis of motion or sensation, or both. Convulsions, disturbances of the special senses, and coma may also follow. The ocular muscles may be rendered paretic, thus causing strabismus and diplopia.

Localized passive hyperæmia may be one of the effects of cerebral thrombosis, and thus give rise to a train of symptoms indicative of that condition. (See subsequent pages.)

Thrombosis *may go on to suppuration.* In such a case the symptoms of cerebral abscess or of general pyæmia may develop. In a brochure upon this subject * I reported two cases which came under my

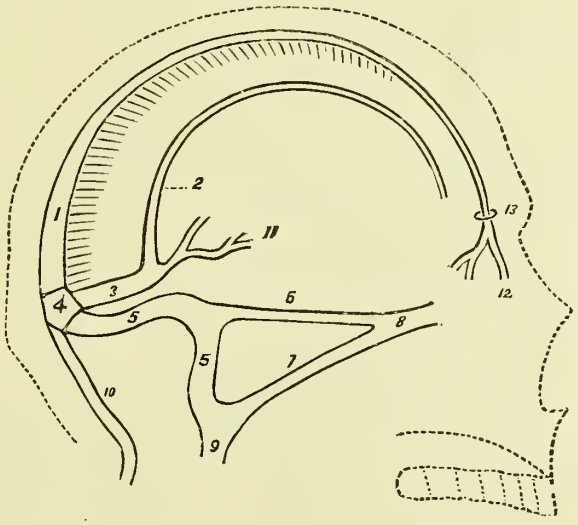


FIG. 74.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE CEREBRAL SINUSES IN PROFILE. 1, superior longitudinal sinus; 2, inferior longitudinal sinus; 3, straight sinus, deriving blood from 1 and also from the veins of Galen (11); Nos. 1, 2 and 3 bound the falx cerebri; 4, the torcular Herophili, where four sinuses meet; 5, lateral sinus; 6, superior petrosal sinus, joining the lateral sinus (5) with the cavernous sinus (8); 7, inferior petrosal sinus, joining the cavernous sinus (8) with the jugular vein (9); 8, cavernous sinus; 9, internal jugular vein formed by two sinuses (5 and 7); 10, occipital sinus; 11, venæ Galeni; 12, vein passing to nasal cavity; 13, foramen cæcum.

personal observation, where a trivial contusion of the scalp which failed to break the skin had induced thrombosis of the diploë and caused death by pyæmia. The autopsies showed extensive secondary thrombosis of the cerebral sinuses and metastatic abscesses in nearly every organ of the body.

In children exhaustive diarrhœa may induce thrombosis of the cerebral sinuses. We may expect to encounter in such a case rigidity of muscles of the neck, and sometimes of the back and limbs; the develop-

* *Pyæmia and Septicæmia.* Annals of Anatomy and Surgery, Nov., 1881. A lecture delivered by the Author before the Anatomical and Surgical Society of Brooklyn, N.Y.

ment of nystagmus, ptosis, or strabismus, facial paresis (in some cases), and somnolence, coma and collapse. It is well to state, however, that these symptoms are not pathognomonic of cerebral thrombosis in a child. They may indicate only a state of general cerebral anæmia.

Differential Diagnosis.—The probable cause of the thrombosis must be taken into consideration in connection with the symptoms presented by each case. Both may assist greatly in the diagnosis. If the throm-

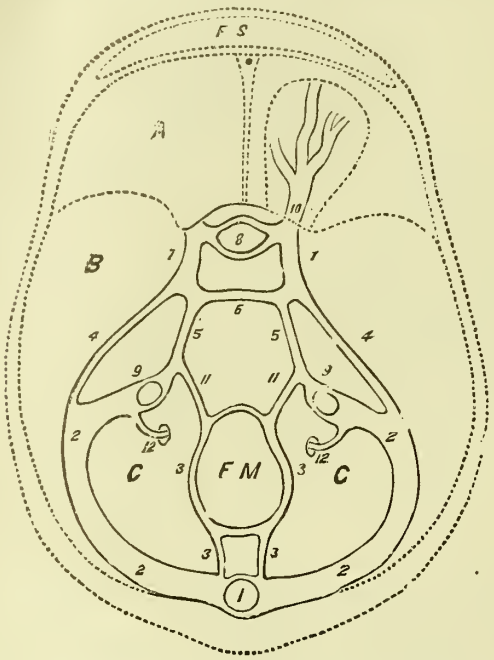


Fig. 75.—A Diagram Designed by the Author to Show the Venous Sinuses of the Dura-Mater, as Seen after a Horizontal Section through the Cranium. *A*, anterior fossa of skull; *B*, middle fossa; *C*, posterior fossa; *F S*, frontal sinus; 1, torcular Herophili; 2, 2, lateral sinuses; 3, 3, occipital sinuses; 4, 4, superior petrosal sinuses; 5, 5, inferior petrosal sinuses; 6, transverse sinus; 7, 7, cavernous sinuses; 8, circular sinus; 9, opening into internal jugular vein; 10, ophthalmic vein, communicating with cavernous sinus (7); 11, a branch joining the occipital and inferior petrosal sinuses; 12, veins from posterior condyloid foramen to lateral sinus.

bus be due to a tumor within the cranium it will perhaps be impossible to separate the symptoms of each. It is well to know that thrombosis has been known to follow rheumatism, erysipelas, a carbuncle of the neck, some of the fevers, injuries to the head, and surgical operations, in addition to the other causes mentioned on a previous page.

The following differential diagnosis is quoted with slight modifications from the author's work on "Surgical Diagnosis:"—

CEREBRAL EMBOLISM.	CEREBRAL THROMBOSIS.
Sex Affected.	
Most frequent in females.	Equally frequent in the sexes.
Onset.	
Sudden. No prodromal symptoms.	Gradual. Prodromal symptoms.
Causes.	
A history of heart-disease and the physical evidences of a valvular lesion are usually to be discovered. The embolus may spring, however, from an aneurism or a suppurating thrombus and be carried into the circulation to the brain.	Chronic nephritis. Atheroma. Syphilis. Pachymeningitis. Hyperinosis. Pyæmia. Pressure upon the veins or arteries. These morbid conditions all tend to cause a coagulation of blood within the vessels affected.
Changes in the Exterior of Skull.	
No changes in the vessels of the exterior of the skull are to be detected. The veins of the neck are symmetrical in point of size.	Epistaxis, œdema of the frontal veins, and exophthalmus may occur if the *superior longitudinal sinus* is obliterated. The external jugular veins may not be of the same size—the one on the obstructed side being the smaller—if the *lateral sinus* be occluded. Painful circumscribed œdema behind the ear may arise from a thrombus of the *transverse sinus*.
Suppurative Changes.	
Suppurative effects are sometimes produced within the brain-substance (embolic abscess), but seldom in distant parts.	*Suppuration of the ear* is very common in connection with thrombosis of the cerebral sinuses. *Abscesses in distant parts* are liable to form on account of a suppurative disintegration of the thrombi.
Convulsions.	
Convulsions are rare.	Convulsive attacks are common and may exist for months in attacks of venous thrombosis.
Paralysis.	
A sudden hemiplegia usually occurs—generally of the *right side* *Aphasia* exists in the majority of cases.	Comes on gradually, if at all, in the venous variety. It may be absent. May occur suddenly in arterial thrombosis. Aphasia may be developed, but is not the rule.
Coma.	
The patient seldom loses consciousness completely during the attack or after it.	Profound coma often follows the paralysis or accompanies the attack. In venous thrombosis it may occur without paralysis having preceded it.

Symptoms in Common.

Both commonly affect the young and early adult life.
Both may cause aphasia, and coma.
Both may cause hemiplegia, local paralysis, convulsions, etc.

Prognosis.—This disease is liable to prove fatal sooner or later. It is possible that pyæmia may be induced by it, provided the thrombus becomes disintegrated by suppuration. Emboli are thus formed. These are subsequently carried to other organs by means of the circulation. Infarctions and embolic abscesses of the lungs, liver, spleen, kidneys, etc., may be produced in this way. Softening of the brain is a frequent sequel to thrombosis. Death may occur in a few days if the cerebral anæmia is very profound.

Treatment.—No remedial measures can affect an existing thrombus. When the exciting cause can be discovered it should be removed if possible. The symptoms should be treated as they develop, with the hope that the more serious complications of the disease may not occur and the patient survive the attack.

THROMBOSIS OF THE CEREBRAL CAPILLARIES.

The capillaries of the brain may be extensively affected by small thrombi or emboli. This condition is distinct from that comprised under the common acceptation of the terms "cerebral embolism" or "cerebral thrombosis."

Morbid Anatomy.—The microscope alone can detect the points of occlusion. Atheroma may be detected. The capillary vessels will be found to contain pigment-granules, fatty masses, crystals of lime-salts, etc. The larger vessels are not atheromatous, nor are they occluded.

The brain may exhibit spots of softening or of suppuration, and the cortex of the organ is generally anæmic.

Etiology.—Pigmentary embolic occlusion of the cerebral capillaries may be induced by malarial diseases. Fatty masses may be formed at the seat of occlusion as a result of a fatty degeneration of the capillaries, or they may be swept along in the blood-current from distant foci of fatty metamorphoses in the bones, viscera, heart-cavities, etc. Pus-cells or the white blood-corpuscles may also occlude the cerebral capillaries, chiefly in connection with acute diseases, causing a marked elevation of temperature, and with leucocythæmia. Fibrinous masses may be present in the blood-vessels of the brain, especially in rheumatism and inflammatory diseases; and cancerous material may act as emboli. Finally, lime-salts may be absorbed from diseased bones (Virchow) and be carried to the brain.

Symptoms.—These are vague and not well understood. Mental disturbances are prominent, such as delirium, loss of memory, loss of emotional control, and hallucinations. Headache, nausea, dizziness, trembling of the extremities, and paresis may develop. Bastian advances the view that disturbances of the sensorium, when occurring in acute

diseases attendant with high fever, may be due to capillary occlusion and consequent anæmia of the cerebral cortex.

CEREBRAL EMBOLISM.

The term "embolus" is commonly used to designate any foreign body in a blood-vessel which floats in the blood-current, or, in case it be stationary, that has been transported by the blood from some situation more or less distant from the seat of lodgment. The term "embolism" must not be employed synonymously, therefore, with "thrombus;" because the latter term applies only to a *blood-coagulum* within a blood-vessel (unhealthy as a rule), which has been formed at the site of occlusion of the vessel, and not transported there by the blood.

Morbid Anatomy.—The most frequent seat of cerebral embolism is in the middle cerebral artery of the left side. This is because that artery forms the termination of the *most direct channel* from the heart. The axis of the left carotid is so situated in reference to the curve of the aorta as to assist the passage of floating particles in the blood into its mouth. The internal carotid is, moreover, the direct continuation of the common carotid of each side; and the middle cerebral artery is similarly placed in reference to the internal carotid after that vessel enters the skull.

Next in point of frequency comes the right Sylvian artery. This is because the innominate artery, although much larger than the left carotid, leaves the aorta at an angle opposed to the current of blood in that vessel. The middle cerebral artery nourishes, in addition to other convolutions, the so-called "speech area" of the cerebral cortex; hence plugging of the main trunk of that vessel deprives those convolutions of blood that are physiologically concerned in the coördinated movements of articulate speech. The motor speech area is shown in Fig. 5.

Cerebral embolism is a frequent cause of *extravasation of blood*, because it tends to induce infarction. Again, it may result in localized softening of the parts that are imperfectly nourished, provided the embolus is large. If the embolus is of suppurative origin the parts in which the nutrition is impaired by the embolus suppurate, and a so-called "embolic abscess" results.

An *infarction* is a wedge-shaped spot of consolidation and discoloration within an organ dependent upon occlusion of a blood-vessel and the subsequent rupture of neighboring capillaries. Whenever a vessel of an organ becomes occluded, the parts nourished by the occluded vessel are deprived of blood until a collateral circulation is established. Now, it is found that, after such occlusion, those parts which are at first deprived of blood become subsequently the seat of a rupture of the *capillary blood-vessels* (as the result of an excessive pressure produced by

the collateral fluxion). Hence the infarction is wedge-shaped, as a rule, owing to the distribution of the blood-vessels. Its apex (corresponding to the seat of the plug) usually points toward the centre of the organ in which it is detected. If the circulation is not speedily restored, the result of defective nutrition caused by the embolus is evidenced in one of three ways: either in *gangrene*—if the part be totally cut off from its blood-supply; *fatty degeneration* and *absorption of the embolus* and blood-coagula—if the nutrition be only partially cut off; or more or less extensive *suppuration*—if the plug in the vessel be derived from a suppurative focus or be septic in its origin. We usually find, therefore, that old infarctions are liable to appear pale, and to be firm and incompletely organized, provided that the character of the plug (an embolus or thrombus) does not create suppuration; in which case disintegration

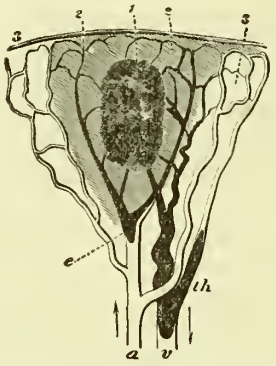


FIG. 76.—A DIAGRAM OF AN EMBOLIC INFARCTION. (After Weber.) *a*, artery obliterated by an embolus (*e*); *v*, vein filled with a secondary thrombus (*th*); 1, centre of the infarction, which is becoming disintegrated; 2, area of extravasation of blood into the tissues; 3, area of collateral hyperæmia.

takes place rapidly in the centre of the infarction, and an abscess results,—"embolic abscess."

The more complete the obstruction, the more vascular the tissue, and the less the vessels are supported, the greater is the amount of infarction and the more rapid the softening and disintegration that ensues. The development of "*metastatic*" or "*embolic abscesses*" is one of the distinctive pathological features of pyæmia, and no case can be properly so called when these abscesses are not found after death.

Etiology.—For some unexplained reason the female sex is more frequently affected with cerebral embolism than the male sex. It is also more common in youth and adult life than in old age.

Floating particles in the circulation (which become emboli within the vessels of the brain) may spring (1) from the *heart-cavities* or from the *mitral* and *aortic* valves; (2) from the *walls of the aorta;* (3) from

the *cavity of some aneurism;* (4) from the *disintegration of some thrombus* in other parts of the body (the loosened particles being swept into the circulation); (5) from particles of connective-tissue growths which affect the vascular system; (6) from chalky concretions; and (7) from *foreign bodies* introduced into the circulation from without.

Affections of the heart, especially endocarditis, are liable to be followed by attacks of cerebral embolism. Aneurisms of the aorta or carotids are the next most common source of emboli. Pyæmia or suppurative phlebitis may induce emboli by disintegration of blood-clots. Oppolzer records a case where a syphilitic gumma of the cardiac wall broke through the sinus of Valsalva and caused embolism. A rheumatic history should point you to an examination of the heart for valvular defect. Cardiac thrombosis has been known to excite embolism. Carcinoma, tuberculosis, and empyema may also act as etiological factors of this condition.

The *size* and *number* of the floating particles modify the seat and number of the emboli. If small, the capillaries of the brain alone may be occluded; if large, one or more of the main trunks are liable to be plugged, and a much larger area of brain-substance is thus deprived of blood. Not infrequently many vessels are simultaneously obstructed at the same time. Sometimes all the main vessels on one side, and at other times vessels of both sides, are more or less occluded.

Symptoms.—The common effects of occlusion of a large trunk are *sudden aphasia* and *hemiplegia* (usually of the right side of the body). When the capillaries alone are involved and the main trunks escape, the effects and symptoms vary with the seat of the embolus, because only certain limited portions of the brain are then deprived of their nutrition. The collateral circulation of the brain takes place almost entirely through the larger arteries. It forms, therefore, an exception to the other tissues.

There are usually no premonitory symptoms that indicate the approach of the attack. The patient does not (as a rule, at least) lose consciousness; although there may be a slight confusion of the mental faculties for a time. In some instances, however, coma accompanies the attack of paralysis. Although so eminent an authority as Nothnagel states that embolic attacks are, as a rule, accompanied by profound coma, my experience does not confirm that view. I regard such cases as exceptions to the general history of embolism; although it cannot be denied that they are frequent exceptions. Even then the patient recovers consciousness gradually within a few hours. Ptosis, a squint of one eye, and even blindness have been known to accompany the attack; but these complications are rather infrequent. In still rarer cases, paralysis may not be developed in any part of the body. The paralysis tends to exhibit

a marked improvement within forty-eight hours, if collateral circulation is established.

The difficulty in speech which commonly results from cerebral embolism is not, as a rule, due to paralysis of the hypoglossal nerve, as it is present in cases where the movements of the tongue are normal. It is to be attributed to sudden anæmia of the "speech centre" of Broca. This is situated chiefly in the base of the third frontal convolution and the adjacent "island of Reil." There are cases reported where both the hypoglossal and facial nerves have been simultaneously paralyzed as a result of embolism, but they are exceptional.

The pupils are not often affected in embolism. The pulse is commonly small in volume and somewhat weak. The temperature may fall slightly below the normal point. A valvular heart-lesion will frequently be discovered. Vegetations upon the aortic and mitral valves are a prolific source of emboli. The arteries of the retina are frequently enlarged at the onset of the attack, because the ophthalmic arteries arise below the middle cerebral.

Emboli are so rarely conveyed to the brain by means of the vertebral arteries that the symptoms produced by occlusion of the branches supplied by that vessel may be clinically disregarded.

Aneurism of the arch of the aorta, or of the innominate and carotid arteries may prove an exciting cause of cerebral embolism. In these cases, portions of the laminated fibrin or blood-coagulum which line the interior of these sacs become detached and are swept into the circulation. It is well to remember this fact, as symptoms referable to aneurismal tumor may occasionally coexist with those produced by the cerebral embolus.

Convulsions may accompany the development of cerebral embolism. The form of convulsion may be of the typical epileptic attack, or it may be only a slight twitching of the muscles, one of the limbs, or the face. The convulsive attack may precede the paralysis, if of the mild type. With bilateral embolism, convulsions are rarely absent. They are due to the extensive anæmia that is suddenly induced. *Transient delirium* may accompany an attack of embolism.

Differential Diagnosis.—From *cerebral apoplexy* the diagnosis is often difficult. Embolism is to be diagnosed chiefly by the absence of profound coma (although there are frequent exceptions to this rule), the absence of premonitory symptoms, the fact that any age may be attacked, the frequent coexistence of a valvular lesion of the heart, or possibly of an aneurism of the aorta or the carotid artery, the predisposition of the right side of the body to paralysis, the simultaneous development of aphasia, and the slow improvement in the paralysis, provided it remains well established on the fourth day after the attack.

From *cerebral thrombosis* the diagnosis is less difficult. The suddenness of the onset, the rapid development of aphasia and right hemiplegia, the absence of prodromal symptoms, the presence of valvular defect of the heart, the absence of atheromatous changes in the vessels, or of any of the well-known exciting causes of thrombosis, the retention of consciousness (more or less complete) during the attack of paralysis, and the absence of suppuration of the ear, epistaxis, circumscribed œdema of the frontal or mastoid regions, are the chief diagnostic points of embolism These points are contrasted on page 227.

Prognosis.—The danger of rapid cerebral softening and possibly of abscess renders the prognosis somewhat grave as regards complete recovery, provided the paralysis does not disappear to a great extent within forty-eight hours after the attack. If a large vessel be occluded, collateral circulation may be established rapidly. The persistence of head symptoms is another omen of evil import. It must not be forgotten that the exciting cause remains and subsequent attacks may be expected.

Treatment.—The symptoms must be treated as they arise. The patient must be kept quiet, and all mental activity avoided for several weeks. Stimulants and digitalis are only indicated when collapse threatens. The faradic current will usually tend to partially relieve the paralysis, in case it persists after the third week.

APHASIA.

This subject has been discussed at some length in a previous chapter (p. 66). The reader is referred to the remarks already made relative to the significance of this symptom, and its bearing upon cerebral localization.

Motor Aphasia is one of the most common symptoms of cerebral embolism, although it occurs often in connection with anæmia from other causes, and also from lesions of a destructive character. It seems proper therefore to consider its clinical significance again in this connection. Too great stress cannot be laid upon the fact that motor aphasia is not pathognomonic of embolism. It may occur in connection with cerebral hemorrhage and many other diseases of the brain.

It should be remembered also that aphasia may be due either to an inability to properly coördinate the muscles of articulation (which are governed to a marked degree by the centre of Broca), or to a loss of memory of articulate sounds or their symbols. Should the meaning of *spoken* language be lost, the lesion is probably situated in the first temporal convolution, and not in the centre of Broca; provided the patient can articulate perfectly, as can be determined by having the patient repeat single test-words, and in other ways. (See page 64.)

Among the aphasic-symptom group may be mentioned the conditions known as "aphasia," "alexia," "amimia," "apraxia," "asymbolia," "agraphia," and "paraphasia." Thus, disturbances of speech (*true aphasia*) may be associated with an inability to read (*alexia*), to make appropriate gestures (*amimia*), to recognize objects in common use (*apraxia*), to sign the name (*asymbolia*), to write or copy (*agraphia*), and to properly select words (*paraphasia*).

The term "*apraxia*" has been employed by some authors to cover a class of cases where by disease of the cortical centres of sight the patient has been rendered psychically blind, not to the meaning of words, but to the most familiar objects and their uses. Such patients, after being disrobed and having their clothing returned to them, have been known to require instruction as to what uses to put each garment to. The most common objects about them are apt to be regarded as things unknown by such subjects.

The lack of power to express the psychical states by means of gestures has been designated "*amimia*." The power of appreciating the meaning of gestures made by others may coexist with the loss of power on the part of the patient to personally execute appropriate mimic interpretations of thought.

Whenever motor aphasia is clearly dependent upon cerebral embolism or general cerebral anæmia, efforts should be made to relieve the vascular disturbance (as far as is possible), and to treat the coexisting symptoms. Such steps will be discussed in connection with cerebral anæmia.

Clinically, however, we often encounter motor or sensory aphasia in connection with circumscribed lesions, such as depressed bone, meningeal or cerebral hemorrhage, suppuration after an injury to the skull, cerebral softening, tumors, etc., as well as a symptom of embolism.

Under such circumstances the question of mechanical relief by the trephine has to be considered.

There is a safe rule to follow whenever the use of the trephine is suggested, viz., *never to employ it when sensory paralysis coexists with motor paralysis, or when the motor paralysis exists on the side of the body corresponding to the probable seat of the cerebral lesion.*

The reasons for this rule are self-evident. The coexistence of motor and sensory paralysis indicates a lesion that is either too extensive or deeply seated (although it may possibly be cortical in character) to be benefited by the removal of a small button of bone. Again, the development of paralysis upon the same side as a cerebral lesion justifies a doubt, to say the least, regarding the hemisphere of the brain which has been injured.

Those cases known as *word-deafness* and *word-blindness*, where the

memory of spoken words or of written characters has been effaced by a cerebral lesion, are now better understood than in the past. We have reason to believe that the memories of sounds are stored in the cells of the first temporal convolutions, and those of sight in those of the occipital lobes. We also know that after a lesion of these gyri has effaced past memories new ones may be often acquired and retained, provided that all of the cells of the affected gyrus are not actually destroyed. Hence we have come to learn that patients afflicted with aphasia of the amnesic type (p. 63) can be slowly taught in some cases the meaning of sounds, and cases of word-blindness those of symbols, in spite of the fact that

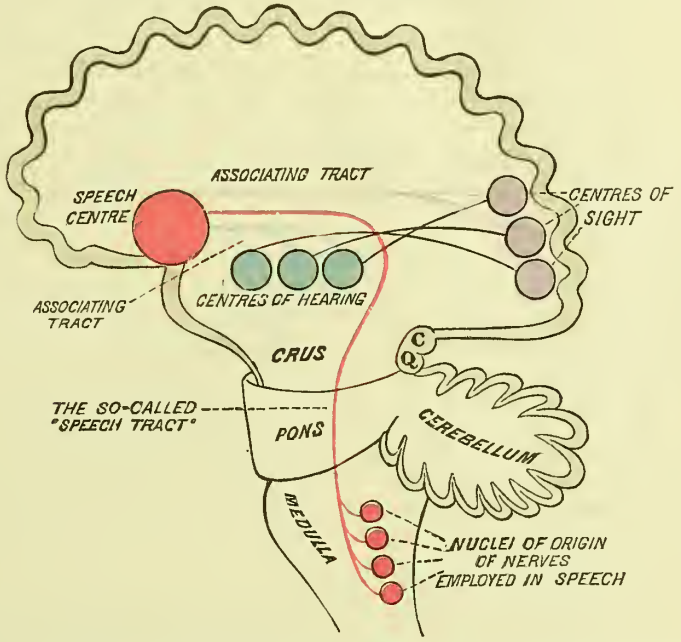


FIG. 77.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE MECHANISM OF THE APPARATUS REQUIRED IN SPEECH.—The reader must not regard this diagram as intended to accurately portray the anatomical relations of the various centres and tracts to each other. The physiological interpretation of this diagram has been given on page 67.

the memories of such have been mechanically effaced. The cortical cells associated with the auditory nerves or with the eyes must be developed anew in respect to their functional attainments, as if the patient were an infant. Words must be repeated again and again, until the patient can retain them in memory and pronounce them properly, in case of "word-deafness," and after the development of "word-blindness" the patient must sometimes be taught the letters and numerals in the same way as a child is instructed. The tests for these conditions have been given in the preceding section (p. 183).

In cases where *word-deafness* is suspected to exist it is well to test the patient by instructing him to do certain simple things, such, for example, as putting out the tongue, pointing to selected objects in the room, shaking hands, passing common objects to some third party, etc. It is particularly important that no sign or gesture should be employed while making such tests as will serve to aid the patient in discerning the meaning of any of the requests made as a means of determining the integrity of the cortical centres of hearing.

Embolism of the *left middle cerebral artery* is perhaps the most frequent cause of true motor aphasia, because the same vessels which nourish the motor speech-centre supply also most of the motor centres in the two central convolutions. A *right hemiplegia* is commonly observed in connection with aphasia (when due to embolism).

Agraphia, like aphasia, may be of the ataxic or amnesic types. Whenever the sight and hearing centres are impaired, copying from sight or dictation will be rendered impossible, because centres which control the muscles employed in writing lack the necessary stimulus to perform such coördinated movements.

When the coördinating centre of the muscles of speech is permanently impaired by a lesion of the vessels or brain-tissue, the prospect of a return of the normal power of articulation is not, in my experience, encouraging, although great improvement may sometimes occur (possibly by the aid of the homologous centre in the uninjured hemisphere).

The guides that are employed to-day in trephining for the special centres of the cerebral cortex have been given elsewhere (p. 49).

My friend and late colleague Prof. Wm. H. Thompson, of New York, has lately published a very interesting case of word-blindness which came under his personal observation. I take the liberty of quoting his record of this case in full:—

"On May 1, 1884, I was called to one of my stated patients, a lady of about sixty years of age, whom I found naturally anxious about a peculiar experience which had befallen her. The previous afternoon she had taken a long ride in her carriage to Greenwood Cemetery, to visit the grave of her only son, who died three years before, of phthisis. She said that she had enjoyed the ride, and did not feel particularly fatigued by it, but on returning home began to experience a sensation of unusual weariness. She exerted herself, however, then, before going down to dinner, to write an advertisement to come out in the morning paper for a servant-girl. She was surprised, however, to find that for some unaccountable reason she could not word the advertisement to suit her, and after tearing up some five or six such written attempts she was obliged to ask her sister to write the notice for her. Soon afterward, while at the dinner-table, a severe pain set in at the upper portion of the left temple, which continued to increase until it obliged her to retire to her room, and not long afterward, to bed. This pain persisted through the night, but did not prevent her from having a fair amount of sleep. She rose at her customary hour in the morning, and but for the persistence of the same pain, though in less degree than on the evening before, she would not have noticed anything unusual about herself, had it not

been for the arrival, soon after breakfast, of an applicant in answer to her advertisement. Upon the girl handing her some written recommendations the lady found herself unable to make anything out of either one of them, and had to call her sister in, who then read them without difficulty. Soon another girl came in, and my patient experienced just the same difficulty in attempting to read her references. She said that her first thought was that something was wrong with her eyes, but on looking around the room and inspecting a number of small articles minutely she was satisfied that she could see and distinguish objects as well as ever. The moment, however, that she turned to the writing, while she knew that she could see the written characters as well as she could see worsted work, yet not a single letter conveyed any idea to her mind of its character or meaning. She thereupon took up a newspaper, and at once recognized that something peculiar had happened to her, for she was totally unable to read a word in it. The separate letters could be seen, but an indescribable blur, as she thought, rendered it all indistinguishable; whereupon I was sent for to explain the difficulty.

"I was much interested, of course, in the patient's story, for nothing could have been better described or expressed in words. There was neither hesitancy nor thickness in her articulation, nor confusion in diction or thought, but, on the contrary, she detailed her case with a peculiarly good choice of terms. 'What is it, doctor, that makes that newspaper so illegible to me? I see that there are words there, but I am wholly unable to tell what they are,' were some of her remarks. At first I directed my questions so as to avoid increasing her alarm and excitement, and in time found that she had not experienced another symptom except the above-mentioned pain and her inability to read or write. She felt no numbness or tingling, either in the face or extremities, nor any loss of power, her grasp being the same as usual in each hand, while no difference was perceptible to her between either of the lower extremities in walking. The use of the hands for sewing, buttoning, or tying, and for holding a pencil for writing seemed as good as ever. There was no difference observable in the vision of the two eyes,—no specks, nor mists, nor colored images; no marked difference in hearing on either side, nor any other symptoms referable to the ears; and there was no dizziness whatever. The face showed no distortion, either when the patient was speaking or laughing. Examination of the radials showed them to be hard and tortuous, and the pulse was of high tension and slightly quickened. I may remark here that a brother of the patient, a few years her senior, had a slight hemiplegic attack, with aphasia, some seven years ago, from which, however, he has quite recovered.

"At my first visit I was soon obliged to desist from experimenting with my patient's inability to recognize written or printed words or figures; for the plainer this strange disabilty became to her by my tests the more she was inclined to become distressed by it and to press for an explanation, so that I feared the effects of excitement upon her cerebral circulation. At my visit the next day the pain in the temple still persisted, and was uniformly described as running along a line which corresponded to the temporo-parietal suture. Some days afterward it was noticeable that she occasionally miscalled words, of which, however, she immediately corrected herself. On cautiously testing her again, I found that her word-blindness at the end of the week was complete. The largest letters, like the heading of the New York *Herald*, and figures were as unrecognized by her, when separately pointed out, as the smallest. With the exception, however, just mentioned, her spoken language was that of a well-educated woman who had learned to express herself fluently and well.

"Her recovery from this condition began in about two weeks and progressed gradually until in three months she could both read and write, especially the latter, with tolerable facility. When she began to write again, however, it was in a very small hand;

but in time she quite recovered her ordinary handwriting. Since then she has shown little or no change, except a marked increase of restlessness and impatience. She now writes all her own letters, but says that, whereas she used to be a good correspondent, the task of answering letters has become very irksome. Reading, however, she finds more difficult than writing, for she can read aloud only slowly; while reading to herself, she says, soon fatigues her."

Differential Diagnosis.—The following table is quoted with some modifications in this connection from the third edition of the author's work upon "Surgical Diagnosis":—

APHASIA.	GLOSSO-LABIO-LARYNGEAL PARALYSIS. (Duchenne's Disease.)
Early Symptoms.	
The loss of speech is usually sudden and only partially complete. The lips are under perfect control and do not tend to separate.	Patient notices a slight impediment in speech early in the disease, or a tendency in the lips to separate and remain apart.
Deglutition.	
Swallowing is not interfered with.	Swallowing is imperfectly performed later on in the disease. The palate becomes affected, and attempts at swallowing induce symptoms of strangulation.
Face.	
The face is normal in expression.	Saliva dribbles constantly from the mouth, in the advanced stages of the disease; and the face is altered by the attitude of the jaw and the separation of the lips.
Speech.	
In the *ataxic* or "*motor*" *variety*, speech is impaired in a variety of ways (page 64). In the *amnesic* or "*sensory*" *variety*, some form of memory of words or symbols is lost.	The lingual and dental consonants are first pronounced with difficulty, and, later on the labials.
Mastication.	
Mastication is performed as in health.	The food accumulates in the cheek during eating, as the tongue cannot control the bolus properly.
Respiration.	
Respiration is not affected.	Respiration becomes impaired, from paralysis of the muscles necessary to that act. The patient often cannot cough or breathe deeply.
Voice.	
The voice is normal.	Phonation becomes impossible when the larynx is paralyzed.
Atrophic Changes.	
No atrophic changes in muscles occur throughout the disease.	As the disease tends to extend into the spinal cord and involve the cells of its gray matter, symptoms of muscular atrophy develop.

Motor Paralysis.

The *right side* of the body is usually rendered hemiplegic, when the aphasia is of the motor variety. This is especially true if an embolus exists as its cause.

Motor paralysis is not developed as a result of the spinal changes. The patient becomes unable to walk, however, as a result of general debility and the atrophy of the muscles.

Memory.

The memory may be impaired, in the amnesic variety, in respect to words, figures, foreign tongues, familiar objects and their uses, gestures, etc.

The memory of words is intact, but the ability to use the tongue and lips interferes with articulate speech.

Dementia.

Dementia seldom, if ever, occurs.

In exceptional cases, dementia develops late in the disease.

Symptoms in Common.

Both may be associated with impairment of the normal use of the tongue.
Both may be associated with defects of speech.

Prognosis and Treatment.—The duration, course, and severity of the abnormal conditions described under the general head of aphasia depend to a great extent upon the exciting cause and its exact seat. In the first section of this work, the localization of the lesion and the complications most frequently encountered in connection with aphasia have been quite fully discussed. To these pages the reader is referred.

The treatment of the various lesions which may induce aphasia will be discussed later, under the heads of cerebral hemorrhage, tumors, softening, abscess, etc. Some points relative to the treatment of cerebral embolism and thrombosis have already been given.

HYPERÆMIA OF THE BRAIN AND ITS COVERINGS.

Contrary to opinions of the past,* it is now quite well determined that the quantity of blood within the cavity of the skull may admit of variation and be increased under certain circumstances to an abnormal extent, constituting the condition known as cerebral hyperæmia.†

Two forms are commonly recognized,—the active and passive. Both may be general or localized.

* The experiments of Kellie led him to deny that the cerebral circulation could be affected by bleeding, the ligation of veins, etc.

† Burrows, Donders, Kussmaul and Tenner, Jolly, Leyden, Ackermann, and Ermann have demonstrated that the view of Kellie was untenable.

The functions of the *cerebro-spinal fluid* were imperfectly understood until Magendie, Longet, and Ecker brought them to light. The *perivascular lymph spaces* unquestionably assist also in the imperfectly understood mechanism by which the cerebral circulation is regulated. Maingien believes that the *thyroid gland* plays an important part in preventing over-distention of the cerebral vessels,—a conclusion that is sustained by as high an authority as Guyon. The *thyroid lobes* become turgid and swollen during excessive muscular exercise, and overcomes (by compressing the carotid arteries) the danger of *venous congestion* of the brain.

In the *active* variety, the blood-vessels are dilated and (although the amount of blood is proportionately increased) the current is very rapid. In the *passive* form, the blood-vessels are dilated and the amount of blood is also proportionately increased, but the current is slower than normal.

The term "cerebral congestion" is properly applied to the latter variety only, in spite of the fact that authorities of note sometimes employ it when speaking of both forms.

Some neurologists have attempted to classify cerebral hyperæmia on a basis of its symptomatology. Andral speaks of eight varieties, and Hammond of six. These have been designated by the terms "apoplectic," "paralytic," "convulsive," "maniacal," etc., in accord with the most prominent of the symptoms exhibited.

Morbid Anatomy.—Great variations in the intensity of cerebral and meningeal hyperæmia are observed.

If the condition be of the *general variety*, in contradistinction to local hyperæmia, the vessels will be found to be engorged and the membranes altered in their color. Some of the cerebral convolutions may be slightly compressed and flattened. Finally, hemorrhagic points may be detected both in the gray and white substance of the brain on section. In some cases, coagula of blood may be detected in both the arteries and sinuses.

Chronic hyperæmia is recognized chiefly by a thickening and opacity of the membranes and marked dilatation of the vessels. The cerebellar meninges are commonly more vascular than those of the anterior portions of the brain. In some insane patients, the cortex may assume a brownish and pigmented condition.

Localized areas of hyperæmia are occasionally observed. These sometimes coexist with embolism or thrombosis. The basal ganglia (corpus striatum and optic thalamus) occasionally exhibit isolated congestions.

It is not uncommon to meet an increase in the subarachnoidean fluid and distension of the choroid plexuses, in connection with cerebral congestion. Moreover, particles of hæmatin may often be found in contact with the blood-vessels, and miliary aneurisms are liable to be found.

Etiology.—In a general way it may be stated that the so-called "active" form of cerebral hyperæmia is dependent upon any cause that tends to increase the arterial supply of the brain or its coverings without interfering with the venous return from the vessels within the skull, and that the "passive" variety results from defective venous return, irrespective of the arterial supply. This axiom is not strictly true in all cases, because the passive form may develop as a secondary result of the active; but it will hold good in the majority of cases and prove of assistance often in diagnosis.

THE ACTIVE VARIETY (*cerebral fluxion*).—A *heated atmosphere* is often a factor in the development of the active variety. The passive form, on the other hand, is more frequent in extremely cold weather, as sustained by the observations of Andral and Hammond; but cold may induce either variety.

An *increased heart's action* (as in the case of fevers, mental excitement, severe physical exertion, and cardiac hypertrophy) may be an important factor in producing the active or "fluxionary" type.

Again, the *cerebral arteries* may *possess weak walls;* so that they yield to an increased pressure of blood sooner than the rest of the arterial system, thus causing what is termed a "rush of blood to the head."

As a fourth factor, *increased lateral pressure within the carotids* may be mentioned. This occurs in connection with constriction of the thoracic or abdominal aorta, or compression of the abdominal aorta by distended intestines or abdominal tumors, and of the thoracic portion of that vessel by mediastinal growths, emphysema, etc. The effect of cold upon the surface of the body, which checks the determination of blood to the skin and drives an excess of blood to the viscera, may also be evidenced in the cerebral vessels.

Again, *vaso-motor paralysis* may induce active cerebral hyperæmia. An excess of alcohol, indulgence in some drugs, malarial poisoning, prolonged mental labor, and emotional excitement may also lead to this condition. Opium-eaters and drunkards have chronic cerebral hyperæmia, as a rule.

Finally, *cerebral atrophy* may possibly be associated with that form of cerebral hyperæmia which is occasionally encountered during convalescence from severe attacks of illness.

THE PASSIVE VARIETY (*cerebral congestion*) must of necessity depend to a great extent upon causes that interfere with the return of blood from the cerebral sinuses. Among these conditions the following may be prominently mentioned:—

1. *Compression of the Jugulars.*—This may be induced by wearing of tight clothing around the neck, enlargement of the thyroid or the lymphatic glands, new growths in the neck, or by strangulation, etc.

2. *A Dependent Position of the Head.*—Acrobats are not infrequently attacked with cerebral congestion. Any labor performed with the head down, or with the body in a stooping posture, may induce it. Attacks have been brought about by straining at stool or the buttoning of the shoes.

3. *Violent Expiratory Efforts.*—Playing upon wind instruments, severe paroxysms of coughing, etc., tend to prevent the entrance of blood into the chest, and thus to force too much blood into the general circulation. Such acts may prove an exciting cause of this condition.

4. *Valvular Disease of the Heart.*—When valvular defect exists at the mitral or aortic orifices, sufficient hypertrophy of the auricle or ventricle may be developed to compensate for the deficiency. In this case the organ exhibits no marked impairment of its function; but, when the compensation is disproportionate to the valvular lesion, the return of blood from the head is seriously interfered with. Under such circumstances anything that tends to over-excite or weaken the heart's action produces marked cerebral disturbances.

5. Symptoms of *cerebral congestion* are occasionally developed as

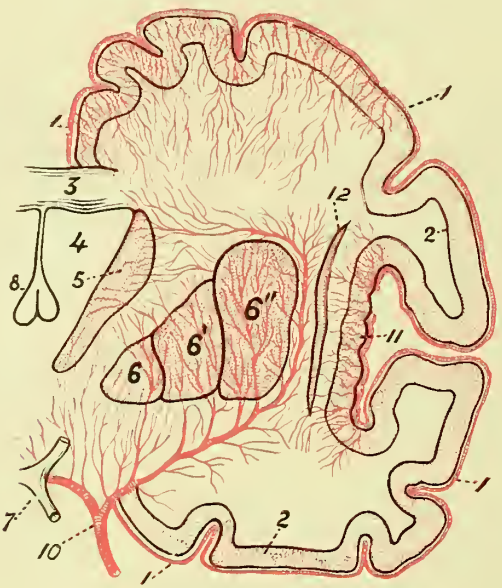


FIG. 78.—DIAGRAM OF A TRANSVERSE VERTICAL SECTION OF THE LEFT CEREBRAL HEMISPHERE, SHOWING THE ARTERIAL DISTRIBUTION. (Modified slightly from Westbrook.) 1, arteries ramifying in the pia, and sending off cortical and medullary branches; 2, gray matter of cortex; 3, corpus callosum; 4, cavity of the ventricle; 5, caudate nucleus; 6, 6′, 6″, members of lenticular nucleus (Glieder); 7, internal capsule; 8, septum; 9, optic chiasm; 10, arteries from circle of Willis, sending branches to basal ganglia; 11, convolutions of Island of Reil; 12, claustrum.

the result of *plethora*, caused by excess in eating or drinking. It is questionable to my mind if this form is not always associated with some organic changes in the arteries,—probably of the atheromatous type.

6. *Persistent Anxiety or Emotional Excitement.*—This is perhaps the most common and potent of all the etiological factors of cerebral congestion. The prolonged activity of the brain protracts the determination of blood to the head (which is requisite to maintain that activity) beyond its proper limits. The continued over-distension of the cerebral vessels causes the coats to lose their contractility, and thus active hyperæmia (which at first existed) becomes passive. Examples of the marked

effect of emotions upon the circulation of the head are well exhibited in the turgid face of anger, the blush of shame, and the pallor of fear.

7. *Eye-strain* (from an uncorrected refractive error or imperfect adjustment of the ocular muscles) often tends to excite and to maintain passive cerebral hyperæmia. This, in my experience, is a very common cause.

Symptoms.—The manifestations of the active and passive varieties differ markedly. Some authors describe the symptomatology of cerebral congestion as capable of being divided into two stages,—those of active hyperæmia constituting the first, and those of passive hyperæmia the second. It seems to me illogical, although perhaps it is clinically true in a certain proportion of cases.

The active form is an independent condition. It may be transient; and is not invariably followed by the passive variety. It is, moreover, chiefly if not exclusively encountered in those subjects that have suffered from prolonged anxiety, emotional excitement, or mental strain that we meet the two as consecutive stages.

Galton first called attention to the utility of an examination of the drum of the ear as a means of determining the existence of cerebral congestion, and Hammond and Roosa have used it as a guide to the condition of the intracranial circulation in their experiments with the internal administration of quinine. I have had reason in several instances to acknowledge the utility of this step. It is necessary in many cases to clear the ear of wax before the drum can be utilized for this purpose. The tympanum will be congested over the handle of the malleus and be pink in color when cerebral hyperæmia exists.

Symptoms of the Active Variety.—The cause of the hyperæmia will modify the symptoms in each individual case. There is a widespread error (often injurious to the patient) that all cerebral disturbances which cannot be traced to some definite cause must be dependent upon hyperæmia or anæmia of the brain or its coverings. That this is an error is proven conclusively by the fact that the quality of the blood as well as its quantity may exert an influence upon the cerebral functions (as clinically observed in fevers, poisoning by alcohol, drugs, etc.), and that a general overheating of the body may produce the symptoms of sunstroke without actual exposure to the sun's rays. The cerebral hyperæmia which accompanies alcoholism, for example, plays probably but an insignificant part in the development of the symptoms of that condition, provided the use of alcohol has been long continued.

The symptoms of cerebral hyperæmia may be classified under two heads, viz., those dependent upon *cerebral irritation* and those indicative of *cerebral depression*.

Under the former, the following may be prominently mentioned:

1, headache, which may be more or less severe and accompanied by throbbing; 2, an increased sensitiveness to light or sound; 3, abnormal phenomena connected with the special senses, such as sparks before the eyes, buzzing in the ears, formication in the limbs, and ill-defined pains; 4, motory symptoms, such as restlessness, vertigo, muscular startings, gnashing of the teeth, vomiting, convulsions, etc.; 5, disturbance of the psychical functions, such as hallucinations, illusions, delusions, melancholia, etc.; 6, persistent insomnia, which is perhaps more marked than any symptom in most cases; and 7, a loss of control over the emotions. These patients are apt to talk a great deal, and to exhibit other manifestations of mental exaltation.

The effects of *cerebral depression* may be indicated by any of the following symptoms: 1, insensitiveness to external irritation, such as a bright light, loud noises, etc.; 2, an altered condition of the pupils; 3, sluggishness of the intellectual faculties; 4, slowness of muscular movements, which may go on to paresis or paralysis; 5, marked somnolence, possibly deepening into coma; and 6, slowing and deepening of the breathing, which may become stertorous.

In all cases, the symptoms are increased by the recumbent posture, by deep inspirations, and by stimulants.

All possible combinations of the symptoms of either of the two varieties described may be encountered in any individual case. Some of the more important deserve special mention.

Insomnia is a very common symptom of cerebral irritation. If sleep is not actually prevented, it is liable to be markedly disturbed by bad dreams and fails to refresh the patient.

Headache and throbbing in the head is a very constant symptom. The pain may be either of a dull, aching character, or extremely severe. It is greatly aggravated by stooping, deep inspirations, or muscular and mental exertion. Stimulants usually increase it. In many cases compression of the carotids relieves it.

Prolonged mental efforts bring about a *confusion of intellect* early, in many cases. This is particularly noticeable when mental exercises requiring concentration, such as adding up columns of figures, solving of mathematical problems, reading of philosophical works, etc., are attempted. To this symptom may sometimes be superadded delusions, illusions, hallucinations, melancholia, morbid fears, and a loss of emotional control.

Vertigo is a very constant and important symptom. It may be so severe as to prevent the patient from attending to his business. It may, furthermore, be associated with unnatural sensations in the head, such as a sense of constriction, a snapping noise, an encircling band, a burning sensation, etc.

The *motory symptoms* are sometimes confined to one side, but this is not always the case. Paresis or actual paralysis sometimes develops suddenly in an arm or leg, or both. All of the limbs are simultaneously affected in rare cases. Convulsive twitchings may develop in the muscles of the face and limbs. The heart is liable to exhibit a marked acceleration in the frequency of its beats after slight exertion, and to develop paroxysms of palpitation. This is independent of any organic lesion, and is to be attributed to cerebral disturbance. Respiration may become similarly disturbed when the heart becomes unduly excited. Aphasia is sometimes developed, either independently of or in conjunction with paralysis of the limbs. It is usually transient. Convulsions may occur in the advanced stages of cerebral hyperæmia. They differ from those of epilepsy in the absence of an aura and the epileptic cry.

The *psychical symptoms* are sometimes prominent. A gradual stupor may develop in some cases, accompanied with pain in the head and dilated pupils. Again, mania may occur, with active delirium and a tendency to acts of violence. These states, however, are preceded in every case by some of the premonitory symptoms that have been previously enumerated.

Symptoms of the Passive Variety.—The symptoms of irritation are less marked in this form than those of cerebral depression. Although all of those enumerated in previous pages may occur in the passive variety as well as in the active, it is more common to meet with somnolence early instead of insomnia, and to find all the irritative symptoms subordinate.

Whenever the congestion is attended with an *exudation of serum* we are liable to encounter paralysis, convulsions, deep coma, maniacal attacks, or aphasia. This form of hyperæmia is more liable to serous effusion than the active, and is therefore a more serious disease.

Passive cerebral hyperæmia is particularly prone to pass into that stage where alarming symptoms appear. It is not uncommonly fatal. In some cases it induces a condition of body that is diagnosed with difficulty from apoplexy.

If *convulsions* develop, they are generally of longer duration than if due to active hyperæmia, and are followed by a more profound stupor. The tongue may be severely bitten. The fit may be followed by paralysis. The mind appears to suffer rapid deterioration after the convulsive attacks develop.

Whenever *mania* ensues the patient seldom exhibits as active delirium as in the previous form of cerebral hyperæmia, nor are acts of violence as common.

The ophthalmoscope will usually show a marked turgescence of the retinal veins in this form of cerebral hyperæmia.

Differential Diagnosis.—The history and examination of the patient is an important factor, because it aids in deciding as to the existence of hysteria, chlorosis, alcoholism, venereal excesses, seminal weakness, diseases of the heart, lungs, or abdominal viscera, and many other conditions that often tend toward the development of cerebral hyperæmia.

From *cerebral hemorrhage* this condition differs in that consciousness is seldom completely abolished; that the paralysis is not unilateral as a rule; that sensation and motion are seldom simultaneously impaired or lost; and that the symptoms are of shorter duration.

From *embolism*, it differs in that premonitory symptoms have existed; that the paralysis is of shorter duration; that the pulse is slowed rather than accelerated at the time of the attack; that the temperature of the head is elevated; and that cardiac disease is usually present in embolism.

From *epilepsy* it is to be told by the absence of an aura and of the epileptic cry; the existence of premonitory symptoms; the staggering before an attack; the absence of facial pallor before the fit; and the infrequent biting of the tongue.

From *uræmia* the diagnosis is made by the waxy pallor of the face in renal disease; also the absence of albumen and casts in the urine; the infrequency of nausea or vomiting; the absence of œdema of the eyelids and possibly of the extremities, and the infrequent convulsions.

From *cerebral softening* it is distinguished by the aggravated character of mental impairment of that malady, together with the progressive interference with articulate speech. The gradual onset of paralysis preceded by feebleness of gait in some cases, and the development of a persistent hemiplegia with a sudden loss of consciousness would point to the more grievous malady.

From *stomachic vertigo*, the presence of marked gastric derangement in connection with such attacks of dizziness (which is often wanting in congestive vertigo) aids in the diagnosis.

Treatment.—The multiplicity of causes of this affection would naturally suggest that the treatment must be modified by the history of the patient. It is important, furthermore, that the diagnosis be carefully made before any line of treatment is commenced. This is not always an easy matter. It is especially difficult in some cases to decide between cerebral hyperæmia and anæmia.

The ACTIVE VARIETY requires measures that will tend to lessen the action of the heart and diminish the quantity of blood in the cerebral vessels. Leeches to the temporal region or within the nostril, or at the neck close to the skull, will often prove of immediate benefit. The actual cautery applied to the neck near to the base of the brain, and the galvanic current so used as to stimulate the main sympathetic nerves in the neck, both tend to cause a diminution in the calibre of the cerebral

vessels, and are therefore useful. My experience with the cautery has led me to believe that it is more prompt in its effects than any other single remedial measure. It is comparatively painless when properly used. Heavy static sparks from Leyden-jars applied to the neck are sometimes of great benefit.

Sleeping with the head raised (especially with the arms placed above the head) will help to decrease the amount of blood in the brain. Stooping should be avoided. Severe muscular exercise increases the heart's action; hence it should be prohibited in this class of cases. The clothes should not press tightly upon the neck, as it tends to impede the venous return from the head.

Warm baths to the feet, mustard plasters over the stomach, and cold effusions to the head and neck are valuable adjuncts to treatment in some instances, since they all tend to decrease the amount of blood in the cranial vessels.

Among the internal remedies employed by me in this affection (after all recognizable causes have been removed without a cessation of the symptoms) are the bromides of sodium and potassium, ergot, oxide of zinc, phosphorus, strychnia, and arsenic. The internal administration of hot water, according to the rules given in my paper upon this agent,* is often attended with great benefit.

The bromides and the ergot exert an immediate effect upon the amount of blood within the cerebral vessels, causing a very marked decrease, as was first proven experimentally by Hammond. I have found ergot a very valuable adjunct to the bromides. They are best given by dissolving the bromide in a fluid extract of ergot. The following formula is an excellent one:—

℞. Potasii bromidi . . . . . ʒ j.
Ergotæ ext. fluidi . . . . . ʒ iv.
M. Ft. sol.
Sig.—Dose, a teaspoonful after each meal.

In place of ergot, ergotin may be substituted (in doses of five grains) in pill three times a day, where the taste of the fluid extract is disagreeable to the patient.

I am inclined to lay stress upon the beneficial effect of the oxide of zinc, when given in connection with ergot, the bromides, and the hot-water treatment, which will be described later. It should be given three times a day (in doses of two grains) in pill after eating, as it is less liable then to cause nausea. By the use of these three drugs and hot water as a beverage the symptoms will usually disappear inside of two weeks. It is then advisable to begin a course of tonic treatment.

The tonics that are commonly employed are quinine, strychnia, arsenious acid and other arsenic preparations, hydrobromic acid, and

* *N. Y. Med. Jour.*, October 17, 1884.

phosphorus. I have used Warburg's tincture with excellent effects in doses of from one to two drachms after each meal.

In regard to quinine, I have tried the hydrobromic acid with good results in combination. It undoubtedly relieves the unpleasant head-symptoms of the quinine.

Phosphorus should be given, when indicated, in doses varying from one-fiftieth to one-hundredth of a grain. It may be administered either in an emulsion, a capsule, a pill (by the aid of resin), or in the form of the phosphide of zinc.

Arsenious acid is highly recommended, in doses of one-fiftieth of a grain, as a substitute for other arsenical preparations. My experience with it is too limited as yet to enable me to form any positive conclusion regarding it.

Dyspeptic symptoms should be treated by keeping the bowels open and the *continued use of hot water as a beverage*, a gobletful being drank one hour and a-half before each meal, with the temperature as high as it can be borne (110°–150°). Twenty minutes may be consumed in sipping a goblet-full when necessary.

Irrespective of dyspeptic symptoms, I have been in the habit, however, of recommending this treatment to nearly all of my nervous patients. Its effects have proven quite remarkable in my hands. It increases downward peristalsis, which warm water does not (as the latter is an emetic); it stimulates the secretion of urine, and alters its character with great rapidity; it produces a gentle perspiration after drinking it, and a sense of warmth in the skin; it relieves dyspeptic symptoms better than charcoal and bismuth, if continued long enough; finally, it seems to act upon the sympathetic system (probably by affecting the solar plexus), as is shown by the relief of most forms of local hyperæmia. It must be taken, however, with absolute regularity, one hour and a-half before every meal, so as to get the effect of the heat and to wash out the stomach before the ingestion of food. A little lemon or flavoring of any kind may be added, if the taste is disagreeable; although patients soon learn to crave it without such additions. Carlsbad salts may be added to the morning dose, if constipation exists. In some cases it becomes necessary to restrict certain articles of food during the hot-water treatment. I have published the methods employed by me in full in a brochure* upon the subject, to which the reader is referred.

In the PASSIVE VARIETY of cerebral hyperæmia the indications for treatment are to increase the heart's power and assist the venous return from the cerebral sinuses.

Stimulants are strongly indicated, therefore, in many cases, in conjunction with the other methods of treatment previously suggested to

* *N. Y. Med. Jour.*, October 16, 1884.

relieve the congestion of the vessels. Alcohol, sulphuric ether (by inhalation or the stomach), carbonate of ammonia, and digitalis may be employed often with marked benefit.

The habits of the patient should be controlled, provided that excesses of any kind exist, and are thought to be injurious. Tobacco, tea, coffee, opium, etc., may be factors both in the causation and persistence of the cerebral congestion.

Success in treatment of passive cerebral hyperæmia will depend chiefly upon the removal of the exciting cause of the condition. It should be coupled with such advice as to exercise, mental work, bathing, eating, drinking, etc., as will tend to prevent its return by promoting a general improvement in health.

Static insulation and a fusillade of sparks to the spine seem to give these patients relief. I often employ the umbrella-head-electrode in these cases with satisfactory results. The static machine used must be sufficiently powerful (when this is employed) to generate large quantities of electricity.

ANÆMIA OF THE BRAIN AND ITS MEMBRANES.

Kussmaul, Donders, and Tenner have observed the phenomena of marked cerebral anæmia through a glass plate inserted in the skull of animals; and Nothnagel, Loven, Mayer, and Pribam, and many others have proven the possibility of artificially producing it. These facts are mentioned because, for a time, the existence of this condition as a distinct disease was denied. Cerebral anæmia may be localized or general. The latter is the most common variety. The symptoms of this condition are modified (1) by the variety and (2) by the method of its development (whether sudden or gradual), irrespective of its intensity.

Morbid Anatomy.—The meningeal vessels are usually nearly empty; although, in some cases, meningeal hyperæmia may coexist with cerebral anæmia. The pia usually contains some serum in its meshes. The medullary substance of the brain is of a dull, white color, and presents on section few, if any, vascular spots. The masses of gray substance are poorly defined in their outline, in sagittal or transverse sections of the brain. They are dryer and firmer than normal, in case the anæmia has persisted for some time, or when it exists in connection with general anæmia or chlorosis.

In connection with partial cerebral anæmia it is common to find the adjacent areas markedly hyperæmic. This hyperæmia may occasionally exist also in parts somewhat removed from the anæmic territory. It is due to excessive arterial tension in the vessels whose supply is not interfered with.

If the condition of cerebral anæmia has been developed as a result

of localized pressure (as in the case of cerebral tumors, large extravasations of blood, encephalitis, etc.) the convolutions of the brain may be flattened or otherwise distorted.

According to the researches of Golgi, in all forms of cerebral anæmia the perivascular spaces are enlarged, even if œdema be present.

Etiology.—The causes of this condition, when widely diffused or partial, can be classified under several heads, as follow:—

1. *Those Causing a Diminution of the Space Within the Skull.*—Under this head may be mentioned all forms of exudation, new growths, and blood extravasations. Tumors and blood extravasations tend to produce definitely localized pressure upon the adjacent areas of the brain. Exudations (when very extensive) may result in a more diffused pressure. The brain may be more or less distorted in its outlines from all of these causes.

2. *Compression or Obstruction of the Arteries that Supply the Brain with Blood.*—Under this head come embolism, thrombosis, ligation, aneurism, pressure of new growths upon the vessels, etc. Fortunately for life, the "circle of Willis" allows of a collateral circulation in case of ligation or other causes of obstruction to the vessels of one side of the neck. Ligation of both carotids or a severe loss of blood from any large artery or vein is followed invariably by the symptoms of general cerebral anæmia.

The circle of Willis unquestionably prevents in the majority of subjects general cortical anæmia of one hemisphere, in case of ligation of the carotid or other obstruction to its calibre. But it is questionable if the basal ganglia are not more liable to ischæmia from such causes than the cortex. The anastomoses are less frequent in these gray nuclei than upon the surface of the cerebrum, and sometimes the branches of the circle of Willis are impervious or imperfectly developed. Ehrmann found about 20 per cent. of cases (selected at random) to exhibit defects in the vessels mentioned.

3. *Overloading of Other Organs with Blood.*—Severe catharsis, extensive dry cupping, an enfeebled heart's action, and simple gravity may induce cerebral anæmia. As an illustration of the last cause, convalescents from protracted fevers or debilitating diseases often faint when they attempt to sit up.

4. *Direct Abstraction of Blood from the Brain.*—In case of a severe hemorrhage from some vessel of the trunk or extremities, the brain is rendered anæmic early.

I have witnessed in a few instances the most profound symptoms of cerebral anæmia in connection with severe epistaxis, metrorrhagia, intestinal hemorrhage, and after an operation for hæmorrhoids. The application of Junod's boot may deprive the brain of its blood, and thus induce cerebral anæmia mechanically.

5. *Poverty of the Blood.*—In this case the amount of the blood within the cerebral vessels may be normal, and still the brain be anæmic because the quality of the blood fails to properly nourish it. Examples of this are encountered in bottle-fed babies, and in connection with the fevers, leukæmia, chlorosis, tuberculosis, malarial cachexia, prolonged lactation, chronic suppuration, starvation, etc.

6. *Vaso-motor Disturbance.*—Strong and sudden emotions, shock, cerebral concussion, electric stimulation of the sympathetic cords, etc., are not infrequently followed by symptoms of cerebral anæmia, such as pallor, vertigo, insensibility of some of the special senses, and a weakness of the action of the heart.

Symptoms.—It is important but not always an easy matter to diagnose cerebral anæmia from the condition of cerebral hyperæmia. Not only is this discrimination important from a scientific standpoint, but the life of the patient may be imperilled by an error in diagnosis (especially in infancy) because the treatment of the two conditions is directly opposed.

Sudden cerebral anæmia produces symptoms that differ from those of the form which is gradually developed. In the former a "fainting fit" is produced. An attack of this character usually begins with dizziness, a sense of impaired vision, and a loss of the normal appreciation of sensory impressions. The patient becomes incapable of voluntary movement, gradually sinks to the ground and loses consciousness. The pupils dilate, the face becomes pale, the respirations are slow and shallow, and slight spasms of the muscles occur. Gradually the patient regains consciousness, and the other symptoms slowly disappear. In rare cases, however, death ensues without a return of consciousness.

In infants, cerebral anæmia is liable to be confounded with acute hydrocephalus. The child is restless and capricious; tosses about, gnashes the teeth, and cries out in sleep. The face is often flushed in the early stages, but tends to become pale. The temperature and pulse may be increased. Sleep is interrupted by attacks of crying in many instances. Twitchings of the limbs and even convulsions are liable to be developed. The fontanelle is depressed. Later in the attack the patient becomes insensible to light, noises, or objects held before the eyes; the eyelids remain half closed; the pupils, which were contracted in the early stage, now become dilated; the pulse flutters and is irregular; the respiration grows shallow, noisy, and infrequent; the sphincters are not controlled; and death comes at last, preceded by complete coma. Strabismus and rigidity of the muscles at the nape of the neck may occasionally be observed during the attack.

In older patients the symptoms of gradual development of cerebral anæmia differ (1) with the variety present—general or partial—and (2) with the severity (so to speak) of the anæmia.

The more prominent signs of the *general variety* are headache, vertigo, nausea, dimness of vision, and fainting or convulsive attacks. Muscular weakness, drowsiness, flashes of light before the eyes, roarings in the ears, a dread of mental or physical effort, tremor after exercise, and mental confusion may precede the attacks of syncope or convulsions.

The *partial variety* is commonly due to tumors, œdema, embolism, or thrombosis. The symptoms will vary with the area of the brain that is rendered anæmic. Motor paralysis (monoplegia or hemiplegia) may follow if the cortex of the "motor area" of the cerebrum is deprived of its nutrition. Again, aphasia may be the result of anæmia of the base of the third frontal convolution, the island of Reil, or the adjacent medullary substance. Vision may be impaired if the cortex of the occipital convolutions are affected or the other mass of gray matter with which the optic fibres are known to be associated. Finally hearing or smell may be impaired by anæmia of the temporal lobes, and tactile sensibility may be impaired if the parietal cortex be deprived of its normal blood-supply. Fig. 5 will enable the reader to appreciate the grounds for these statements. Some of the more important points in cerebral localization will be discussed later, chiefly in connection with cerebral hemorrhage.

Delirium and hallucinations are not infrequently observed in connection with cerebral anæmia. Occasionally the delirium may assume a maniacal type. In other instances melancholia may be a prominent symptom.

Nothnagel states that smell and taste are never affected in cases of cerebral anæmia. My own experience leads me to doubt the accuracy of this statement. It is more common to observe symptoms referable to the optic and acoustic apparatus, but hyperosmia and hypergeusia have been present in some cases that have fallen under my observation.

Differential Diagnosis.—The diseases most liable to be confounded with general cerebral anæmia are cerebral hyperæmia and hydrocephalus.

From *cerebral hyperæmia* the diagnosis is often difficult. The apparent cause is an important factor in the discrimination. Moreover, the pallor of the face, the fainting attacks, the dimness of vision, and drowsiness are characteristic of anæmia. The ophthalmoscope may enable the physician to detect anæmia of the retinal vessels. The vertigo of anæmia is diminished by a recumbent posture, and increased by standing. Finally, the effects of stimulants and the inhalation of a few drops of the nitrite of amyl will be markedly beneficial in anæmia, while such agents will increase the symptoms of congestion.

From *hydrocephalus* the diagnosis is to be made by the absence of a history of tuberculosis in the parents, the presence of some of the exciting causes of anæmia, and the age of the subject. There is probably little difference in the two diseases, as far as the condition of the cere-

bral vessels is concerned. The tubercular deposit at the base of the brain in hydrocephalus undoubtedly produces most of its effects by pressure upon the cerebral vessels.

Prognosis.—In adults, the prognosis in cerebral anæmia is favorable if we are able to relieve the exciting cause. In children, cerebral anæmia may prove fatal if following an exhausting diarrhœa, marasmus, or debility. Kussmaul states that when the pupils have become dilated a return of the mobility is a favorable omen.

Treatment.—The indications for treatment vary with the exciting cause. Stimulants and the inhalation of from four to eight drops of the nitrite of amyl three times a day by an adult will generally cause a rapid improvement in the symptoms after the cause has been removed. Alcoholic liquors should be given in small doses, and repeated frequently till the heart-power is increased and the symptoms show improvement. They should not be pushed beyond reasonable limits. The diffusible stimulants are only advisable when a rapid effect is sought for, or when alcohol disagrees with the patient. Opium tends to increase the flow of blood to the head; hence it is sometimes very happy in its effects when administered in one-quarter-grain doses three times a day for a few weeks.

When anæmia of the blood exists, iron and some of the bitter tonics are of great benefit. If hemorrhage has been a factor in the case, the recumbent posture should be maintained rigorously until the patient has regained strength and manufactured blood to fill the depleted vessels. Tying the arms and legs after a serious hemorrhage, in order to force the blood to the head, may sometimes be demanded. Raising the foot of the bed, upon which the patient is lying, upon two chairs will also help to determine blood to the brain. In obstetric practice, this manœuvre is often employed to arrest the brain symptoms after flooding.

During convalescence, restrictions regarding excessive physical or mental exercise should be placed upon the patient. All forms of excitement should be avoided. I have twice known insanity to follow emotional excitement, after this condition of cerebral anæmia has existed to marked degree.

The advisability of employing galvanism, although sustained by Hammond and other authorities of note, is to my mind questionable. Personally, I have seldom observed any decidedly beneficial effects from its use. Whenever it is employed, the current should, as a rule, be an extremely mild one, and the duration of its employment short. I greatly prefer static insulation, followed by sparks to the nape of the neck, when such applications are feasible.

Finally, care in regulating the diet and the functions of the abdominal organs is very essential to a complete recovery. I would again urge here the use of the hot-water treatment, which I have described on a

previous page in discussing cerebral hyperæmia. Any form of disturbance of the circulatory apparatus seems to be modified and generally improved by the action of heat upon the solar plexus. The treatment of the most important symptom of partial cerebral anæmia (aphasia) has been considered under the treatment of cerebral embolism.

CEREBRAL HEMORRHAGE.

The blood-vessels of the meninges or of the brain sometimes rupture, and thus allow of an escape of blood into adjacent structure.

If the extravasation be meningeal, its effects are exerted chiefly upon the gray matter of the convolutions (the *cerebral cortex*). If within the substance of the cerebrum, tracts of fibres are torn across by the escaping blood, and are thus separated from their connection with the cortical cells.

Intra-cerebral hemorrhages may exert pressure-effects upon the ventricles, in case the blood fails to enter these cavities, and thus create a more or less complete obstruction to the ingress and egress of the cerebro-spinal fluid.

Because the symptoms of pressure upon the different parts of the brain are observed in patients afflicted with tumors, depressed bone, abscess, and extensive exudation, as well as in connection with hemorrhage, any remarks made in reference to the localization of blood-clots applies as well to many of these conditions.

Morbid Anatomy.—The middle meningeal artery is the most frequent source of surface hemorrhage—the extravasation that occurs in connection with pachymeningitis being excluded from this head, as it is of inflammatory origin. This vessel of the brain is particularly liable to be involved in direct injuries to the cranial vault. Its area of distribution to the meninges corresponds approximately to that portion of the cerebral cortex which contains the motor centres (see Fig. 5). For this reason the blood extravasated from that vessel is particularly prone to press upon the motor convolutions beneath. Actual damage may be done to these cortical centres if the blood escapes in sufficient quantities to impair the structural integrity of parts beneath the clot. Otherwise the cortex is rendered simply anæmic at the seat of pressure. It may regain its function, in such a case, when the pressure is relieved by the use of trephine or the gradual absorption of the clot.

Structural changes in the motor convolution are almost invariably followed by a *descending sclerosis* of those fibres that are anatomically associated with the cortical cells destroyed. This sclerosis can often be traced into the substance of the spinal cord. In this way the motor function of Türck's columns and of the crossed pyramidal tracts in the spinal cord (Fig. 29) has been established beyond dispute.

The following simple diagram will possibly aid the reader in mastering the more essential anatomical facts that pertain to motor paralysis of cerebral origin. It is designed purely for the purpose of teaching, and must not be construed as a representation of the parts in their proper relations to each other :—

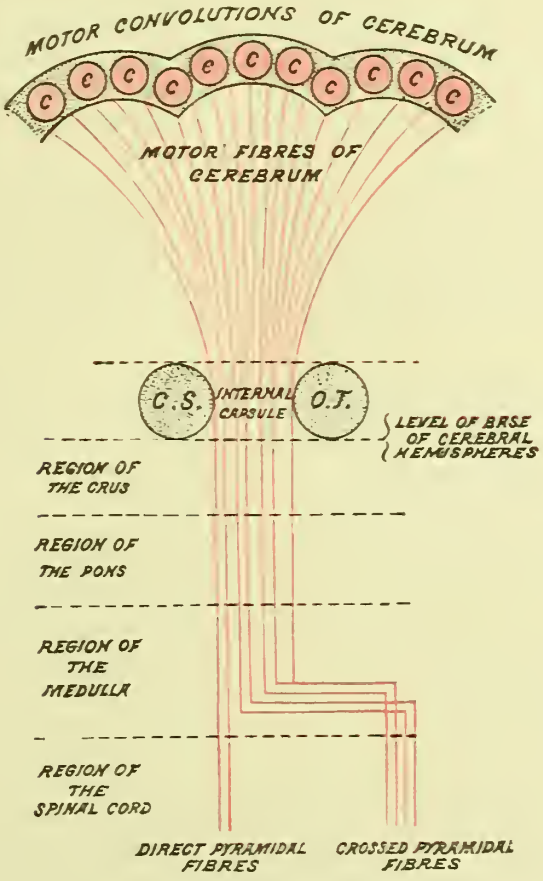


FIG. 79.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE EFFECTS OF CORTICAL AND NON-CORTICAL LESIONS OF THE CEREBRUM.

It will be perceived that the motor convolutions of the cortex contain special centres of motion (*C.*). From these centres fibres of the so-called "*pyramidal* tracts" can be traced through the medullary centre of each hemisphere (the white matter of the cerebrum) to enter the posterior half of the internal capsule that passes between the corpus striatum (*C. S.*) and the optic thalamus (*O. T.*). At the level of the base of the cerebrum these fibres are continued downward successively through the crus cerebri, the pons varolii, the medulla, and into the spinal cord. At

the lower part of the medulla the pyramidal tracts undergo a change. Some of the fibres (usually about 10 per cent.) pass down the same side of the cord in Türck's columns (see Fig. 29). The remainder cross to the opposite side of the cord and compose the so-called crossed pyramidal columns of the cord (see Fig. 32).

Flechsig has shown that the percentage of crossed to direct pyramidal fibres varies in different individuals. In a few cases all have been observed to cross, and in other instances all have been seen to pass directly into the cord without decussation. Both of these conditions, however, must be regarded as exceptions to the rule. Cerebral lesions that induce hemiplegia of the opposite side are commonly associated with a more or less extensive paresis of the same side,* on account of the direct pyramidal fibres. This paresis is, nevertheless, overshadowed by the hemiplegia, and is often unrecognized for that reason. In those rare cases where the hemiplegia is upon the same side as the cerebral lesion the direct pyramidal fibres are in excess of the crossed, if not exclusively present.

Surface hemorrhages are sometimes observed in connection with miliary aneurisms and thrombosis of the cerebral sinuses and the meningeal veins; also as a result of a collateral circulation following embolic occlusion of the middle cerebral artery. They may exist, furthermore, around localized morbid processes, such as foci of softening, tumors, abscess, etc.

Intra-cerebral hemorrhages may appear as small spots of discoloration if due to a rupture of the capillary vessels; or, if the ruptured vessel be a large one, as clotted masses within the white substance of the hemispheres, the basal ganglia, the crura cerebri, the pons, the cerebellum, and the medulla. In some cases the ventricles are more or less filled with blood. This is more liable to occur when the caudate nuclei or the thalami are involved.

The most common seat of intra-cerebral hemorrhage is within the substance of the caudate and lenticular nuclei of the corpus striatum and the thalamus of either hemispheres. The right side appears to be more frequently affected than the left, but the relative proportion is nearly equal as regards the ganglia. The pons Varolii and cerebellum are often the seat of clots.

The basal ganglia† of the cerebrum are nourished by vessels that pass

*The "direct pyramidal fibres" (Fig. 19) usually disappear in the middle dorsal segments. According to some observers a certain proportion of these fibres cross in the white commissure of the cord, and after crossing become associated with the cells of the anterior horn. Ferrier seems to incline toward the view that this is the rule rather than the exception,—a deduction which I cannot fully accept as proven.

† A term applied to the corpora striata and the optic thalami because they are situated near to the base of the cerebrum.

from the circle of Willis through the anterior and posterior perforated spaces. These ganglia lie in intimate relation with the paths of conduction of motor and sensory impulses. Whether any of the fibres of the internal capsule are structurally related with the cells that compose these ganglia is still a disputed point. The later investigations of Flechsig seem to disprove it.

Clinically the question in dispute is of no importance, because any *increase in size of these gray masses* (as would occur from a clot within them) would inevitably cause pressure to a greater or less extent upon the motor or sensory tracts of the internal capsule. Fig. 79 will make this apparent.

Again the caudate nucleus of the corpus striatum and the thalamus of each hemisphere enter into the formation of the ventricles;* hence any lesion of these ganglia would be liable to displace cerebro-spinal fluid. Such a displacement is believed by Duret to account for the *loss of consciousness* that usually accompanies attacks of intracerebral hemorrhage.

Again, certain fibres associated with the special senses of smell, sight, hearing, taste, and tactile sensibility run in the internal capsule and are liable to be destroyed by clots within the basal ganglia of the cerebrum.

The method of recovery from an extravasation of blood into the brain-substance is as follows: 1, the clot generally becomes encapsulated by the formation of a false membrane; 2, a serous exudation combined with fatty metamorphosis softens and dissolves the clot and the débris of brain-tissue, and changes them into a yellowish fluid; 3, bands of connective tissue form from the sides of the cyst so produced and traverse it in all directions; 4, after a lapse of time these connective-tissue bands contract and draw the sides of the cyst in apposition, the fluid contents becoming absorbed to a greater or less extent; 5, a stellate and pigmented cicatrix often forms. Apoplectic cysts are formed, as a rule, in about two months after the hemorrhage, in favorable cases.

I have already referred to the fact that secondary changes in nutrition are observed, in the case of destruction of the cortical cells, when the motor fibres are destroyed by intra-cerebral clots. We owe to Türck our first intimation of these secondary sclerotic and degenerative changes in nerve-fibres. His discovery,† which was for a time buried in the archives of the Vienna Academy of Sciences, has proved of inestimable benefit to anatomists, since it enables them to trace the course of special

* Wilder doubts if the thalamus actually forms a part of the floor of the corresponding lateral ventricle.

† A paper read in 1851 upon the results of pathological observation respecting the results of old cerebral lesions.

bundles of fibres within the substance of the brain and spinal cord. The degenerated fibres become very distinctly outlined from the healthy fibres in all transverse sections of the nerve-centres, and thus Nature

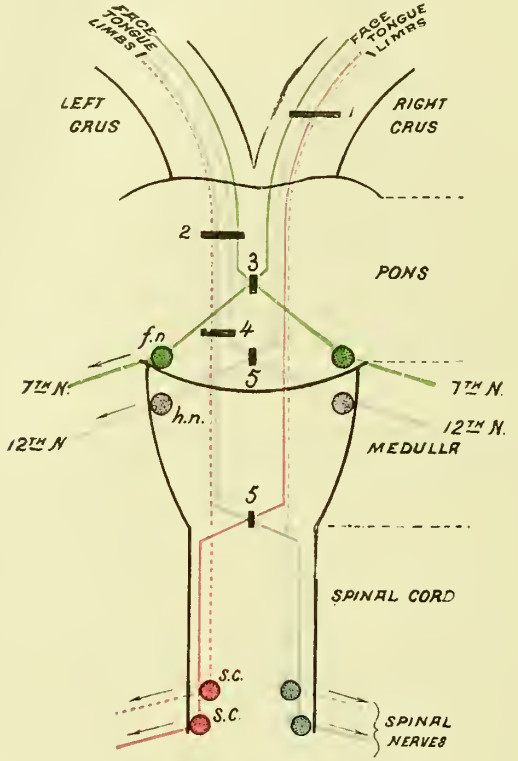


FIG. 80.—A DIAGRAMMATIC REPRESENTATION DESIGNED TO SHOW THE COURSE OF THE MOTOR TRACTS AND THE EFFECTS OF LESIONS OF THE CRUS CEREBRI, PONS VAROLII, AND MEDULLA OBLONGATA UPON MOTILITY. (Modified from Starr by the Author.) The *red fibres* represent the motor fibres which govern facial movements. These decussate in the middle of the pons. The *blue fibres* are connected with tongue movements. These decussate at a lower level in the pons. The *purple fibres* are the motor tracts, which preside over voluntary movements of the arms and legs. Some of these decussate at the lower level of the medulla (the "crossed pyramidal tracts"), while others do not decussate (the "direct pyramidal tracts").

Each of these three bundles of fibres are associated with a *group of motor cells* after leaving the crus. The facial fibres terminate in the facial nuclei (*f. n.*); the tongue fibres in the hypoglossal nuclei (*h. n.*); the pyramidal tracts in the cells of the anterior horns of the spinal gray matter (*s. c.*).

A lesion at 1 and 2 might affect any of these tracts separately, or various combinations of the three might exist, and manifest its presence by a *disturbance of motility* without affecting the sensibility of any part to impressions of touch, pain or temperature. If the tongue, face or limbs were paralyzed by such a lesion the parts would be deprived of motility upon the same side,—that opposed to the seat of the lesion.

A lesion at 3 would cause *facial diplegia*. This is at the middle level of the pons,—at the raphæ.

A lesion at 4 would cause *hypoglossal paralysis and hemiplegia of the opposed side.*

A lesion at 5 would cause *bilateral paralysis of the tongue.*

A lesion at 6 would cause *motor paresis of both arms and legs*, rather than a complete paralysis of motion. The non-decussating pyramidal fibres would still be unimpaired; hence some voluntary movements would be unaffected by the lesion.

Paralysis of the tongue, if unilateral, causes the tongue to deflect toward the paralyzed side when protruded, if bilateral protrusion of the tongue becomes impossible, and chewing, swallowing, and talking become very difficult.

perfects a dissection that no human hand could possibly make. We have been enabled to acquire of late many facts in anatomy which aid

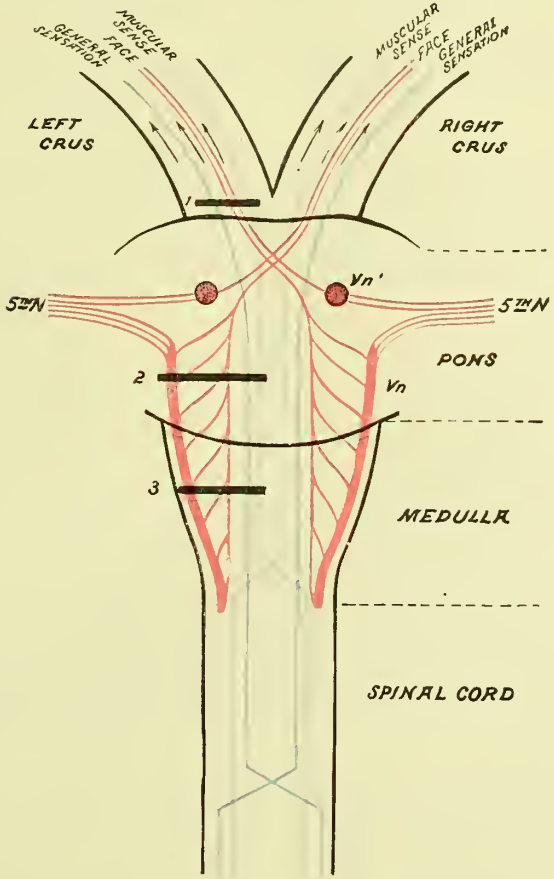


FIG. 81.—A DIAGRAMMATIC REPRESENTATION OF THE COURSE OF THE SENSORY TRACTS, AND THE EFFECTS OF LESIONS INVOLVING THE SAME WITHIN THE CRUS CEREBRI AND THE MEDULLA OBLONGATA. (Modified from Starr by the Author.) The red fibres represent the sensory fibres of the face which pass to the cerebral hemispheres by means of the pons and crus. They spring from two sets of nuclei (Vn and Vn'). The purple fibres represent the tracts for the "muscular sense." The blue fibres represent the tracts for the transmission of impressions of touch, pain, and temperature. The arrows indicate the direction of the impulses carried by each set.

A lesion at 1 (tegmentum of the crus or upper part of the pons Varolii) would cause *hemianæsthesia* of the *opposite side of the face*, and *also of the opposed limbs*, and *the opposed side of the trunk*. The muscular sense might also be disturbed on the opposed lateral half of the body.

A lesion at 2 or 3 (the lower part of pons in the formatio-reticularis or in the medulla) would cause *crossed hemianæsthesia*, the face being affected upon the side corresponding to the seat of the lesion, and the body and limbs upon the side opposed to the lesion. This statement holds true to all lesions which affect the tract below the point of decussation of the trigeminal fibres, viz., at the middle level of the pons.

us directly in determining the seat of localized lesions during life. Many of these have been mentioned in the first section of this volume. The

methods of research brought to professional notice by Türck, Fleehsig, Gudden, Fritsch, Witsig, and others have been of inestimable value in the study of neuro-anatomy and neuro-physiology.

Apoplectic clots may assume two forms: (1) those where the blood is collected into a circumscribed mass, a so-called "apoplectic focus," and (2) those where minute points of capillary extravasation are alone detected.

Apoplectic foci vary in size from that of a small pea to that of a large orange. They are commonly of the size of a hazel-nut, and are usually more or less globular in form. When blood is extravasated upon the surface of the cerebral hemispheres or into the substance of the pons, the clot generally assumes the form of a disseminated layer rather than a circumscribed mass. The number of foci that develop simultaneously is apt to vary. Not infrequently homologous regions in each hemisphere are attacked at the same time. Although it is the rule to encounter a single focus, several may exist in different parts of a brain and give unmistakable evidences of a simultaneous formation.

Respecting the relative frequency of clots in different parts of the cerebrum, Andral reports 386 cases, in which he found that the corpus striatum was involved in 61, the optic thalamus in 35, the centrum ovale in 27, and the centrum ovale and basal ganglia combined in 202. Rosenthal gives the statistics of 103 autopsies at the Vienna General Hospital, which show that the caudate nucleus alone was involved in 32 cases, the lenticular in 20, both nuclei of the corpus striatum in 8, the caudate nucleus and the thalamus in 7, the optic thalamus alone in 20, centrum ovale in 3, parietal lobe in 2, lenticular nucleus and other parts in 6, the basal ganglia of the two sides in 2. It thus appears that the lenticular and caudate nuclei of the corpus striatum were attacked in a very large percentage of all the cases, and that the thalamus ranks as the next most frequent seat of apoplectic foci.

Etiology.— *Vascular changes*, in the vast majority of subjects, have preceded a rupture of the cerebral vessels, unless it be dependent upon traumatism. Among the conditions that tend toward rupture, miliary aneurisms, atheroma, and fatty degeneration of the vessels stand foremost. The various causes of cerebral thrombosis and embolism may also indirectly produce a hemorrhage. These have been discussed in previous pages.

Degenerations of the brain-substance may have preceded the rupture of vessels. We not infrequently encounter sudden paralytic symptoms in connection with softening of the brain. In such cases, the softening is accompanied by secondary hemorrhage.

Certain *diseased conditions* of the organs and tissues may be factors in producing apoplectic extravasations. Among these may be mentioned

(1) simple hypertrophy of the left ventricle; (2) valvular lesions of the heart; (3) chronic nephritis, which induces changes in the coats of blood-vessels; (4) a congenital defect in the construction of the arterial coats or miliary aneurisms; (5) compression of the jugular veins or the superior cava from tumors, aneurism, etc.; (6) certain abnormal blood-conditions (chlorosis, scurvy, syphilis, typhus, gout, chronic alcoholism, Bright's disease, rheumatism, etc.).

Statistics show that *age* exerts a marked influence upon the development of apoplexy. The percentage increases gradually from the age of twenty to sixty.* It is rare before twenty except in infancy. After the fiftieth year the relative percentage becomes very large.

For some unexplained reason, a large proportion of cases are attacked between the hours of three to five in the afternoon, and two to four in the morning. Males are more commonly attacked than females. The cold seasons of the year seem to be especially liable to induce cerebral hemorrhage. The so-called plethoric habit has little, if anything, to do with apoplectic seizures.

Among the *exciting causes* of cerebral apoplexy may be mentioned all mental or physical conditions which tend to increase the heart's action or to suddenly intensify the blood-pressure.

Attacks of coughing or laughing; severe physical exercise; straining at stool; over-indulgence in alcohol; sudden rage, grief, or mental excitement; the sexual act; a recumbent position of the head; the eating of a hearty meal; the use of opium; a cold bath; and many other similar occurrences have been reported factors in producing a rupture of an artery whose walls have been impaired by the predisposing causes mentioned.

Symptoms.—Prodromal symptoms indicative of cerebral irritation are frequent in subjects inclined toward apoplectic attacks.

A headache of a dull and ill-defined character is often present on rising. Insomnia may have existed for some time. Vertigo may have been often noticed. The intellectual faculties are sometimes impaired to a greater or less extent. There may be flushing or pallor of the face, bleeding from the nose, ringing in the ears, persistent specks before the eyes,† nausea or vomiting, irritability of temper, a sense of weight or of numbness in the limbs, transient disturbance of speech, tremor of the muscles, and disseminated pains of a neuralgic type. I regard epistaxis in old subjects as a symptom of evil import, as a rule. If it accompanies the other prodromal phenomena of apoplexy it is particularly ominous.

* Loomis states that the increase of percentage never stops, and that the small number of persons who live after seventy years is not taken into the computation by those who limit the danger.

† These may be due to small extravasations into the retina.

It should be borne in mind that apoplectic attacks may often occur without any of the prodromal symptoms enumerated. On the other hand, it often happens that some of them have been persistent for a greater or less period of time. The evidence drawn from statistics regarding the prodromata of apoplexy are, to my mind, of little value as a basis of diagnosis. Vertigo, epistaxis, headache, muscæ volitantes, change in the disposition, a sense of weight or numbness in the limbs, drowsiness, lethargy, etc., do not justify in every case the alarm which a diagnosis of a liability to an apoplectic attack would necessarily create in a patient. I regard attacks of thickness of speech, slight paralyses of short duration, double vision from paresis of the ocular muscles, etc., as possessing greater clinical significance than the others; but even these may be due to vascular disturbances which are not always followed by cerebral hemorrhage. A physician cannot be too careful in expressing an opinion calculated to excite anxiety and alarm either in the mind of a patient or interested friends until the examination warrants a positive diagnosis.

The actual symptoms of "apoplectic stroke," as it is commonly called, will be modified (1) by the amount of blood that escapes, (2) by the damage that it causes to the brain-cells or nerve-fibres, and (3) by the seat of the clot.

The *onset* of an apoplectic attack may be sudden or gradual. The situation of the clot and its size will determine the character of the initial symptoms. Clots in the medulla, pons, or cerebellum are liable to fell the patient to the ground without warning, as if struck by a blow, even if the hemorrhage be small. A hemorrhage into the ventricles may do the same, and, if extensive, may sometimes produce instantaneous death. These cases do not produce the typical form of attack, however, because they are less frequent than those where the hemorrhage occurs in or upon the cerebral hemispheres. In many instances the coma comes on gradually, and is preceded by pain in the head, nausea, confusion of the intellect, dizziness, incoherent speech, and other of the prodromata enumerated in preceding pages. Occasionally the attack is ushered in by clonic convulsions, or by a paralysis of the arm, leg, face, eye, or some other part. I recall a case where one of my patients was seized with a weakness of the leg, which was followed by paresis of the arm, then by complete paralysis of that side, then by stupor which deepened rapidly into coma, and finally by convulsions before death ensued. I have met cases where coma did not occur throughout the attack. Trousseau, Andral, and others report similar instances.

The coma of apoplexy generally becomes profound, and lasts for hours or even days. The muscles are relaxed, the face is usually red or cyanotic, the abolition of consciousness is complete, the head and eyes

may be rotated to one side, the pupils are apt to be irregular, the temperature of the body falls to about 96.5°, the breathing may be regular and quiet, or it may be stertorous; finally, the urine and feces may be passed involuntarily.

Although many theories have been advanced to explain the mechanism of apoplectic attacks, I believe that all may be interpreted as resulting from the following conditions: (1) from the disturbance that the lesion has created upon the intracranial circulation directly by pressure; (2) from the displacement of cerebro-spinal fluid from the ventricles or the cranial cavity, thus disturbing the circulation of the organ in an indirect way; (3) from an increase of intra-cerebral pressure; and (4) from direct injury done to the nervous elements, and the irritation of surrounding parts.

Respecting the *duration of life* in severe cases, Abercrombie reports an instance where death occurred in five minutes after the attack; but, as a rule, it seldom occurs before two or three hours, and it may be delayed for one or two days. Prolonged coma may induce pulmonary œdema, and lesions of the medulla or pons may interfere with the vagus nerve, and thus produce death. I have never observed a case of recovery where the coma has lasted more than two days. A very marked rise in temperature above the normal point is a very serious omen.

In favorable cases, *consciousness* is regained gradually after a short interval of coma, which has lasted for only a few hours. The reflex excitability of the limbs usually returns before consciousness, as detected by irritating the skin with a pin or by tickling the soles of the feet. The temperature of the body returns to the normal standard. After the patient begins to exhibit evidences of consciousness, a sense of pain in the head and of general discomfort is complained of; the answers to questions are inappropriate, or badly articulated; a drowsy condition persists; the patient is apt to be apathetic, peevish, or surly; the movements of the muscles not paralyzed are feeble; and, in some cases, delirium may be developed. We are apt to encounter a rise of temperature on the second, third, or fourth day, and other febrile manifestations. Pain in the paralyzed limbs is frequently complained of by these patients. The emotions are not well controlled. The appetite and the habit of normal sleep is not usually regained for some months. Whenever cerebritis is to follow, the symptoms of that affection will usually be developed by the beginning of the second week.

In order to interpret the more common symptoms, some knowledge must be had of the functions of the component parts of the brain and the course of the more important tracts of fibres. A close study of Figs. 3 and 5 will aid the reader in appreciating the results of cortical lesions. Subsequent diagrams will help to interpret the results of

pressure upon the fibres of the internal capsule. Lesions of the basal ganglia, the crus cerebri, the pons, the cerebellum, and the medulla have already been considered separately in this volume.

It may assist the reader if we start with the statement that the existence of suddenly-developed lesions within the cranial cavity may be indicated by some or all of the following symptoms:—

1. Motor Paralysis, which will vary in its extent, duration, and degree.
2. Sensory Paralysis, which will vary in its extent, duration, and degree.
3. Disturbances of Consciousness.
4. Disturbances of the Special Senses.
5. Abnormal Attitude of the Head and Eyes.
6. Tremor, which may present one of several forms.
7. Changes in the Pupils, Pulse, Temperature, and Respiration.
8. Abnormal Phenomena of the Bladder, Rectum, Kidney, etc.
9. Mental Impairment.

In the first section of this work, attention has been called to some of these symptoms. It is not necessary to repeat them here. A few of the more important facts may, however, be summarized.

Cerebral lesions which affect the motor convolutions or the fibres that spring from them induce either a *monoplegia* or a *hemiplegia* of the opposite side.

The duration and degree of the paralysis will aid in determining the severity of the structural changes that the lesion has produced. Paralysis of motion may be recognized, even when profound coma exists, by tests that have been mentioned in previous pages.

If a limb is paralyzed it will drop inertly if raised. Tickling the soles of the feet will often cause the patient, even when partially comatose, to draw up the lower limbs when not paralyzed. If this fail, painful impressions, as a pin thrust, will usually create sluggish movements in comatose subjects if paralysis be absent.

Monoplegia, or that form of paralysis in which a distinct group of muscles is paralyzed, indicates a circumscribed lesion that *impairs the free action of those cortical motor centres* which preside over the muscles affected. The type is a guide to the seat and extent of the lesion (Fig. 3).

A monoplegia may usually be diagnosed from paralysis of a spinal nerve by the fact that the muscles paralyzed are not supplied by one nerve.

We encounter the *crural type* of monoplegia when the superior parietal convolution of the opposite cerebral hemisphere is the seat of the

lesion, the *brachial type* in lesions of the upper part of the ascending frontal and the base of the first and second frontal gyri,* and the *facial type* when the middle of the ascending frontal convolution is involved. Lesions of the ascending parietal convolution are apt to produce a *monoplegia of the hand.* The coördinated movements of speech become affected (*aphasia* of the motor type) when the base of third frontal gyrus, the island of Reil, or the medullary substance which carries the fibres connected with the cells of these regions, are affected by the lesion. Figs. 3 and 24 will explain these deductions, and pages 50 and 51 relate to this subject more fully than is deemed wise here. The late views of Horsley are there reviewed.

Hemiplegia, or paralysis of one lateral half of the body, may indicate a lesion either of the brain or of the spinal cord.

If of cerebral origin, the lesion must affect the greater part of the fibres which compose the pyramidal tract of the opposite side (Fig. 29); hence it is seldom cortical, as it would have to be sufficiently large to destroy the function of the entire motor area. As a rule the lesion is confined either to the white substance of one cerebral hemisphere, the basal ganglia, the internal capsule, the motor bundles of the crus and pons, or the anterior pyramids of the medulla.

If the hemiplegia be of spinal origin, the lesion must be situated high up in the cord (above the origin of the nerves to the upper extremity) and exert its effects upon the lateral half of the cord that corresponds to the motor paralysis.

In the first section of this work the varieties of motor paralysis have already been discussed in a general way. Later in this volume, the special types will be given further consideration. It may be well to give here, as an aid to the study of the various types, a few of the clinical facts that will prove of aid in diagnosis:—

1. Cerebral paralyses occur chiefly on the *opposed side* of the body below the head. This is true of both motor and sensory paralyses.

2. Motor paralysis of cerebral origin is *liable to be associated with more or less disturbance of sensation* when the lesion is non-cortical. This is not the case, as a rule, when the lesion is situated upon the surface of the brain.

3. When *sensory* and *motor paralyses coexist*, as a result of a cerebral lesion, they are upon the same side; the reverse is true of spinal lesions.

4. Lesions within the cranium, which *cross the mesial line*, are liable to produce paralysis upon both sides of the body.

5. *Lesions of the base of the brain* are more liable to produce paralyses of cranial nerves than are those of the hemispheres or basal ganglia. Vomiting and choked disk are also frequently observed in these cases.

* The term "gyrus" is synonymous with "convolution."

6. The sensory areas of the cortex commonly give rise (when circumscribed lesions tend to impair or destroy their functions) to disturbances of vision, hearing, smell, or touch. If the lesion be very extensive, hemianæsthesia may be produced. Munk, Tripier, and Moeli have lately shown that the cortex of the entire motor area, as well as the remaining parietal convolutions, must be destroyed in animals to produce complete hemianæsthesia. Hence it is seldom of cortical origin.

7. *Consciousness* is not usually lost, at the time of the attack, with purely cortical lesions. Apparent exceptions to this statement occur; but they are to be attributed to effects exerted by the lesion upon deeper parts.

8. *Epileptic attacks* (which are characterized by the development of paralysis of a transient character after the fit) indicate an *irritation of the cortical motor centres* by the lesion. This symptom is often spoken of as "Jacksonian epilepsy," being named after the author who first interpreted its phenomena correctly (Hughlings-Jackson).

Cortical Cerebral Lesions may be indicated (1) by monoplegia of the opposite side; (2) by disturbances of some special sense; (3) by the presence of consciousness at the time of the attack; (4) by an early rigidity of the paralyzed muscles; (5) by circumscribed pain at the seat of the lesion, which may be elicited or increased by percussion over the lesion; and (6) possibly by Jacksonian epilepsy, if the lesion creates simply an irritation of the cells of the cortex.

When sensory and motor disturbances coexist with a purely cortical lesion it indicates that the lesion involves both the motor and sensory areas of the cortex. Trephining would be contra-indicated in such a case, because the lesion is of necessity diffused over a large area. The existence of a well-defined traumatic monoplegia without sensory complications is an indication for immediate surgical interference, provided that the paralysis is on the side opposed to the injury. The situation of the cortical motor centres are of assistance also in determining the seat at which to trephine for circumscribed lesions within the skull that are not of a traumatic character.

The convolutions of the *frontal lobes* are not associated with motion, excepting the ascending, and the bases of the first, second, and third frontal convolutions. Outside of this area lesions of the frontal lobe apparently produce no symptoms. If Broca's centre be destroyed, *motor aphasia* follows.

Irritative lesions of the occipital convolutions sometimes tend to produce *colored perception* of objects and other *ocular spectra*. The power of vision, as well as the memory of past visual impressions, seems to be markedly impaired by lesions of this lobe.

The convolutions of the *temporal lobe* are associated with the special senses of smell and hearing. Some cases of aphasia have been

induced also by lesions of this lobe,—the so-called cases of "word-deafness."

Our ability to localize lesions of the sensory regions of the brain is less positive than of the motor area.

The *parietal lobe* is physiologically associated with the tactile sense, as far as we are able to judge by well-reported cases.

Cortical paralysis may often be *transitory*, if the lesion be slight and superficial; or it may be *permanent*, if deep and impinging upon the medulla oblongata.

Secondary degeneration appears to follow destruction only of the convolutions of the motor area and the para-central lobule.

Intra-cerebral Lesions.—We are now prepared to discuss the effects of intra-cerebral hemorrhage.

It may be well to preface our remark upon this head by the general statement that such lesions may be accompanied by profound motor paralysis (usually of the hemiplegic type); a loss of consciousness, as a rule, at the time of the attack; simultaneous paralysis of sensation (more or less marked); marked disturbances of some or all of the special senses, and late rigidity of the paralyzed muscles. These symptoms are indicative of lesions within the substance of the cerebrum rather than upon its surface.

Some of these symptoms (especially hemiplegia, hemianæsthesia, hemianopsia, loss of consciousness, and impairment of the special senses) have been discussed in the first section of this volume. The reader will find Figs. 6, 21, and 36 of assistance in following the deductions given.

A few facts may be given here as a summary of previous pages with possible advantage.

Hemiplegia.—A paralysis of the lateral half of the body may occur in connection with lesions of the cerebral hemisphere. If this form of motor disturbance coexist with a slight or severe *impairment of sensation upon the same side*, the existence of an intra-cerebral lesion may be strongly suspected.

This combination is to be attributed to pressure upon the tract of fibres known as the "*internal capsule*" of *the cerebrum;* because the fibres of the middle part of this tract are motor, while those of the posterior one-third are sensory in function. Both the motor and sensory fibres of this tract decussate in order to supply that lateral half of the body which is opposed to the cerebral hemisphere through which they pass.

The situation of the internal capsule can be made clear to the reader by a diagram (see Fig. 82). Let us suppose a section to be made from the forehead to the occiput at such a level as to intersect the masses of gray substance buried within the cerebral hemispheres, and known as the

"basal ganglia." We encounter in such a section three prominent masses of gray substance in each hemisphere,—the caudate nucleus, the lenticular nucleus, and the optic thalamus. The caudate and lenticular nuclei are but halves of the same body—the corpus striatum—which are separated in the plane of the sections by fibres of the internal capsule. The optic thalami are united to each other by the gray commissure which crosses the mesial line of the brain. This is not shown in the cut.

Now, to understand this diagram, let us suppose that a mass of fibres like hairs are thrust vertically to the plane of the section between the

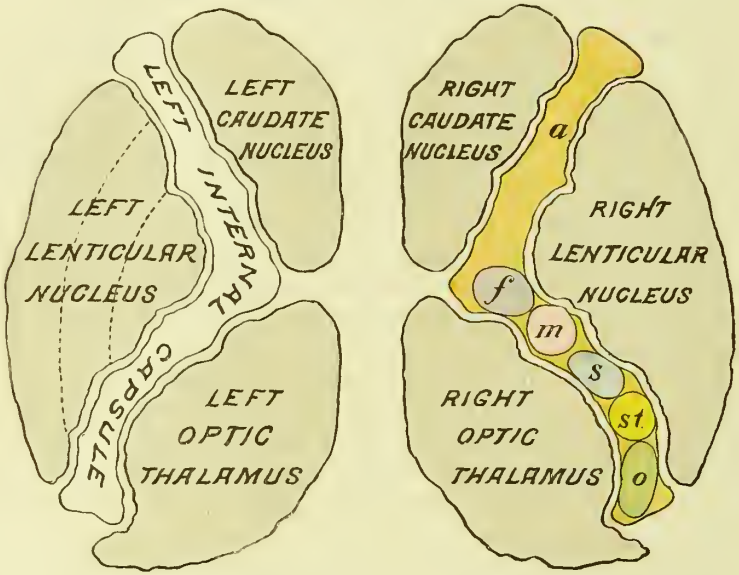


FIG. 82.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE A HORIZONTAL SECTION THROUGH THE CEREBRAL HEMISPHERES AND THE BASAL GANGLIA, SHOWING THE "INTERNAL CAPSULE" OF THE CEREBRUM.—The lettering of the figure will be explained in the text. It may be remarked here that similar subdivisions of the internal capsule exist in the left half as schematically depicted in the right half of the figure. Some of the statements made with reference to the subdivisions of the internal capsule are not to be considered as incapable of modification by the results of subsequent research. The bend observed in the internal capsule is termed "the knee" of the capsule. *a*, the so-called "caudo-lenticular" portion of the capsule; *f*, *m*, *s*, *st*. and *o*, constitute collectively the so-called "thalamo-lenticular" portion of the same.

lenticular nucleus of each hemisphere and the adjacent caudate nucleus and thalamus. Such fibres would constitute those of the internal capsule of the cerebrum. The capsular fibres arise from the cerebral convolutions above the plane of the section and become gathered and compressed into a bundle which presents the peculiar outline shown in the figure. After they escape from the cerebrum they are continued downward into the crus cerebri of the corresponding side (Fig. 11). They cease to be capsular fibres (properly speaking) when they leave the limits of the basal ganglia of the cerebrum.

It will be observed that the right internal capsule is divided in the diagram into distinct regions. The region (*a*) contains fibres whose distribution and functions are not as yet thoroughly understood, although they are probably indirectly connected with the cerebellum; the region (*f*) probably contains fibres going to the face exclusively; the region (*m*) appears to contain fibres of motion to the opposite extremities and lateral half of the body; the region (*s*) contains sensory fibres to the same; the region (*s t*) contains the fibres of the motor speech-tract (Wernicke), shown also in Fig. 24. Finally, the region (*o*) contains fibres that are apparently designed to join the optic nerves with the convolutions of the occipital lobes, and possibly some other fibres whose function is not yet ascertained.

Now, it must be apparent to all that any hemorrhage into the substance of the caudate nucleus, the lenticular nucleus, or the optic thalamus of either hemisphere, or the development of any morbid condition that would tend to enlarge them, would create pressure upon the adjacent internal capsule and affect its component fibres. The seat of the lesion will greatly modify the results of such pressure. If the anterior part of the capsule posterior to its "knee" be pressed upon, motor effects will follow; if the part still farther back be involved, the general sensory tract and the optic fibres may be affected. Both sensory and motor effects may be simultaneously produced by lesions of the thalamus or lenticular nucleus.

Lesions confined to the internal capsule do not differ materially in their effects upon motion and sensation from lesions of the masses of gray matter that lie adjacent to it. In either case, the compression of fibres or their actual destruction will result in the separation of the limbs and trunk from connection with large areas of the cortex. A lesion of moderate size in the region of the internal capsule would produce as profound effects upon motion or sensation as would one of extreme size if confined to the cortex.

If an apoplectic clot within the substance of the brain does not produce death by direct injury or the filling of the ventricles, the patient generally recovers consciousness at the end of several hours. The mental faculties, however, are more slowly regained. The memory is confused and the movements of the tongue imperfect for some hours after the patient seems to be conscious. We have reason to anticipate death, if profound coma exists and is extremely prolonged; if the pupils remain immoble; if the sphincters are relaxed; if the pulse is slow; and if the respirations are markedly diminished in frequency. The reflexes may be abolished. The respiratory and circulatory centres are liable to be paralyzed if the hemorrhage involves the ventricles or the medulla oblongata.

In favorable cases, the paralysis of motion developed at the time of

the attack gradually improves; but it seldom disappears entirely. The upper limb is more persistently affected than the lower. Nothnagel explains the fact that the lower limb is the first to improve after a hemiplegic attack on the ground that the movements of the healthy side tend to call into play "associated movements" of the unhealthy member, which are more easily excited in the leg than in the arm. He supports this view by the clinical observation that those movements in which both legs are employed (as in walking for example) are performed by the enfeebled limb for some time before individual movements of it are possible. He also brings forward many illustrations of associated movements in cases where paralysis of the face and upper limb has existed. The extensors of the fingers and wrist are usually the last to improve. In exceptional cases, the lower limb is the last to show improvement. After a lapse of time, post-paralytic contracture of the flexor muscles of the arm and fingers is generally observed. The characteristic gait of the

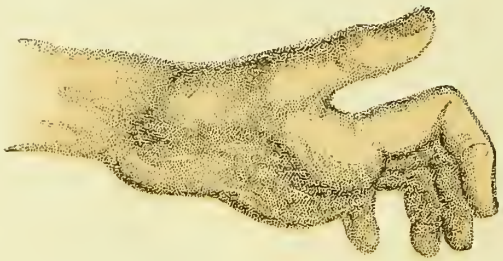


Fig. 83.—One of the Attitudes of a Hand Caused by Post-Hemiplegic Contracture. (From a photograph.)

hemiplegic subject is hardly to be mistaken for any other condition. (See page 162.)

The *face* may be turgid during the apoplectic attack, or pale. The former condition is most common. As the coma deepens, asphyxia is manifested by a dusky and livid countenance. It is stated by some authors that a pale face, if persistent during the attack, indicates a gradual hemorrhage.

Paralysis of the facial nerve alone has been observed by Duplay, Gruveilhier, Chevostek, and Dupuytren (quoted by Nothnagel) as a result of cerebral hemorrhage within the thalamus and corpus striatum.

The marked relaxation of all the limbs which is commonly observed in connection with apoplectic attacks of a severe type is to be attributed chiefly to an anæmia of the parts adjacent to the clot. This anæmia has been produced by the pressure consequent upon the extravasation. The compensatory changes which take place in the capillary vessels soon overcome the anæmia; hence the muscular relaxation disappears rapidly in those members whose nervous communication with the cerebral hemi-

spheres has not been severed by the actual destruction done to fibres by the escaping blood. Should cerebral œdema be an additional factor in its causation (as it often is), the muscular tone will be more slowly regained.

The fact has been mentioned that some of the fibres of the internal capsule terminate, anteriorly, in the *motor convolutions* of the cerebral cortex. Although there are still some neurologists of note (chiefly Goltz and his followers) who deny the value of the late attempts of Fritsch, Hitzig, Broca, Ferrier, Charcot, Hughlings-Jackson, Pitres, Landouzy, Exner, Chouppe, and a host of others, to locate special centres within the convolutions of the cortex, clinical and pathological observations are constantly being brought forward in support of the more generally accepted views. The region which embraces these motor centres appears, however, to be somewhat limited. A critical review of the recorded cases shows, I think, beyond cavil, that the white centre of each hemisphere of the cerebrum, as well as the cortex, may in some instances be extensively diseased or injured without any motor or sensory results which can be determined. Pathological evidence seems to demonstrate, however, that the region so impaired must not be situated where *fibres of the internal capsule*, which lie posteriorly to its knee (Fig. 82), suffer destruction or pressure if we expect to meet with negative results. Abscesses of immense size have been found in the anterior part of the frontal lobe, as well as in certain portions of the temporo-sphenoidal lobes of the cerebrum without any sensory or motor paralysis during life to indicate the existence of such a lesion. Tumors, softenings, and the most severe types of traumatism have likewise occurred without creating serious effects. Certain tests have been referred to on page 183, which may aid materially in the diagnosis of lesions of the white substance of the cerebral hemispheres.

In the case of the parietal, occipital and temporo-sphenoidal lobes, to which some of the posterior fibres of the internal capsule are distributed, sensory and psychical symptoms in addition to disturbances of nerves of special sense have been observed to follow circumscribed lesions. A careful consideration of such cases demonstrates the functions of these convolutions more or less clearly. Some arguments have been advanced of late to prove a relationship between the occipital lobes and the mental faculties in opposition to the more commonly accepted and probably correct doctrine that the frontal lobes are alone connected with the highest intelligence. The temporal lobes seem to exert an influence upon the special senses of smell and hearing. An apparent connection of the optic and auditory functions with the cerebellum and optic thalamus exists. The bearing of morbid phenomena of the special sense of sight upon diagnosis has been considered in previous pages.

Let me suggest, that it is by no means certain that lesions, which primarily effect the constricted portion of the internal capsule, may not, in themselves, create sufficient pressure upon the corpus striatum and the optic thalamus to cause interference with the free action of some of the *special centres* which are said by Luys to exist within those bodies. If this be the case, many of the interesting phenomena due to lesions of the optic thalamus, would *coexist* with those symptoms of disease within the internal capsule already mentioned. Ritti's views respecting the relations of the optic thalamus to hallucinations, and those of Luys pertaining to its olfactory, optic, and acoustic functions have a special interest in this connection.

Sensory Paralysis.—In most cases of intra-cerebral hemorrhage, sensation is impaired to a greater or less extent upon the opposite lateral half of the body. Both electro-cutaneous and electro-muscular sensibility may be diminished. The anæsthesia may even affect the smaller joints. Fig. 81 will aid the reader in localizing lesions which involve the sensory tracts.

The restoration of sensibility after an attack of apoplexy, while often not complete, is usually more rapid than that of the power of motion. Subjective sensations may remain, however, such as formication, hyperæsthesia, the feeling of cloth upon the skin, etc. I have met with exceptions to this rule, in which the anæsthesia remained complete after the motor paralysis had nearly disappeared. I believe that in these cases, the lesion affected the sensory fibres of the internal capsule more severely than the motor.

The abolition of sensation is not always of the same kind. In rare instances, sensations of touch are not impaired, while those of temperature and pain are. The appreciation of temperature and pain may also be separately destroyed.

Disorders of Intelligence and Coma.—These may both precede and follow a cerebral hemorrhage. The more common of these effects are evidenced in the memory and emotional faculties. Some patients cry more or less persistently after an apoplectic stroke. Occasionally uncontrollable fits of laughter are induced. Insanity is comparatively infrequent.

The memory is often seriously affected, and the patient relapses into childish methods of thought or into a state of apathy. From personal observation, I am inclined to think that the amount of destruction done to the brain influences these sequelæ, rather than the seat of the lesion. They are more marked in cases where the symptoms of the attack have been severe than when mild.

The abolition of consciousness that accompanies some attacks and not others, is to be attributed, in my opinion, to the displacement of

cerebro-spinal fluid from the ventricles, as explained by Duret. This symptom is certainly less frequent in cortical lesions, that seldom affect the ventricles, than in intra-cerebral lesions, which do so to a marked degree.

Impairment of the Special Senses.—Respecting the possibility of existence of special centres of smell, sight, hearing and sensation within the substance of the thalamus there are differences of opinion among authors of note. Some clinical facts point strongly to a relationship between nerve-fibres connected with certain special-sense perceptions and the internal capsule. It is impossible, with our present knowledge, to definitely place the situation of all the cortical centres which preside over the various special senses, or the course of separate fibres which seem to be associated with them; but we are forced to admit that some of the fibres of the posterior part of the internal capsule have a direct or an indirect association with smell, sight, hearing, tactile sensation, and taste.

One peculiar fact cannot be omitted, however, viz., that hemianopsia sometimes occurs in connection with lesions of the internal capsule, with a difficulty in discrimination of color.

When the radiating fibres of the internal capsule are involved in a lesion which creates a gradually increasing pressure (as in the case of tumors which grow slowly) the *fundus of the eye* exhibits morbid changes in the region of entrance of the optic nerve which are of value in diagnosis. The condition so produced is commonly known as the "*choked disk.*" (See Fig. 87.)

In exceptional cases of destruction of the internal capsule, the *sense of smell* has been found to be abolished on the side opposite to the seat of the lesion. This fact requires special consideration, as it has been shown that the centre proper for olfactory perceptions seems to be in the hemisphere of the same side. Meynert claims, however, to have demonstrated the existence of an olfactory chiasm in the region of the anterior commissure (in animals where the bulbs are largely developed); and fibres have been traced in the region of the "subiculum cornu ammonis," or the tip of the temporo-sphenoidal lobe, which connect the olfactory centres with each other. Some experiments of Ferrier tend to disprove the decussation of the olfactory paths in the anterior commissure; so that the question still remains unsettled. The sense of smell is more commonly affected in the nostril of the side which corresponds to the seat of the lesion.*

Among the fibres of the internal capsule which are distributed to the temporo-sphenoidal lobe some appear to have some association with

*Ferrier reports a case where smell and taste were simultaneously abolished by a blow upon the top of the head. Ogle records a similar instance.

the *sense of hearing;* but experimentation upon animals to determine the exact seat of the centres of hearing and the effects of their destruction are exceedingly difficult, because the evidences of impairment of this sense are more or less vague. Ferrier thinks, however, that the *superior temporal convolution* is unquestionably connected with acoustic perceptions. The area which he maps out as acoustic in function is quite extensive, and the late researches of Starr respecting the effects of cortical lesions in man upon the sense of hearing tend to confirm this deduction.

The cortex of the *parietal lobe* seems to be chiefly connected with *tactile sensibility*, because its destruction has been found to create a total loss of that sense on the opposite side of the body. (Munk, Tripier, Moeli, and others.)

As regards *taste*, the results of experimentation upon the monkey tribe seem to point to the *lower portion of the middle temporal convolution* as the probable seat of the centres which are related to that sense.* When this region is subjected to irritation, certain reflex movements of the lips, cheek, and tongue are observed, which seem to point to an excitation of the gustatory sense. Its destruction causes abolition of taste.

Tremor.—A symptom which points strongly to an existing lesion of the internal capsule is *choreiform movements* following hemiplegia or hemianæsthesia. These movements vary in type and degree. In some cases, the movements exhibit the peculiarities of athetosis, the fingers or toes being thrown into active motions which cannot be controlled by the patient; in others, true ataxia may be developed; again, the spasmodic movements partake of the character of genuine chorea; finally, a tremor, more or less marked may be detected.

It is not uncommon to find that both hemiplegia and hemianæsthesia may coexist with these post-paralytic forms of spasmodic disease; but one usually overshadows the other, the hemiplegia being, as a rule, the more marked. How we are to explain these late phenomena is not definitely settled. They are probably to be classed with other morbid manifestations which paralyzed muscles sometimes exhibit, chiefly that of "late rigidity" so often seen, concerning the cause of which many conjectures have been advanced, but nothing of a positive nature demonstrated.

Effects upon Temperature.—It has been observed that lesions of the internal capsule, if very extensive, are often followed by a very marked *rise in the temperature* of the body. We have yet much to learn

*This may help to explain the fact that injuries received upon the vertex and occipital protuberance cause, in some instances, an abolition of taste. The temporal lobe being injured by concussion against the adjacent bone.

concerning the vaso-motor centres which are variously disposed within the substance of the brain and spinal cord.

Abnormal Posture of Head and Eyes.—We have now considered some of the more prominent symptoms which are produced by lesions of the internal capsule, and I pass to one which I believe to be of great value in aiding the recognition during life of an extensive and rapidly developing lesion of the white centre of the cerebral hemisphere, viz., *conjugate deviation of the eyes and head.*

When, in connection with rapid softening or an extravasation of blood into the substance of the cerebrum above the level of the basal ganglia, this peculiar symptom is developed (either simultaneously with or following paralysis and coma), the patient's head and eyes will be observed to be *turned constantly away from the paralyzed side* and toward the side which is the seat of the lesion. Various attempts have been made by late authors to throw discredit upon the clinical significance of this symptom as particularly indicative of a lesion of the cerebral hemisphere, but I am convinced that it is a valuable differential sign. Ferrier has demonstrated that a cortical centre, which he locates in the first and second frontal gyri near to their bases, presides over conjugate movements of the head and eyes, and causes dilatation of the pupils. He attributes this symptom, when occurring in connection with hemiplegia of cortical or ganglionic origin, to the unantagonized action of the corresponding centre of the uninjured hemisphere, thus explaining the fact that the distortion is toward the side of the lesion. Clinical evidence of the correctness of this view has been brought forward by Hughlings-Jackson, Priestly Smith, Chouppe, Landouzy, Carroll, and others; and, in some cases reported, the situation of the lesion has been verified by pathological observation. The opportunity to record pathological observations upon cases where this symptom was well marked during life is, unfortunately for science, a comparatively rare one. It is impossible, therefore, to speak positively concerning the diagnostic value of this symptom, although the weight of clinical evidence seems to be strongly in its favor.

Special Symptoms of Cerebral Lesions.—In connection with cerebral hemorrhage and other lesions that disturb the functions of the brain mechanically, special types of paralysis and other evidences of impairment of special functions are liable to be encountered. A few general statements are all that space will allow of in reference to them. Figs. 77, 78, and 79 will aid the reader in appreciating the clinical significance of many symptoms caused by focal brain-lesions.

Of the *ocular muscles*, the internal rectus of one side may alone be paretic. Dilatation of the pupil, ptosis, and external squint coexist when the motor oculi nerve is paralyzed. If these symptoms accompany a hemi-

plegia of the opposite side, the lesion is within the *substance of the crus cerebri*, and on the side corresponding to the eye symptoms. In such instances, it is not uncommon to observe a paretic condition of the lower part of the face. (See Fig. 77.)

If the *facial muscles* are paralyzed upon one side and hemiplegia coexists, the seat of the lesion is within the *substance of the pons Varolii*. If in the upper part of the pons, the facial and body palsy will be upon the same side. If in the lower part of the pons, the facial and body paralysis will be on opposite sides. In rare cases, a paresis of individual facial muscles has been observed to follow a cerebral clot. (See Fig. 77.)

The *sense of smell* is liable to be lost in one nostril when the caudate nucleus, optic thalamus, or the internal capsule of the corresponding cerebral hemisphere is the seat of the lesion.

A peculiar form of blindness, known as *hemianopsia*, has been described in the first section of this volume. It may assume several types. The clinical deductions to be drawn from this symptom have been already given.

Ataxic manifestations, occurring in connection with evidences of impairment of the sense of sight, open a wide field for speculation. The proximity and intimate structural relations of the *cerebellum* with the optic lobes, basal ganglia, crus, and medulla, suggest the possibility of cerebellar lesions when these two symptoms are present to a marked degree.

The clinical value of *aphasia*, as a diagnostic symptom, has been discussed in preceding pages. (See index.)

In the closing pages of the first section a general summary of the guides to the localization of cerebral lesions has been given. Many additional points have been given there in detail.

The so-called "*contracture of muscles*," which occurs in connection with hemorrhagic foci and local diseases of the brain, deserve a passing notice. Rigidity of muscles may be divided, clinically, into those which accompany the onset of the exciting lesion, those which develop soon after the onset, and those which appear late as post-paralytic manifestations (usually from two to five months after the attack).

The first form is most commonly observed in connection with cortical lesions. It disappears within a few days. It is due to mechanical irritation of the cortical centres.

The second form is to be attributed, according to the researches of Todd, to the irritation produced by a reactive inflammation in the neighborhood of the cerebral lesion.

The third form possesses greater clinical interest than the others, because of its persistency and the deformities which it is liable to produce. It never affects all of the paralyzed muscles with equal inten-

sity. The upper limbs are more often attacked than the legs; the latter being affected in conjunction with the arm, forearm or hand, if at all. The trunk muscles escape. Those of the face and neck are affected infrequently. The upper limb is usually flexed at the elbow. The hand is pronated and flexed, with the fingers drawn toward the palm, in a large proportion of subjects, although the opposite condition may be encountered in exceptional cases. The arm is generally drawn close to the side or upon the anterior aspect of the chest. The knee is usually flexed when the lower limb is affected. The foot may be either flexed or extended.

The intensity of post-paralytic contracture varies. The joints tend to become immovably fixed, after it has existed for years without treatment. After prolonged quietude, as in the morning on awakening from slumber, the contractured muscles often become relaxed; but they soon return to their state of rigidity when movements are attempted.

Vaso-motor and Trophic Disturbances.—Apoplectic clots, in common with other forms of cerebral disease, may be productive of more or less disturbance of the vaso-motor nerves, which result in modifications in the temperature, color, and nutrition of the paralyzed limbs and the trunk.

Not infrequently the paralyzed parts are *redder* than the healthy side, and exhibit an *elevation of temperature.* The skin may become œdematous, and the limb may appear swollen from the infiltration of the subcutaneous tissues. Sweating of the paralyzed limbs is observed in some instances; while in others the reverse condition may ensue, causing the limb to be dry and scaly.

Bed-sores are developed in some subjects soon after the apoplectic stroke. The nates, knee, and heel are the most common seat of these sores. They are to be regarded as due to some impairment of the so-called "trophic nerve-fibres."

The *nails* sometimes undergo post-paralytic changes. They tend to become of a yellowish color, and are often disfigured with ridges. They are more brittle than in health.

Occasionally the *hair* upon the paralyzed parts grows long, thick, and dark.

The *joints* may become inflamed after paralysis. The larger joints of the paralyzed side sometimes begin to exhibit the symptoms of acute synovitis after the lapse of a few weeks in cases of cerebral hemorrhage. The smaller joints are rarely attacked. Disorganization of the shoulder-joint has been recorded by Nothnagel, Hitsig, and others. It may result in dislocation of the humerus.

Finally, cerebral paralysis is less frequently followed by *atrophy* of the paralyzed parts than if due to spinal or peripheral causes. This is

due partly to the fact that a hypertrophy of the skin is particularly liable to be developed. It may exist to so great a degree as to give the paralyzed limbs an appearance of increased size.

Differential Diagnosis.—In hospital cases, where often the clinical history of the subject cannot be obtained, it is not always easy to make a positive diagnosis of cerebral hemorrhage. In the first place, the coexistence of coma and paralysis may be dependent upon many other causes, such as cerebral embolism, cerebral tumors, cerebral softening, and compression of the brain from depressed bone, pus, and exudations of various kinds, as well as upon an apoplectic clot. Again, coma, if unaccompanied with paralysis of motion, may possibly be an indication of the poisonous effects of alcohol, opium, uræmia, and many other substances, as well as some functional nervous derangements. In the third place, patients of this kind frequently have wounds upon the head, as a result of falling. This may sometimes mislead the physician in attributing the coma to a cerebral cause.

From *cerebral embolism* the diagnosis of apoplexy is to be made by the tendency of the coma to deepen in bad cases rather than to improve within twenty-four hours; by the profoundness of the paralysis; by the irregularity of the pupils; the slow pulse; the slow, stertorous, and puffing respiration, and other evidences of cerebral compression; and usually by the absence of aphasia. The history of the patient will also assist in the diagnosis, because embolism has no prodromal symptoms. Furthermore, embolism occurs at any age; it is frequently dependent upon some valvular lesion of the heart; and it occurs when no evidences of arterial degeneration are present.

From *cerebral tumors* apoplexy is to be distinguished by the fact that the headache which may have preceded an attack of cerebral hemorrhage is of a less severe type and not as well defined as in tumor; by the absence of the "choked disk" on an ophthalmoscopic examination; by the fact that some of the cranial nerves are liable to be separately affected, in case of tumor, before the body is paralyzed; by the absence of spasmodic conditions and neuralgias, so often encountered during the development of tumor; by the fact that syphilis is a common cause of cerebral growths; finally, should bilateral or alternating paralysis develop, the diagnosis of tumor is strongly suggested.

From *uræmic coma*, apoplexy is distinguished by the existing paralysis (either of motion or sensation), the absence of albumen and casts in the urine, the irregularity of the pupils, the flushed face, and the absence of œdema or general anasarca. It should be remembered that convulsions and profound coma may exist in both of these conditions, and the prodromal symptoms may not be markedly dissimilar.

In *alcoholic coma* the pupils will be regular, the limbs will not be

paralyzed, the coma will not be as profound as in apoplexy, the breath will smell of liquor, and alcohol may be detected in the urine.

The *coma of opium and chloral* is not attended with paralysis, and the pupils are markedly contracted. The breathing and pulse resemble those of cerebral compression in some respects.

Compression of the brain from *inflammatory* or *serous exudation* could hardly be mistaken for apoplexy. The history of the case would not point to cerebral hemorrhage, nor would the attack be instantaneous.

The differential diagnosis between apoplectic clots in the different parts of the brain have already been considered at some length. Further hints will be given in the closing pages of the section upon the diseases of the brain, which deal with the localization of cerebral lesions.

Prognosis.—Whether it is possible for a patient to recover perfectly from an attack of cerebral hemorrhage is a subject of great importance to every patient and his friends. The question, if asked, can be intelligently answered as follows: (1) the changes which have occurred in the brain are of necessity permanent to some extent; (2) the clot will do more permanent injury in some parts of the brain than in others; (3) the scar, which tends to form in the most favorable cases, is liable to cause, by its presence alone, more or less disturbance of the cerebral functions; (4) in most cases, a tendency to a recurrence of the hemorrhage exists; (5) sometimes many years elapse before a recurrence takes place; (6) finally, the term "recovery" must always be taken in its restricted sense when applied to apoplectic subjects.

The prognosis is the most *grave* when the coma is prolonged beyond the usual limits, when the temperature rises to an extreme point after the attack; when the vagus nerve shows the effect of impairment; when the symptoms of pulmonary œdema are developed; when the sphincters are paralyzed; when prolonged or frequent convulsions accompany or follow the attack, and when the pupils are widely dilated.

In aged subjects, the prognosis is more grave than in those of middle life.

Treatment.—At the onset, in cases of moderate severity, the head should be kept elevated and cold compresses may be applied to arrest the escape of blood. The stomach may be evacuated by running the forefinger into the pharynx of the patient, or by a stomach-pump if the attack should follow a hearty meal. The room should be kept at a comfortably cool temperature (about 60°). No food should be given to the patient for twelve hours after the attack. Cool acidulated drinks will do no harm and are often grateful. If the bowels be constipated, they should be moved by a purgative enema or some mild saline cathartic. Whenever the urine is not passed, catheterism should be resorted to. It

is best, as a rule, to do as little as possible outside of these simple measures until all signs of cerebral irritation have disappeared.

In severe cases it is the custom of some practitioners to administer croton oil at once and to bleed the patient. I would caution the reader in reference to both practices, although they are sustained by some authors. Bleeding is never performed by me, and catharties of an active character should never be given, in my opinion, unless the bowels have been obstinately constipated for several days in succession. Ice-bags to the head and nape of the neck tend to arrest the hemorrhage, and are advisable at the time of the attack. No attempts at medication, in order to promote absorption, should be made until all signs of cerebral irritation have subsided. To dose these patients with enormous amounts of the iodide of potassium early is to my mind nonsensical, and decidedly opposed to all known pathological facts. The aim of the treatment should be to preserve physical and mental quietude by every known means, and to avoid everything that will tend to disturb it.

If any of the symptoms of inflammation appear, after the symptoms of onset have subsided to a greater or less extent, counter-irritation by the use of blisters or the actual cautery to the back of the neck and the renewal of the ice-bags will be of service. If the pulse becomes rapid, small doses of aconite may be indicated. Opium in small doses will often prove of aid in quieting restlessness and mental excitement.

In about a week, provided the case is to go on toward recovery, the patient will show some desire to move his paralyzed limbs, and a slight improvement in motion may be detected. By the end of the second week the actual treatment of the paralysis should generally be commenced. I question the propriety of ever beginning electrical treatment before ten to fourteen days have elapsed, even in the most favorable cases. I would also make the same remark in reference to massage, and the internal administration of phosphorus and strychnia. I believe that nothing is lost, and much often gained, by delaying active treatment beyond the time when anxious friends are apt to clamor for it.

Nutritious food should be administered to the patient as soon after the attack as the stomach is well able to bear it. I have a decided preference for milk over any preparation of beef. I think that an egg broken into a goblet of milk and made palatable with sugar or nutmeg contains more nutrition in a condensed and acceptable form, which can be easily digested, than any other known combination. Still, Liebig's and Valentine's extracts of beef are reliable and valuable preparations, and answer well as a means of administering nourishment.

Respecting the actual treatment of the paralytic symptoms, massage and electricity are the mechanical agents upon which to rely, and phosphorus and strychnia the drugs that seem to be the most beneficial.

Charcot has lately advocated the use of powerful magnets applied against the skin of the paralyzed parts, and has accomplished some apparently startling results. They are too expensive and uncertain as yet in their results, however, to be recommended for general use, even if their curative properties are to be regarded as well established.

Massage should form an important part of the treatment of motor and sensory paralysis. It not only hastens the recovery of motion after apoplectic attacks, but it helps also to prevent the extreme post-paralytic contractures that frequently follow. It should be employed for ten or fifteen minutes once or twice every day by a person experienced in the art. Massage does not consist of rubbing, *per se*. It is an art in itself, and should be scientifically performed when employed.

The faradaic current will answer well in the treatment of apoplectic paralysis, provided that degenerative changes in the muscles do not develop. The strength of the current should be sufficient to cause slight muscular contractions, but not so intense as to create severe pain or fatigue. If the muscles show secondary degenerative changes the static or galvanic current is advisable. It should be employed until the faradaic current begins to show its normal reactions. A rapid improvement will usually follow the use of electricity, even in very bad cases.

The wire brush, as the active pole, is the best method of applying the faradaic current for the relief of sensory paralysis, in case it exists. The anæsthesia often disappears spontaneously, however; the proximal parts of the limbs usually being the first to exhibit improvement.

The question of the advisability of venesection in apoplexy must, to my mind, be decided in the negative. The abstraction of blood from the general system, after a clot has formed in the brain-substance, or upon its surface, cannot affect the existing lesion, and must necessarily tend to weaken the power of reaction.

Strychnia often aids in effecting a cure. It may be given by the mouth or hypodermically. The dose by the former method is about one-twenty-fourth of a grain three times a day, and by the latter about one-thirtieth of a grain once a day. This drug acts particularly well in old cases of cerebral hemiplegia.

Phosphorus is another drug which may be indicated. It may be administered separately or in combination. Phosphide of zinc is a favorite salt with some authorities. The combination of nux vomica, mentioned in connection with the treatment of cerebral congestion, acts well in many cases.

Post-paralytic contractures are benefited greatly by massage, when it is employed persistently from the date of their first appearance.

Regarding mental treatment, I advise my patients to avoid, for many months after an apoplectic attack, all forms of occupation that demand

prolonged mental effort, or which occasion over-excitement or fatigue. I instruct them particularly to dismiss anxiety, as an important step in the cure. Travel, abstinence from tobacco or alcohol, and judicious exercise in the open air are often important aids in re-establishing the health. An excessive use of the eyes, as in reading, is to be prohibited. The digestive apparatus should be carefully watched, and tonics may be given with benefit in many cases. I prefer quinine to all other tonics after the case has progressed well toward a cure.

HEMORRHAGE OF THE CEREBRAL MENINGES.

In this variety of hemorrhage, blood may be found (1) between the dura and the skull; (2) beneath the dura; and (3) in the sub-arachnoidean space. In the previous section we have discussed cerebral hemorrhage in a general way (so far as its symptomatology is concerned); but some facts of value may be given regarding this form in contradistinction to apoplexy.

Morbid Anatomy.—Clots *between the dura and the skull* are almost invariably due to injury. Hammond quotes five cases collected by Gintrac which were apparently of idiopathic origin, but they are to be regarded as exceptional.

The *sub-dural variety* is closely allied to hæmatoma of the meninges (pachymeningitis interna). It differs from the internal form of pachymeningitis, however, in that the hemorrhage precedes the formation of the investing membrane (which is usually detected in both); and also in that the membrane does not present a well-defined laminated appearance and a net-work of newly-formed vessels between the layers. Furthermore, the hemorrhage is less distinctly circumscribed than in the cases of hæmatoma observed. Finally, this variety of hemorrhage may be disseminated over a very extensive area of the brain's surface.

In the *sub-arachnoidal variety* the clot is generally imperfectly organized, because of the admixture of cerebro-spinal fluid. It is frequently found at the base of the skull. The arteries are generally atheromatous. An investing membrane to the clot is not present. This form of hemorrhage is often associated with aneurismal dilatations of the large arteries forming the "circle of Willis."

Etiology.—Infancy and old age appear to be more frequently affected than middle life. It is more common during the first and second years of life, and after fifty than between those ages. The veins as well as the arteries appear to be liable to rupture, especially in young children, after injury received upon the head. Later in life, alcoholism, insolation, excesses in eating and venery, severe muscular exertion, constipation, amenorrhœa, and atheroma tend also to excite it.

Symptoms.—Much that has already been said when apoplexy was

discussed, respecting cortical lesions, bears directly upon this field. Some clinical deductions may be drawn, however, between lesions of the extra-dural and sub-arachnoidal varieties. The dura is so closely attached to the base of the skull that the former variety may be anatomically excluded from that region. Hence the effects of pressure of extra-dural hemorrhages must, of necessity, be exerted chiefly upon the cerebral hemispheres. On the other hand, sub-arachnoidal clots are frequent at the base of the skull, and may involve the crura cerebri, the pons, the medulla, the cerebellar peduncles, and the cranial nerve-trunks as well. We would therefore be more liable to encounter disturbances of the special senses, or the motility of the eyeball, the evidences of facial palsy, vomiting, choked disk, rotary movements, and extensive body-paralysis in the subarachnoidal variety than in extra-dural hemorrhage. Coma, vertigo, headache, and convulsions may occur in both of these varieties. If the medulla is involved, reflex automatic movements, such as the heart's action, the respiratory rhythm, the act of swallowing, etc., are liable to be more or less disturbed if death does not immediately occur.

Small meningeal clots upon the convexity of the hemispheres are indicated often by some special type of monoplegia, amnesic or ataxic aphasia, or an impairment of some special sense. Extensive surface hemorrhage of the convexity would result in a train of symptoms closely allied to those of intra-cerebral clots. These have already been described.

Differential Diagnosis.—The reader is referred to previous pages and to two tables which follow for information respecting the diagnosis of apoplexy and surface hemorrhage.

Prognosis.—Extensive surface hemorrhage generally produces death within a longer or shorter period, varying from a few hours to a few weeks. I have known a child to live many days with a clot that covered nearly an entire hemisphere. On the other hand, I lately saw in consultation a gentleman of sixty years of age who died of surface hemorrhage in a few hours after coma set in. In some instances, recovery takes place by the clot becoming encysted and undergoing absorption.

Clots at the base of the brain are liable to cause instantaneous death by the pressure exerted upon vital centres. The development of vomiting, or of the so-called "Cheyne-Stokes respiration" is a symptom of evil import, whenever encountered in connection with a cerebral lesion.

Treatment.—Nothing can be said here in addition to the suggestions offered in the preceding section.

In closing the discussion of cerebral hemorrhage, I take the liberty of quoting (with slight modifications) from the third edition of my work on "Surgical Diagnosis" the following differential tables, which relate more or less directly to this condition :—

PARALYSIS FROM CORTICAL CEREBRAL LESIONS.	PARALYSIS FROM NON-CORTICAL CEREBRAL LESIONS.
Consciousness.	
Is seldom completely lost at the onset of paralysis, unless the lesion be extensive or due to traumatism. If ushered in with an epileptic attack, consciousness is of course lost.	A sudden loss of consciousness usually accompanies the development of the lesion or its manifestation in the form of paralysis. Convulsions are not usually present during the "paralytic attack."
Pain.	
Local pain within the head is often complained of at the time of the attack.	The patient is usually unconscious at the time of the attack and for some time after; and (even after the attack) pain in the head is a less constant symptom.
Percussion.	
Percussion over the seat of the lesion often elicits pain.	Negative in its results.
Paralysis.	
Monoplegia (in any of its forms) is typical of this condition, whenever it exists. *Special groups* of muscles are paralyzed, and some more than others. The paralysis is often transitory, if the lesion be slight or superficial. The group of muscles, which is the last to show improvement, may be a valuable guide in localizing the seat of injury. Sensibility is usually unimpaired.	*Hemiplegia* or *hemianæsthesia*, more or less profound, follow the development of the lesion, as a rule. Both may coexist in some cases. It is slow in recovery. The improvement is comparatively uniform, so far as special groups of muscles are concerned. More or less anæsthesia usually coexists with the motor paralysis.
Muscular Rigidity.	
The paralyzed muscles often exhibit rigidity at an *early* date.	Early rigidity of the paralyzed muscles is rare in central cerebral disease.
Choreiform Movements.	
Infrequent as a sequel to the paralysis.	Frequently follow the development of the hemiplegia or hemianæsthesia.
Electro-Contractility.	
The paralyzed muscles exhibit normal electro-contractility.	May be impaired or modified, some time after the onset of paralysis.

Symptoms in Common.

Both are associated with motor paralysis.
" may be associated with post-paralytic rigidity of muscles.
" " " " sudden advent.
" " " " traumatism.
" " " " convulsions.

IRRITATIVE LESIONS OF THE CEREBRAL CORTEX. (Jacksonian Epilepsy.)	DESTRUCTIVE LESIONS OF THE CEREBRAL CORTEX.
History.	
Syphilis is by far the most frequent cause of this condition.	Syphilis is only one of many causes of this condition, and by no means the most common.
Convulsions.	
The patient is seized with convulsive attacks of the epileptic type, which are followed by *transient* paralysis. The part which *first shows rigidity* during the convulsion points toward the motor centre for that part as the seat of greatest irritation. It may thus assist in localizing the seat of the lesion.	Convulsions are usually absent.
Paralysis.	
The paralysis is somewhat of the "*monoplegic*" type, but is usually transitory. It is not so well defined as in the case of destructive lesions. It exists, as a rule, on the side opposite to the lesion.	A well-marked "monoplegia" is developed, which is more or less permanent according to the character of the lesion. It generally affects the side opposite to the lesion. The groups of muscles affected with paralysis will aid in deciding as to the seat and extent of the lesion.
Prognosis.	
Good—on account of its frequent syphilitic origin.	Depends entirely upon the character of the lesion, its seat, and extent.

CEREBRAL HEMIPLEGIA.	SPINAL HEMIPLEGIA.
Form of Attack.	
Onset usually sudden. Consciousness is often lost when the lesion is centrally situated in the hemispheres.	Onset may be gradual. Consciousness is not lost.
History.	
That of some *cerebral disease*, such as apoplexy, embolism, softening, tumor, etc.	That of some *spinal lesion* situated in the cervical region, and involving only one *lateral half* of the spinal cord.
Pupils.	
Are liable to be irregular.	Are unaffected, unless the cilio-spinal centre within the cervical region of the cord be involved. If so, the "Robertson pupil" may exist.
Ophthalmoscopic Examination.	
May reveal the "choked disk."	Negative.

Cranial Nerves.	
The cranial nerves are frequently involved, causing paralytic symptoms—commonly in the nostril, eye, or face. The spinal senses are often modified.	The cranial nerves are not involved, unless a sclerosis of the cord extends upward late in the disease.
Crossed paralysis (in any of its forms) may be present.	Crossed paralysis is never present.
Reflex Phenomena.	
Are usually normal.	Some of the various reflexes are liable to be impaired or lost.
Spasmodic Phenomena.	
The paralyzed muscles are not rendered particularly susceptible to spasm.	Spasms of the limbs are very frequent.
Electro-Muscular Phenomena.	
Usually normal.	Modified according to the parts of the cord which are affected by the lesion.
Sensory Phenomena.	
Anæsthesia or Analgesia, whenever they exist, are on the *same side as the motor paralysis.*	Anæsthesia or Analgesia, when present are on the *side opposite to the motor paralysis.* Sensations of burning, pricking, formication, coldness, and heaviness often exist at the onset. Hyperæsthesia follows. Subsequently anæsthesia may be developed.
Respiration.	
Respiration is seldom affected.	Difficulty in breathing is often experienced when the spinal lesion is above the origin of the phrenic nerve.
Sphincters.	
The sphincters are not involved, as a rule.	The vesical and anal sphincters are often affected with inertia or paralysis.
Sexual Functions.	
The sexual power is commonly retained.	The sexual power is occasionally abolished.

Symptoms in Common.

Both are associated with hemiplegia.
Both may be associated with abnormal sensory phenomena.

PACHYMENINGITIS; OR, INFLAMMATION OF THE DURA MATER.

This condition is usually circumscribed, and rarely spreads over the whole convexity of the brain. It is of two forms, the suppurative and the non-suppurative. Because it is frequently associated with extravasation of blood, it is described by some authors under the name of "hæma-

toma of the dura mater." Other authors classify it, as regards the surface of the dura involved, into the external and the internal,—the latter being sanguineous, if of the chronic type.

The cause of this variety of inflammation often governs its seat, extent, and variety; hence its symptoms will be modified somewhat by its method of origin.

Meningeal hemorrhage is classed by some authors under the head of pachymeningitis. To my mind this is illogical and opposed to pathological data. Hemorrhage may be one of its causes, as well as one of its results, but not one of its varieties, because pachymeningitis is accompanied by changes in the dura.

Morbid Anatomy.—Suppuration more commonly accompanies the hemorrhagic variety and that produced by caries of the bones. It may occur also after traumatism, especially if thrombosis follows. A general arachnitis is then liable to be induced as a complication of the circumscribed inflammation of the dura. The base of the brain is usually exempt from this form of meningitis, except as a sequel of traumatism, tumors, or diseases of the vertebræ. Hæmatoma is very common at the vertex, and sometimes affects both hemispheres of the brain simultaneously by crossing the mesial line.

In the *non-suppurative variety* the dura becomes composed of superimposed layers (as high as twenty in some instances). These are rich in vessels (which tend to rupture where the arterial tension is excessive from any cause). The dura subsequently becomes united to the arachnoid. The layers of a hæmatoma occasionally ossify. When the newly-formed vessels sometimes rupture, they tend to create circumscribed sanguineous cysts. When syphilitic caries is the exciting cause, the dura may become gangrenous.

The *external variety* is always a secondary inflammation. The dura is found to be injected, softened, and ecchymotic in its initial stage. Later on, new connective-tissue formations induce a thickening and opacity of the dura, and adhesions take place between it and the skull, provided suppuration does not occur. Pigmentation of the dura and the formation of osteophytes in that membrane are sometimes encountered.

Secondary thromboses of the cerebral sinuses may follow a pachymeningitis. These may suppurate and induce metastatic abscesses of the viscera (as explained on page 230).

Etiology.—The causes which tend to produce pachymeningitis are as follow: (1) injuries to the cranial vault; (2) syphilitic disease of the bones of the cranium, most commonly of the temporal; (3) hemorrhage between the dura mater and the bone; (4) diseases of the vertebræ and their ligaments; (5) thrombosis of the cerebral sinuses, chiefly the transverse and petrosal; (6) chronic inflammation of the middle ear; (7)

suppurative inflammation of the orbit; (8) chronic alcoholismus; (9) pyæmia; (10) Bright's disease; (11) rheumatism; and (12) acute infectious diseases.

Symptoms.—Lesions of this character may excite paralysis of parts supplied by cranial nerves which lie adjacent to them; and when the pressure becomes extreme, paralysis of the limbs and even coma may follow.

Localized pain is usually present over the seat of the disease; and percussion of the skull over the lesion tends, as a rule, to increase the pain.

The defective blood-supply (in those convolutions of the brain which lie adjacent to the lesion) that ensues from pressure upon them may lead to softening. When suppuration occurs, the symptoms are greatly aggravated.

In many cases, the symptoms of the EXTERNAL VARIETY are very obscure, especially at the onset. When somnolence, vertigo, headache of a localized type, delirium, photophobia, convulsions, or coma follow any of the causes enumerated among the etiological factors, it may be well to suspect the existence of one of the two varieties.

Chronic inflammation of the *middle ear* is often regarded by parents as of trivial importance, and physicians not infrequently advise them to that effect; yet it is one of the most prolific causes of this serious condition, and is liable to produce death if not properly cared for.

If *thrombosis* of some of the cerebral sinuses has existed and created this complication, the prodromal symptoms will be those enumerated in connection with thrombosis.

Syphilis is a prolific cause of pachymeningitis, because it often induces a carious state of the cranial bones and an extension of the inflammation to the adjacent dura, which tends toward suppuration. Cerebral symptoms, when occurring in connection with syphilis, should always be the subject of careful inquiry.

The INTERNAL VARIETY of this disease may occur idiopathically,—chiefly in connection with chronic alcoholism, the hemorrhagic diathesis, blood-poisoning from urea, scurvy, pernicious anæmia, leucocythæmia, general paresis, hydrocephalus, cerebral atrophy, cerebral tumors, and pyæmia. It may also develop secondarily to the external variety.

The symptoms of the internal variety are liable to be more distinctly defined than are those of the external, because the development of a hæmatoma of the dura creates localized pressure upon the brain or some of the cranial nerves, the severity of which depends upon its size and seat. Hence we are liable to encounter evidences of impairment of intellect, temporary loss of consciousness, aphasic symptoms, early contraction and subsequent irregularity of the pupils, exacerbations and

remissions of temperature, slowing of the pulse, choked disk, and a gradual paralysis of motion or sensation, which may be preceded by convulsive attacks. Headache is a marked symptom as a rule, especially in the syphilitic variety. The hemorrhages that occur in this variety are constantly recurring because the newly-formed vessels are in a loose connective-tissue formation, and are often atheromatous. This fact possibly helps us to explain why these patients have short periods of loss of consciousness, transient paralyses, convulsive attacks at infrequent intervals, and periodical headache. Sudden death may follow an extensive hemorrhagic extravasation. Bilateral paralysis may result from hæmatoma of the dura, when it is situated at the vertex and crosses the sagittal suture.

The SYPHILITIC VARIETY is classed as distinct from the others by some authors, because the deposit upon or beneath the dura is usually of a gummatous nature. In this variety, localized headache is very persistent and usually grows worse as night approaches. Special cranial nerves are not infrequently involved before the cerebral functions are disturbed in any way. I have made the diagnosis of this condition in several cases by the development of hemianopsia; also by the existence of motor oculi paralysis, and facial paralysis alone. I once saw a patient who lay in profound coma with dilated pupils, stertorous breathing, and relaxed sphincters from this cause for ten days and eventually recovered. Cases of this type of pachymeningitis may exhibit symptoms of insanity early. On the other hand, they may become dull, stupid, and apathetic, before other symptoms develop which may assist in definitely localizing the seat of the lesion. In exceptional cases, I have seen hemiplegia develop from this cause without any prodromal symptoms.

Differential Diagnosis.—The obscurity of the symptoms in all forms of pachymeningitis renders its differential diagnosis rather a matter of conjecture than of scientific accuracy. The following hints may aid, however, in the discrimination:—

1. In case necrosis of the cranial bones, otitis media, fracture of the cranium or traumatism of the skull has existed and been followed by evidences of gradual cerebral compression, we may strongly suspect the existence of dural inflammation.

2. When a hæmatoma is developing, the symptoms may resemble those of either acute or chronic meningitis or cerebral softening, and a positive diagnosis may be impossible. Bilateral paralysis is strongly indicative of hæmatoma. The following symptoms are, in my experience, also strongly diagnostic: Contraction of the pupils; localized headache; transient and recurring attacks of unconsciousness or paralysis; a slow pulse; strabismus; ptosis; and facial palsy.

3. In connection with syphilis, the development of cerebral symptoms

of any kind should excite a suspicion of the existence of pachymeningitis of the gummatous variety. The cranial nerves, especially the olfactory, optic, motor oculi, and facial, are apt to be involved and to give clinical evidence of more or less impairment of function.

The following table is quoted from the third edition of the author's work on "Surgical Diagnosis:"—

EXTERNAL PACHYMENINGITIS.	INTERNAL PACHYMENINGITIS. (Hæmatoma of the Dura Mater.)
Causes.	
Traumatism of the calvaria. Diseases of the cranial bones. Caries and necrosis of the cervical vertebræ. Suppurative diseases of the vertebral ligaments. It rarely follows syphilitic or rheumatic conditions of the cranium or erysipelas of the scalp.	Chronic alcoholism and syphilis. Acute febrile disorders (fevers, rheumatism and puerperal diseases). Chronic diseases of the heart, tuberculosis, and the paralysis of the insane may be associated with it. Old age. Males more frequently affected than females.
Headache.	
Intense and circumscribed headache usually exists.	Periodical headache is commonly produced; gradually reaching extreme intensity whenever the acute form exists.
Convulsions.	
Slight convulsions are common at the onset.	Convulsions are rare.
Brain Symptoms.	
Vertigo, nausea, and vomiting are frequently met with at the onset of the disease.	Weakness of memory, apathy, somnolence, and delirium are the more common symptoms.
Pupils.	
The pupils are apt to become unequal, if the pressure upon the brain is severe.	The pupils are not necessarily affected, because the pressure of the sanguineous cyst is more limited than that of a pus exudation. They may be contracted.
Pulse.	
The pulse is at first accelerated, but becomes slow and irregular when cerebral compression is produced.	The pulse fails to exhibit the effects of general cerebral compression, except in severe and fatal cases.
Late Symptoms.	
Coma and paralysis follow if cerebral compression or abscess is produced.	Feebleness of the limbs, unsteady gait, and changes in the nutrition indicate the latent progress of inflammation of the brain.

Symptoms in Common.

Both may be associated with headache.
" " " " convulsions.
" " " " coma.
" " " " paralysis.
" " " " syphilitic history.

Prognosis.—Patients may recover from pachymeningitis, in spite of the fact that the prognosis is usually grave. In alcoholic patients, the disease is generally fatal. If suppuration is developed, the dangers to life are materially enhanced; hence the prognosis is grave if it be induced by caries, necrosis, otitis media, cancer, traumatism, etc.

Hæmatoma of the dura is fatal in a very large percentage of those attacked. These patients may die from hemorrhage, cerebral thrombosis, cerebritis, dementia, pyæmia, and other complications, before the tumor produces death by direct pressure upon the brain. Life may terminate between the limits of two weeks and two years, according to the circumstances incident to the case.

In the syphilitic variety of pachymeningitis I have witnessed some very remarkable recoveries. When the gummatous deposit is small, it may be absorbed by active specific treatment. If it be large, the brain may be softened and permanently impaired before the cause can be removed. I have often observed a tendency toward a return of the symptoms in many of these subjects that have been greatly benefited at first by treatment. If chronic alcoholism is a factor in the causation, as well as syphilis, the prognosis is very grave; but recovery may occur even under these circumstances. I lately saw a remarkable instance of that character in connection with my late colleague Prof. A. L. Loomis.

Treatment.—When pachymeningitis is of the suppurative type, surgical interference may overcome the difficulty to a large extent by evacuating the pus. It is not always possible, however, to localize the situation of the pus or even to determine its presence with certainty. The cause may aid in this discrimination, in some cases. Traumatic causes, otitis media, and caries or necrosis are especially prone to purulent deposits between the dura and the bone. A careful study of the symptoms (from the standpoint of cerebral physiology) may, in exceptional cases, indicate the seat of the abscess. I have given the surgical guides for the employment of the trephine in such cases in my work upon nervous anatomy,* and also in the first section of this volume.

During the development of a hæmatoma of the dura life may be prolonged by a judicious use of ergot, stimulants, and a nutritious diet, in conjunction with enforced physical and mental quietude and counter-irritation. I do not believe that any line of treatment will effect a cure.

In the gummatous variety, I have found the best results to follow mercurial fumigation and the internal administration of large doses of the iodide of potash (grs. xxx. to lx. after each meal, and even larger doses if necessary). I prefer the black oxide of mercury (one drachm to a bath) to any other preparation when fumigation seems advisable,

*"The Applied Anatomy of the Nervous System." D. Appleton and Co., N.Y., 2d. edition 1888.

although calomel baths act well in some cases, if watched carefully to avoid salivation. I have frequently employed inunctions of one drachm of the 20 per cent. oleate of mercury night and morning with benefit, when the baths were not to be obtained; but the effects are less certain and the treatment more disagreeable to the patient than by the bath. In one case I carried the iodide of potash up to a daily administration of one and a half ounces for several consecutive days before I got satisfactory results. The subcutaneous sublimate injection, recommended by Lewin,* has yielded excellent results, in some instances, under my observation. I frequently employ it in connection with mercurial fumigation. In those cases where the iodide of potash is badly tolerated, I have employed the iodide of calcium with excellent results. This salt is unstable, and should be carefully bottled by the manufacturers so as to prevent its decomposition. It may be given in the same doses as the other salt. All of the iodides should be administered in combination with Vichy water, and very largely diluted.

The employment of cod-liver oil and tonics is advisable in these cases, as they are anæmic and badly nourished as a rule. All stimulants should be withheld, unless serious damage to the patient is indicated by so doing. In such a case they should be decreased in amount steadily as fast as practicable.

I have personally discarded for some years past the old routine management of cerebral syphilis by the internal administration of the so-called "mixed treatment." It does not accomplish, in my experience, what the fumigation, inunction, or subcutaneous injection methods will; nor is it, to my mind, free from other serious objections. My convictions upon this point are strong, and are based upon quite a large experience in the management of these cases. It is not necessary for me to defend my views here; but it cannot be denied that one essential factor in success in these cases is unimpaired digestion. Even if the improvement was as rapid under the old plan as the new (which it certainly is not) the digestive organs are more frequently deranged.

ARACHNITIS.

Thickening and opacity of the arachnoid has been observed in connection with atheroma, cancer of the abdominal viscera, granular kidney, senile dementia, delirium tremens, tuberculosis, heart-lesions, cerebro-spinal meningitis, hemorrhage into the pia mater, and as a sequel to disease of the bone and the dura mater.

It may be associated with an effusion of lymph or pus, and be either local or general.

The exciting cause will be of the greatest aid in determining its char-

* "Syphilis," by George Lewin. Blakiston, Son & Co., 1882.

acter during life. It is often impossible to discriminate during life between lesions involving the dura mater from those of the arachnoid; and it is still more difficult to separate its morbid conditions from those of the adjacent pia mater which underlies it, and which usually participates to a greater or less extent in its changes.

Atheromatous degeneration of the cerebral vessels seems to be commonly associated with those forms which have their apparent origin either in cancer or the abuse of alcohol. As is the case with all inflammatory conditions of the coverings of the brain, the symptoms are produced either by the hyperæmia in the early stages, or by the pressure of the exudation upon the brain, or from cerebral thrombosis in the later stages of the disease.

INFLAMMATION OF THE PIA MATER—DIFFUSED MENINGITIS OF THE CONVEXITY OF THE HEMISPHERES.

We have already discussed diseased conditions of the dura under the heads of meningeal hemorrhage and pachymeningitis, and it now behooves us to consider the pia mater in its pathological aspects.

Meningitis may be subdivided into four forms: the acute, subacute, chronic, and tubercular. The acute, subacute, and chronic varieties of inflammation of the pia are generally diffused over the convexity of the cerebral hemispheres. The tubercular variety (hydrocephalus) is commonly confined to the base of the brain to a greater or less extent.

The varieties of meningitis will be considered separately, because their pathology and symptomatology differ widely.

ACUTE MENINGITIS.

Synonyms.—This condition is described by authors of note under the following heads: *1. Simple meningitis of the convexity; 2. General arachnitis; 3. Cerebral fever; 4. Non-specific inflammation of the meninges; 5. Acute cerebral leptomeningitis.*

Morbid Anatomy.—On removal of the calvaria, the surface of the hemispheres appears of a *greenish hue.* This is due to the presence of an exudation into the meshes of the pia and the peri-vascular lymph spaces.

The vascularity of the meninges is excessive. The vessels appear to the eye as prominent red lines running over a green background. The base of the brain is usually free from abnormal appearances.

The pia is thickened, reddened, and rendered opaque in spots. In some cases the arachnoid and the dura may be adherent to each other. Along the main fissures and the more important sulci, a creamy exudation (consisting of an admixture of *serum*, *fibrin*, and *pus*, in varying proportions) is found. It constitutes in many cases an approach to a

false membrane, which can be readily detached from the convolutions underneath it.

When this false membrane is raised, the surface of the brain is seen to be dotted over with small red spots. These indicate capillary extravasations from the small branches of the vessels of the pia, which everywhere supply the cortex.

Occasionally the cerebellar meninges are implicated. Whenever this is the case it is common to find the cranial nerves more or less encapsulated in a sheath formed by the exudation.

If the exudation upon the cerebral hemispheres be large in amount, the cerebral convolutions will appear more or less flattened, the sulci will be deeper than normal, and the ventricles will contain little if any fluid.

Cross-sections made through the white substance of the cerebrum will often reveal minute spots of extravasation,—the so-called "puncta vasculosa."

Etiology.—Among the *predisposing causes* of this disease may be mentioned: (1) age, since youth or young adult life is most commonly attacked; (2) sex, because males are more often attacked than females; (3) anxiety, prolonged mental labor, and grief; and (4) excessive indulgence in alcohol.

The *exciting causes* include (1) injuries to the bones of the head; (2) caries or necrosis of the cranial bones; (3) otitis media; (4) insolation; (5) extension of inflammation from the orbit and the vertebral ligaments; (6) metastasis of skin eruptions; (7) blood-poisons, chiefly those of the fevers, pyæmia, rheumatism, uræmia, diabetes, diphtheria, and rheumatism.

Meningitis may be of idiopathic origin in rare instances, according to some authorities. I have always doubted the accuracy of this statement, if the term be used in its true sense.

Symptoms.—In the early stages of acute meningitis the symptoms may be regarded as those of cerebral irritation combined with marked febrile excitement.

A persistent and intense *headache* exists from the onset, accompanied by contracted pupils, vertigo, photophobia, and profectile vomiting in many cases. The attack may begin with a chill. The temperature may rise to 105° or 106° in a severe case; but it seldom exceeds 102° or 103°. The highest temperature usually occurs from the fifth to the seventh day.

Cutaneous hyperæsthesia and muscular twitchings are developed early.

The gait is tottering and indicates an impairment of coördination in the early part of the disease. A continuous high temperature is an indication of a fatal termination.

The frequency of the pulse (which is small, firm, and tense) varies. It ranges proportionately with the remissions and exacerbations of the temperature.

Finally, constipation and retraction of the abdomen are usually present during the stage of headache. With children, it is not uncommon to have the first stage ushered in with convulsions and marked strabismus.

The face is pale, as a rule, during the initial stage of meningitis, and the conjunctiva is injected. Occasionally, the face may be flushed.

As headache is the prominent symptom of this stage, its character should be fully described. It is intense from the onset, and is associated with an excitation of the special senses. It steadily tends to grow worse, instead of better. It may be confined to the frontal, temporal, or occipital regions of the head. It may last for a variable space of time,—from a few hours to as many days.

In most respects the initial stage of acute meningitis resembles that of intense cerebral congestion. Hence what has been said respecting the symptoms of that condition will apply here.

Second Stage.—Delirium usually sets in after the stage of headache has lasted for a longer or shorter period (seldom more than a few days). It is accompanied by an increase of the fever, jactitation, restlessness, and mental excitability, or confusion of intellect. So prominent a feature does delirium now become that this stage is denominated the "*stage of delirium*" by some authors. It sometimes becomes so active that it resembles acute mania. Hallucinations of sight and hearing often accompany the delirium, and the articulation of the patient frequently becomes incoherent. In the aged, the delirium is of the typhoid character, and is manifested by incessant talking, accompanied by irrationality of ideas. The patient may laugh or weep over imaginary occurrences, and is apt to gesticulate wildly. Twitchings of the facial muscles and a peculiar rolling of the eyeballs in the orbits are commonly observed. Occasionally the flexors of the limbs contract powerfully and produce episthotonos. Convulsions in adults are rare, but they sometimes occur. Hemiplegia and paraplegia are infrequent, although I have personally observed the former in one instance. Hammond reports two cases where hemiplegia persisted throughout the entire course of the disease.

When the medulla is implicated or the nerve trunks which arise from it, the acts of respiration and of swallowing may be imperfectly performed before coma develops. The pulse may then become irregular, the abdomen will be retracted, and projectile vomiting may exist.

Occasionally the delirium merges into attacks of insanity, with homicidal or suicidal tendencies. Hence it is well to guard against such accidents by keeping a close watch over the patient.

Finally, an herpetic eruption occurs not infrequently during this stage.

Third Stage.—After several days have elapsed from the onset of the attack, the patient, as a rule, becomes more quiet and gradually sinks into the state of profound coma. The headache of the first stage, and the cutaneous hyperæsthesia, as well as the delirium, the jactitation, and the contractures of the second stage, give place to a stupor that deepens from hour to hour. The pupils begin to exhibit oscillations and gradually become dilated. The pulse becomes very slow, irregular, and intermitting in character. The patient grinds the teeth, picks at the bedclothes, and often develops ptosis and more or less facial paralysis. The urine is passed unconsciously, and the bowels are often moved in the bed. Sometimes retention of urine occurs, and a catheter has to be passed at regular intervals. The temperature remains elevated until death is imminent, when it often falls rapidly. The pulse becomes very rapid as death approaches, and can with extreme difficulty be counted at the wrist. The Cheyne-Stokes respiration develops, the body becomes clammy and bathed in a cold sweat, and life is extinguished as a result of heart-failure, asphyxia, or pulmonary œdema.

In this stage of coma and collapse the face may become alternately pale and suffused, or it may assume a purplish color from asphyxia. The head may be drawn toward one side.

Differential Diagnosis.—This disease may be confounded with delirium tremens, typhus fever, acute uræmia, small-pox, and encephalitis.

From *delirium tremens*, this disease may be told by the absence of the clammy sweat during the stage of delirium, the presence of headache, and the peculiar character of the delirium. The temperature, the pupils, and the pulse are also widely different from those of alcoholism.

From *typhus fever*, meningitis may be diagnosed by the incompressibility of the pulse, the comparatively low range of temperature, the absence of the mahogany or leaden face, the hyperæsthesia of the surface, the projectile vomiting, and the absence of the characteristic eruption of typhus.

From *acute uræmia*, it may be differentiated by a urinal analysis, which will fail to reveal the presence of casts or blood, and by the absence of frequent and severe convulsions. There will be no œdema of the eyelids, and the face is pale rather than turgid. Evidences of cerebral compression will also be present, and the temperature will aid greatly in excluding uræmia.

From *small-pox*, meningitis can be told by the absence of pain in the loins and back, the absence of the characteristic eruption on the third day of the disease, and the presence of projectile vomiting. Until the eruption occurs the diagnosis is difficult.

From *circumscribed encephalitis*, the distinction is made by the intensity of the headache, the activity and peculiar character of the delirium, the severe muscular contractures, the active febrile symptoms, and the rapid development of collapse.

Senile Meningitis.—When meningitis of the acute form occurs in the aged, the symptoms differ markedly from those described as typical of that condition in the infant or young adult. The headache is either absent or not severe. The rise in temperature is not so well marked. The projectile vomiting is usually absent, and gastric derangement is not developed, as a rule.

Senile meningitis so closely resembles cerebral softening that sometimes it is with difficulty differentiated from it. The speech is often incoherent, the memory impaired, and many of the acts of the patient appear irrational. Coma may occur in the initial stage. The delirium, when it exists, is of a low and muttering type. It is common for these patients to develop paresis of all the limbs.

The diagnosis of this type of meningitis from cerebral softening is made chiefly by the rapidity of its progress and the absence of the prodromal symptoms that usually precede softening of the brain.

Prognosis.—In any of the varieties described, the prognosis is always unfavorable. Severe cases usually terminate fatally within ten days; less acute cases may end in recovery. We are justified in anticipating a fatal termination when paralysis of the limbs occurs, when ptosis or strabismus is present, when hiccough exists, when the temperature is high and shows no remissions in the morning, and when the Cheyne-Stokes respiration is developed. The prognosis of any form of meningitis is always more favorable in childhood than in adults.

Treatment.—The first and second stages of this disease are characterized mainly by symptoms of cerebral congestion; hence, general blood-letting by leeches to the neck or temples and by venesection at the elbow are indicated. Leeches may be applied also inside of the nostril (so as to directly deplete the superior longitudinal sinus) or over the mastoid region, to deplete the lateral sinus. I doubt the advisability of ever employing extensive blood-letting in children; but, in adults of a vigorous constitution, as high as fifteen ounces of blood may be abstracted from the vessels.

The second agent to be employed is cold to the head. The hair should be shorn and ice applied to the scalp throughout the first and second stages. The room should be darkened and all disturbing influences should be carefully guarded against. The rubber coil, through which iced water is allowed to flow continuously, is an excellent and perhaps the best way of keeping the head under the influence of cold. It is

best to maintain an elevated posture of the head, so as to assist the venous return.

Active catharsis is a third agent which can be employed to advantage. Croton oil is always reliable and prompt in its action. Calomel is also good, especially if its action is accelerated by the addition of jalap or podophyllin.

The administration of small doses of calomel (one grain every two hours) and also the employment of the iodide and bromide of potash have been suggested. My experience with the iodide of potash has been very satisfactory in some cases. Regarding the use of the bromide of potash, Dr. Hammond reports that very great benefit has been afforded in cases intrusted to his care by the daily administration of from ninety to one hundred and twenty grains of that salt from the onset until the stage of delirium had passed. Three of his cases recovered under this treatment.

I have not had as good results with the continued administration of calomel as those reported by Hammond and others.

In the stage of coma and collapse, blisters may be applied at the nape of the neck or the actual cautery may be employed as a counter-irritant. All medication should be abandoned in this stage, and the patient should be stimulated and nourished. My guide to the employment of stimulants is the effects which they have upon the pulse and temperature. If they tend to bring both nearer to the standard of health, I continue their administration. I believe that many patients die from the lack of stimulants in a crisis of disease, especially in inflammatory conditions of the meninges. Champagne, brandy, and whiskey, are preferable to port and sherry. I am in the habit of giving stimulants often in small doses and in combination with systematic feeding at short intervals. Beef-tea, beef-juice, milk, pure cream, and a raw egg beaten in milk are my favorite methods of giving nourishment in these cases. In the aged, the use of stimulants is indicated early in the course of the disease.

Finally, the condition of the bladder and the bowels should be carefully watched in this stage, especially in the case of extremely old subjects. Retention of urine in comatose patients is liable to be overlooked by the attendants and to cause great distress. A catheter should be introduced into the bladder of patients so afflicted at regular intervals during the day and night.

SUBACUTE CEREBRAL MENINGITIS.

This variety differs from the acute in that it is a secondary condition (being the result of some exhausting disease), and also in that the duration of the attack is longer, the brain-symptoms less marked, and the prognosis proportionately less grave.

Morbid Anatomy.—The pathological changes differ little from that of the acute form, except in that the exudation contains a smaller proportion of pus and fibrin, and is less extensive in amount. The loss of lustre and opacity of the pia is less extensive and adhesions of the pia to the dura or to the cortical brain-substance are less common.

Etiology.—We are liable to encounter this form of meningitis as a complication of chronic Bright's disease, cancerous deposits, typhoid fever, chronic diarrhœa, rheumatism, and other exhausting diseases.

Symptoms.—The development of this complication is usually indicated by delirium. The stage of headache and the initial chill are not present as a rule. If headache exists, it is moderately severe and lasts but a few hours. Jactitation generally precedes the onset of delirium.

The delirium is not as active as in the acute form. The patient develops a desire to constantly get out of bed, but is easily induced to return. Muttering and an unsteadiness of gait develop.

The stage of coma is liable to come on rapidly. The pulse becomes slow, the respirations sighing or puffing in character, and cyanosis rapidly supervenes. Convalescence in patients who pass into the stage of coma is very slow. Death is the more common termination.

Rheumatic meningitis may occur through metastasis, and as a complication of that disease. It may be of the subacute or chronic variety. It is often fatal.

Differential Diagnosis.—Many hints given in connection with the diagnosis of apoplexy from the various forms of coma encountered (p. 278) will apply here, because coma is the prominent symptom of this variety of meningitis. The cause is an important factor in the discrimination in every case of suspected meningitis. Some of the differential diagnoses mentioned when the acute form was considered might be repeated here.

Prognosis.—When subacute meningitis occurs as a complication of chronic Bright's disease and rheumatism, it is generally fatal. The chances of recovery are better when it develops from the blood-poisoning of fevers, or accompanies exhausting diseases.

Treatment.—Blisters at the nape of the neck are often attended with great benefit. If uræmia exists, the elimination of the poison should be hastened by catharsis, hot-air baths, and diuretics. If it be due to exhaustion, stimulants are indicated.

CHRONIC CEREBRAL MENINGITIS.

This variety of meningitis may be confined to the convexity of the cerebrum or to the base of the brain. It is essentially a disease of adult life. It is a common lesion in some forms of insanity. The symptoms will be modified greatly by the seat of the disease. If basilar in type, the cranial nerves are especially liable to become involved.

Morbid Anatomy.—The connective-tissue elements of the pia are chiefly affected in this form of meningitis. We encounter, therefore, an opaque and thickened condition of that membrane, and an abundance of new connective-tissue cells. The coats of the vessels of the pia are often thickened. The branches which nourish the cortex are sometimes enclosed in meshes of new connective-tissue which bind the pia to the brain's surface. It is therefore frequently found to be difficult to remove that membrane without tearing away portions of the brain. The connective-tissue of the brain (the neuroglia) sometimes participates in the inflammatory process. Hence diffuse interstitial encephalitis may be one of the complications or sequelæ of chronic inflammation of the pia.

Chronic meningitis is usually more or less circumscribed in extent. When pachymeningitis has been an exciting cause of this condition, the pia will be adherent to the dura and appear thickened and very opaque.

The cortex may be found to have undergone atrophy, as a result of long-continued pressure or impairment of its vascular supply.

Finally, an effusion which consists of serum, pus, or lymph may be detected in the meshes of the pia. Tubercle has occasionally been observed upon the convexity of the hemispheres in this form of meningitis. Cysts may be developed by an encapsulation of the serous deposit by false membranes. The Pacchionian bodies along the cerebral falx may be increased. The ventricles are liable to contain an excess of fluid over the normal amount.

Etiology.—This form of meningitis is commonly a secondary affection. It is met with as a complication of syphilis, gout, rheumatism, chronic kidney-disease, alcoholism, prolonged exposure of the head to extreme heat, or the rays of the sun, and tuberculosis. It may be developed idiopathically, especially in subjects who are badly nourished, or who have suffered from privation, mental anxiety, grief, or great emotional excitement. I recall one case that was apparently induced by a religious revival. Hammond states that cooks are very prone to develop this disease, on account of the exposure of the head to heat. This disease is seldom met with until adult life. It is more common after the fiftieth year than before it.

This form of meningitis may occasionally follow the acute variety. It may also be induced by injuries received upon the head.

Symptoms.—I have several times encountered the pathological evidences of well-marked chronic meningitis at an autopsy, when its existence had not even been suspected during life. I mention this fact as an illustration of the obscurity of the symptoms in some instances.

In many cases, the symptoms resemble those of general paresis. The mind of the patient is usually affected, so that intimate friends observe a change in the disposition or the intellectual attainments,

although the symptoms of mental impairment might escape a casual observer. Moroseness, peevishness, apathy, somnolence, and delusions are frequently developed.

The muscular power of the patient begins to show impairment later. The act of walking is often rendered difficult, on account of a trembling of the legs. The sphincters begin to act imperfectly, so that the patient is often unable to prevent soiling of the clothing. The articulation of words sometimes becomes affected. The speech is occasionally so blurred and indistinct that only friends can understand what the patient says. Paralysis of motion or sensation may be developed in the limbs, in some cases.

Headache, which is increased by any mental or physical exertion, is one of the most common symptoms. It is persistent, but not particularly severe. In connection with chronic kidney-disease, this symptom forms a peculiarly striking feature. Epileptic convulsions often follow the headache, if the meningitis be due to this form of blood-poisoning.

Vomiting of a persistent type is a symptom that is very frequently present in many cases. It is associated with great physical exhaustion.

The cranial nerves may give evidence of this disease when the base of the brain is implicated. Facial palsy, strabismus, ptosis, irregularity of the pupils, sloughing of the ball of the eye, ischæmia of the papilla of the retina, actual blindness or hemianopsia, choked disk, impairment of the power of deglutition, irregularity in breathing, and sluggish movements of the tongue, may develop and be clinical guides to the progress and seat of the lesion.

Finally, vertigo, buzzing in the ears, specks before the vision, attacks of numbness and of hyperæsthesia in the limbs, and general convulsions may coexist with the symptoms already mentioned.

Profound coma usually occurs before a fatal termination. It is the result either of pressure upon the cerebral hemispheres or a mechanical anæmia of the brain.

Differential Diagnosis.—This is extremely difficult, and often impossible. We have in the majority of cases to deal with a combination of the symptoms of meningeal inflammation and those of cerebral inflammation, combined with the mechanical effects of pressure upon the nerve centres. It is not always possible to decide positively between such a medley of symptoms and those which accompany cerebral softening and cerebral tumors.

The history of the patient may be of the greatest importance in the discrimination.

From uncomplicated *cerebral softening*, chronic meningitis is to be told by the persistency and severity of the headache; the absence of the history of a previous apoplectic attack; the vagueness, so to speak, of

the mental symptoms; the infrequency of muscular contractions; and the presence of undue mental excitement. As chronic meningitis and cerebral softening are often associated, the diagnosis may be impossible.

From *cerebral tumor*, chronic meningitis is to be distinguished by the character of the headache, which is more intense and circumscribed where a tumor exists, by the frequent paralysis of the cranial nerves in case of tumor, and by the slow development of mental symptoms when a tumor is present. Moreover, the ophthalmoscope will usually reveal the characteristic appearances of choked disk (fig. 87) when a tumor is developing within the cavity of the skull.

It must be borne in mind, however, by the reader that tumors of the brain or its meninges are frequently associated with a chronic meningitis of a circumscribed character which is excited by the neoplasm. In such a case the symptoms of both conditions would be so intermingled as to render a differential diagnosis impossible.

Prognosis.—When syphilis is the cause of chronic cerebral meningitis, active treatment in the early stages may effect a rapid cure. Hence the prognosis is much more favorable in patients who give a history of previous syphilitic infection than in those who do not.

The majority of cases of chronic non-specific meningitis do not usually take so favorable a course under any form of treatment, although recovery has been known to occur. Many of this type of patients develop insanity or some complicating diseases of the viscera, chiefly pneumonia and pulmonary œdema. Others may die from inanition or the mechanical effects of direct pressure upon the brain by the exudation.

The tendency of this disease is toward progression, as a rule. The prognosis is therefore unfavorable, provided syphilis can be excluded from the factors of its causation.

Treatment.—If the disease be of syphilitic origin, I invariably employ subcutaneous injections of the corrosive sublimate of mercury or the mercurial bath. I use these in connection with the internal administration of large doses of the iodide of potash or calcium. I am convinced that the effects of the iodides are more lasting (even in tertiary syphilis) when combined with a mercurial treatment than when given alone. The effects of the mercurial bath or of the sublimate injections are more immediately beneficial to my mind than those gained by the administration of mercurials in any other way. In fact, I have arrested symptoms in this way which all other methods have failed to relieve.

In non-specific cases, I have given the iodides in large doses, and sometimes minute doses of the bichloride of mercury by the stomach. I also employ the bromides in combination with ergot.

When paralysis is developed, it is my custom to place the patient at

once upon strychnia and electricity. Strychnia may be injected into the paralyzed muscles if they fail to improve rapidly.

Counter-irritation at the back of the neck, either by the actual cautery, severe static sparks, or blisters, often does good. In some instances a seton may be introduced at this point to keep up active counter-irritation.

The bowels and the bladder should be the objects of careful supervision. It may be necessary in some cases to use a catheter at regular intervals, and possibly to irrigate the bladder when symptoms of cystitis appear.

Finally, the mental symptoms must be treated by enforced quiet of both the mind and body. The milk diet (from two to three quarts a day) and the exclusion of all other foods has given happy results in my hands in many forms of mental disorders. I frequently keep a patient strictly upon this regimen for several weeks. Some of my patients have lived largely upon milk for many months.

The hot-water treatment, to which I have referred elsewhere, is indicated in some cases as a valuable adjunct to the remedies already mentioned.

TUBERCULAR MENINGITIS—HYDROCEPHALUS.

Meningitis of the tubercular type (the so-called attacks of "water on the brain") is most commonly observed during childhood.

The acute variety is described by German authors under the name of "*basilar meningitis*," because the morbid lesions are limited to the base of the skull. English authors employ the term "*acute hydrocephalus*," when speaking of this variety.

The "chronic variety" of tubercular meningitis differs radically from the acute form in its morbid anatomy, symptoms, and duration.

The two will be discussed separately because of their wide dissimilarity.

ACUTE HYDROCEPHALUS.

The pia mater, in this disease, becomes the seat of tubercular deposits and inflammatory exudation at the base of the brain.

It is essentially a disease of childhood, although it may occasionally be observed in the adult.

Huguenin describes under the term "*leptomeningitis infantum*" a form of hydrocephalus of the acute variety, which, in his opinion, is purely inflammatory, and is not associated with the formation of tubercle. He excludes all "predisposition" of an hereditary character to this disease. Among the exciting causes of this variety of hydrocephalus he mentions the following: dentition, eruptive fevers, pulmonary diseases which are accompanied with high fever, concussion of the

cranium, intestinal catarrh, and the ingestion of alcohol. He considers an acute effusion into the ventricles of the brain as the chief pathological manifestation of this variety of hydrocephalus.

Morbid Anatomy.—I shall confine my remarks upon this head to the conditions found in typical cases of acute hydrocephalus of the tuberculous type. These include (1) deposits of tubercle; (2) an exudation of an inflammatory character in the meshes of the pia; and (3) a dropsical effusion into the ventricles.

In the perivascular lymph spaces, chiefly but not exclusively at the base of the brain, we find *deposits of miliary tubercles.* They are most abundant at the bifurcations of the blood-vessels. They tend to mechanically compress the blood-vessels, and when abundant they may occlude vascular trunks or their branches. In the fissure of Sylvius and in the region of the circle of Willis we are apt to find the most abundant deposit of tubercle, although they may also be scattered here and there along the vessels upon the convexity of the brain. I have observed them also in the longitudinal fissure, and upon the cerebellum and spinal meninges.

The *amount of tubercular deposit varies* between extreme limits. If very abundant, the vessels may be completely imbedded in them; again, they may be so scattered as to give to individual vessels an appearance of localized swellings, which has been compared to a "string of beads;" finally, they may in some cases be overlooked by a casual observer on account of their scarcity.

The effects of partial or complete occlusion of cerebral vessels by tubercular deposits may be manifested (1) by multiple hemorrhages from collateral fluxion; (2) by spots of red softening; and (3) by effusions of serum into the ventricles and at the base of the brain.

The tuberculous masses are found in this disease to exist in all stages of development. Some may appear as large as a small pea, from a confluence of smaller nodules; others may scarcely be visible to the naked eye; finally, some may be seen to have undergone granular and caseous degeneration. Under the microscope, normal tubercle appears as small grayish-white, semi-transparent, and partly gelatinous bodies.

A well-marked *inflammatory exudation* is found in addition to the deposit of tubercle at the base of the brain. This may consist of serum alone, which is usually more or less turbid; or it may appear as a yellowish sero-fibrinous layer which is most apparent about the Sylvian fissure, and along the course of the larger blood-vessels. It may cover the entire base of the brain, extending even to the under surface of the cerebellum. The pia will be found to have lost its lustre and to be thickened, opaque, and inelastic. It is more easily torn than in health. Finally, the *ventricles*

are found to be dilated, and to contain a serous effusion. The lining membrane (ependyma) is thickened. The descending horn of the lateral ventricle is particularly liable to become overdistended. The exudation into the ventricles may be purulent, in rare instances. As a rule, it consists of a serous fluid which is more or less turbid from an admixture of white blood-corpuscles and epithelium. Tubercles may be often detected in the ependyma, and along the vessels of the choroid plexus.

The effusion within the ventricles, if large in amount, may exert pressure upon the convolutions of the cerebrum, and thus cause them to appear more or less flattened and distorted. The brain-substance may also appear dryer than normal, on account of an anæmia that has been similarly produced.

In closing my remarks upon the morbid anatomy of this disease let me remind the reader that all the typical changes mentioned may not exist in each case. The yellowish deposit at the base of the brain may be absent; the distension of the ventricles forms an insignificant lesion in some cases, and may even escape detection; finally, the deposit of tubercle may be very slight and limited in extent, or, again, it may be detected everywhere, even in the substance of the brain itself.

Tuberculous meningitis is associated in many cases with the pathological manifestations of general tuberculosis. It is well, therefore, to examine the viscera of the thorax and abdomen as well as the spinal cord at every autopsy.

Etiology.—Between the first and seventh year, children of a tubercular diathesis are particularly prone to develop this disease. It may occur also from acquired tuberculosis, which follows infection from caseous matter, etc. This probably accounts for its occasional presence in adults. It may accompany general tuberculosis as one of its manifestations.

Among the exciting causes may be mentioned any of the exhausting diseases, such as the fevers, diarrhœa, etc.; injuries to the head; dentition; otitis media; eruptions of the face or scalp; and improper nourishment and hygiene.

These causes may occasionally produce the so-called "lepto-meningitis" described by Huguenin, in which all the morbid changes described except the presence of tubercles are found.

Large and densely populated cities exhibit a greater mortality in children from this cause than the rural districts of smaller towns. Children who are not suckled, and who live in poorly-lighted and imperfectly-warmed apartments, are very apt to develop an impairment of the physical powers. This often leads to glandular swellings, the development of hydrocephalus, and other manifestations of tuberculosis or scrofula.

Acute hydrocephalus, when it attacks adults, is most often encountered between the twentieth and fortieth years. Males are more often attacked than women.

Symptoms.—These will vary with the pathological changes which have occurred. Enough has been said already respecting the lesions found in the brain after death to show the basis of this deduction. The extent and amount of the basilar exudation, and the amount of fluid poured into the ventricles will determine the course of the disease and its symptoms rather than the presence or absence of tubercle.

The *advent* of acute hydrocephalus may be very insidious or very rapid. The former method of attack is observed, as a rule. The latter method of onset is frequently attended with convulsions.

It is a custom with some authors, although serious objections can be advanced against it, to divide the symptoms of this form of meningitis into those of distinct stages. Thus Huguenin speaks of a stage of irritation, a second of pressure, and a third of paralysis. Examples of a similar kind might be cited from other authorities. Now, while this may be desirable from an author's point of view for beginners, the exceptions to any type presented are so frequent as to almost disprove the rule.

The *premonitory symptoms* (when the advent is gradual) are such as may attract the notice of the nurse or parents. The child becomes peevish and irritable, is observed frequently to stop in its play and rest the head upon the hands or floor, or upon the knee of the mother; it gradually becomes dull and apathetic, and desires to remain quiet; it sleeps in a restless manner; the appetite becomes very capricious; the tongue may be coated; the breath is apt to be offensive, and constipation often alternates with diarrhœa. Progressive emaciation of the body and limbs takes place. This is a symptom of diagnostic importance, when it is accompanied by those previously mentioned. The temperature of the body may rise slightly in the evening. Sometimes a hacking cough may be developed. The countenance of the child is apt to lose its expression of vivacity, and to become dull and immobile. Pallor is usually developed. Transient attacks of flushing of the face may be observed, but they are of short duration. Headache is not common.

These prodromata may last only a few days in some subjects, while in others they may persist for weeks.

First Stage.—This stage gives clinical evidence of *cerebral irritation*. The advent of the disease is marked by a variety of symptoms of a more marked character than the prodromata. Vomiting is frequently developed. It may be very persistent and projectile in character. Headache is also complained of by the child. It is very severe as a rule, and causes the sufferer to cry and put the hands to the head. It is not

constant, and is usually confined to the frontal region. It may prevent protracted sleep and cause paroxysms of screaming.

In some instances, the onset of this disease is marked by convulsions of an epileptic character. In these cases the prodromata may have been absent.

During sleep the child flexes the thumbs upon the palms of the hands, grinds the teeth together, and rolls the head from side to side or bores it into the pillow. It often awakes during sleep with the so-called "hydrocephalic cry," and spasmodic movements of the ocular and facial muscles are sometimes observed.

Now, when we examine such a child with care, we shall see that the abdomen is retracted, hard, and "boat-shaped;" that the pupils are small; that the skin of the abdomen or thorax will show a red line in about thirty seconds after the finger-nail has been drawn across it (*tache cérébral* of Trousseau); that pressure upon the fontanelle, in a very young child, will increase the symptoms of cranial pain; that the tongue is dry and coated except at the edges and tip, which are generally red; that there is photophobia, and often strabismus; and that the thermometer shows a marked elevation of temperature which exhibits evening exacerbations and morning remissions of about two degrees.

The *temperature* rarely rises above 103° F. It may not exceed 101° F. When convulsions appear at the onset, it may go two or three degrees higher. The exacerbations and remissions of the fever are not as regular as in some other forms of disease.

The *pulse* is increased in frequency, but is regular, full in volume, and compressible, except upon excitement or muscular exertion. It may then be somewhat irregular.

In some cases, the *breathing* may be somewhat irregular; but as a rule the rhythm is normal.

The restless and fitful sleep gradually gives place to an *increasing drowsiness.* The child sleeps constantly unless aroused.

The *duration of this stage* is usually about a week. It may, however, be prolonged to ten or even fourteen days.

Second Stage.—When the brain begins to exhibit the clinical evidences of *depression* (as a result of effusion into the ventricles and at the base of the skull) the symptoms of invasion disappear and new ones take their place.

The pupils, which were contracted, now begin to dilate irregularly. They respond slowly to light. The head becomes forcibly extended by a tonic rigidity of the muscles of the neck so that it bores into the pillow. Sometimes the muscles of the back contract so forcibly as to cause opisthotonos. Muscular spasms and paroxysms of delirium are developed. In some instances, the patient lies motionless upon the back, with the eyes

staring fixedly and unconscious of surrounding objects. The projectile vomiting of the first stage ceases. The urine and fæces may be passed involuntarily.

The *muscles* show the effects of cerebral disturbance in various ways. We have already alluded to the tonic spasm of the back muscles. The muscles of the upper extremity are usually thrown into a state of excitability, as shown by a perpetual movement of the fingers, picking of the bed-clothes, opening and shutting of the hands in an aimless way, etc. General convulsions may occur from time to time. The ocular muscles may be affected. When so, the third cranial nerve and also the optic chiasm or the optic tracts are apt to be simultaneously involved. We are liable to encounter nystagmus, strabismus, ptosis, and pupillary spasm in such cases. Sometimes the facial nerve may be involved, and facial spasm or paralysis may then ensue. I recall an instance where the disease was associated with otitis media, in which the onset was accompanied by facial palsy.

The *temperature* of the body falls during this stage. It may reach the normal point.

The *headache* apparently persists in this stage, although the patient does not complain of it. We are justified in drawing this deduction from the fact that these subjects utter at intervals a peculiar cry, known as the "hydrocephalic cry." It seems to be partly involuntary and to express both alarm and pain. The facial muscles assume the attitude expressive of pain when the cry takes place.

The *respiration* becomes more or less irregular during this stage. In some instances I have observed typical "Cheyne-Stokes breathing." These symptoms, as well as disturbances of the heart's action and impairment of the *power of swallowing*, which often exist, indicate that the pneumogastric nerves are involved.

The *ophthalmoscope* may enable us to detect infiltration about the papilla, varicosities of the veins of the retina, small punctate hemorrhages, and whitish granulations of the choroid and retina in some instances. A choked disk has been observed also.

The *urine* may contain albumen. It is usually high-colored, scanty, and abundantly impregnated with chlorides and phosphates.

The *fæces* are often slimy, and are greenish in color, and very offensive in the large majority of cases.

The *tongue* becomes covered with incrustations, in which changes the lips and gums may also participate.

Some years ago, my attention was called to the fact that the second stage of this disease is often accompanied by a *short interval of apparent convalescence*. The patient may regain consciousness, be free from delirium or pain, eat voraciously, and excite the belief that recovery has

begun. But, in my experience, these intervals are of short duration, and the patient passes after a lapse of a few hours into deep coma again.

Finally, hemiplegia may occasionally be developed. It indicates pressure upon the motor tracts which join the cells of the cerebrum with those of the anterior horns of the spinal cord. (See page 258.)

Third Stage.—This is characterized by an increasing intensity of the symptoms of the second stage, with an exacerbation of the febrile symptoms of the first stage. Huguenin calls it the "stage of paralysis," because that symptom is a very prominent one. Hammond names it the "stage of recurrence," because the febrile symptoms return and the cerebral disturbance is more profound.

We observe, during this stage, the development of a paralysis of some part of the body, which is permanent. Convulsions and contracture of the muscles of the back, neck, and jaw may precede the paralysis. Reflex movements can be excited in the paralyzed limbs, although the power of voluntary motion is lost. The face, eyes, or limbs, may be the seat of paralysis. It is probably due to excessive intra-cranial pressure.

The temperature rises higher than it did in the first stage. I have observed it to reach 107° F., in one instance, a few hours before death occurred. It often rises to 105° F.

The pulse becomes greatly accelerated, small in volume, and intermittent. It resembles the pulse observed in animals after section of the vagus.

Coma becomes profound. It is accompanied by a clammy sweat, involuntary evacuations, dysphagia, dilated pupils, stertorous breathing, a tympanitic abdomen, and paralysis.

Death usually occurs from asphyxia, or convulsions. It may ensue from impairment of the heart's action.

Duration of the Disease.—This admits of the widest latitude of statement. Cases have been reported where death has occurred in a few hours, while others have lasted for many weeks.

Those which begin with paralyses or convulsions are rapidly fatal, in my experience.

The age of the patient may modify the duration of the disease. While I believe that no pathological differences exist between the infantile and adult varieties of the disease, its duration is more apt to be abnormally short in the child. When it develops as a complication of general tuberculosis, death often occurs during the first week. A vigorous constitution may cause the disease to be prolonged over a much longer period. The vague character of the prodromal symptoms in many cases renders it advisable to count the duration of the disease from the first well-marked cerebral symptom.

Differential Diagnosis.—In common with many other observers, I have seen cases of acute hydrocephalus which have resembled those of

acute Bright's disease, infantile remittent fever, acute meningitis, gastro-enteritis, and typhoid fever. A condition called "spurious hydrocephalus," which develops in children, may also closely resemble it in many respects.

The *acute form of Bright's disease* is to be distinguished by the presence of albumen and casts in the urine, the existence of œdema of the face and limbs, the peculiar waxy skin of uræmie poisoning, and the absence of the prodromal symptoms of hydrocephalus. The history of the case may also afford a clue to the cause of the attack.

Infantile remittent fever is accompanied by a very high range of temperature (105°–106° F.), which exhibits *regular* remissions and exacerbations. It exceeds the customary limits of the fever of hydrocephalus (103° F.), and does not fluctuate irregularly. The pupils are normal. The pulse does not intermit or become irregular. The hydrocephalic cry is not developed, nor are the thumbs flexed upon the palms during sleep. The patient does not grind the teeth. The vomiting is not projectile in character; it is accompanied by *retching*. The abdomen is distended with gas, and is tender. "Pea-soup discharges" from the bowels occur.

Acute meningitis is less frequently accompanied by ocular symptoms, and a retracted and "boat-shaped" abdomen. It does not produce the hydrocephalic cry, nor any of the prodromata of the tubercular variety. It is a disease of sudden onset and rapid progress. The temperature runs high, and exhibits very slight remissions. The pulse is not irregular except when death is near. It attacks adults and children in good health. Delirium and convulsions occur earlier than in the tubercular form.

Gastro-enteritis fails to produce the prodromata of tubercular meningitis, and also headache, projectile vomiting, hydrocephalic cry, paralyses, irregular pulse, etc. It is associated with diarrhœa, tenderness of the abdomen, and pain in the bowels.

Typhoid fever is accompanied by diarrhœa, tympanites, a characteristic eruption upon the abdomen, and a typical range in temperature. The prodromata of hydrocephalus, and the more prominent symptoms of that disease mentioned above are wanting.

Spurious hydrocephalus usually follows an attack of cholera infantum. It is sometimes accompanied by a few of the cerebral manifestations of the tubercular variety, but the abdomen is distended and tender, the fontanelle is depressed, paralyses do not occur, and the temperature and pulse do not indicate the more serious affection.

Prognosis. — Tuberculosis of the meninges and brain generally terminates in death. So large is the percentage of mortality that most authors deny that a case of recovery has ever occurred. On the

other hand, isolated cases of apparent recovery have been reported by Formey, Politzer, Rilliet, Hahn, Barthez, and others. Personally, I have never seen a case where recovery has taken place, and I am inclined to believe that lepto-meningitis has existed in all cases where a favorable result has occurred. It is certain that idiocy, epilepsy, and the chronic type of hydrocephalus is dependent upon a tubercular diathesis in some instances; but it is not yet proven that they have ever been preceded by a typical attack of acute hydrocephalus.

Treatment.—My remarks made concerning the prognosis of this disease must preclude any suggestions relative to a cure of this affection. I have tried all the methods of treatment suggested by authors, including the iodide of potash, the mercurial salts, the phosphate of soda, depletion, etc., without any apparent benefits. Notwithstanding the incurability of the disease, the individual symptoms should be ameliorated, however, as long as life exists, by judicious medication. The preparations of opium and the bromide salts are indicated when headache, photophobia, restlessness, and jactitation are present. Ice-bags may often be applied to the head with apparent benefit. The bowels should be moved by laxatives when constipation exists. The patient should be kept in a quiet and darkened room. When the convulsive attacks are frequent and severe, I occasionally administer anæsthetics. Venesection or local depletion is of no benefit, and should not be performed.

The *prophylactic treatment* of this malady deserves more consideration than is often paid to it by medical men. I am convinced that I have saved the lives of many children born of tubercular parents by the steps which I shall here advise.

It is my custom, with children who are predisposed to this affection, to insist that the child shall be nursed from birth by a woman who is free from all hereditary or acquired diseases. The child should be kept in the country and allowed to romp in the open air until seven years old. The hygienic surroundings and the diet of the child should also be carefully looked to until the period of danger has passed. It is my custom to administer cod-liver oil to those who remain thin or poorly nourished in spite of the precautions mentioned. Bathing the body in cool water, and subsequent frictions of the skin are of benefit. It is also well to change the climate and surroundings frequently, so as to avoid unnecessary exposure to cold or dampness during the winter months, or to extreme heat in the summer. By these means many children are reared to adult life when previous offspring of the same parents have died of hydrocephalus.

I would caution the reader also against allowing children with hereditary tendencies to tuberculosis to be subjected to extreme or prolonged excitement of any kind. It is not well to send them to school until the

period of danger has passed. I am convinced that playing with children before going to bed, especially if impressionable or emotional, conduces to imperfect sleep and physical debility. The custom of forcing the young intellect by feats of memory or confinement in the school-room is also very pernicious.

The proportion of *shallow orbits* and *hyperopic eyes* in persons who have a tuberculous ancestry is very large, as proven by the examination of many hundreds of cases by my friend Dr. G. T. Stevens. My own observations (lately published in a paper read by me before the International Medical Congress, 1888) confirm this statement, and lead me to believe that "latent hypermetropia" (p. 127) is remarkably frequent in this class of subjects. This congenital defect (if it exists) should be properly corrected by glasses before the child is allowed to study. I have no doubt that this one factor has more to do with the so-called "tuberculous predisposition" than many of us imagine. The health of any child must of necessity be seriously affected by this well-recognized source of physical disturbance and excessive nervous expenditure.

CHRONIC HYDROCEPHALUS.

This disease is essentially surgical; the acute form comes more particularly under the province of the physician. In either case, however, the "tubercular diathesis" seems to influence its development.

Chronic hydrocephalus appears to be produced by a low grade of inflammation which attacks the *lining of the ventricles* during fœtal life or early childhood. In some instances, it appears later in life. In a few isolated cases, the serous effusion seems to be external to the brain. Some authors state that this latter condition never occurs except as the result of a hemorrhage into the cavity of the arachnoid. They consider the condition which stimulates true external hydrocephalus as an evidence either of a congenital defect in development (the cerebro-spinal fluid taking the place which the brain should have occupied), or of atrophy of the brain-substance that has resulted from the pressure of the fluid within the ventricles.

Morbid Anatomy.—In chronic hydrocephalus, the sutures fail to unite and the calvaria does not ossify as in health. The ventricles are enormously distended, and the channels of communication between them are widely dilated and open. Finally, the convolutions are flattened and the cerebral substance rendered extremely thin and attenuated. The ossa triquetra are often found to be excessive. A complicating meningitis (which often exists) may involve some of the cranial nerves (especially the optic) and induce atrophy of them. Fluctuation may be detected often in the region of the fontanelles and the open sutures.

The deformity of the cranium is evidenced by an overhanging brow,

an increase of the circumference of the cranium and its disproportion to the size of the face, and by open fontanelles and unclosed sutures. The latter are often widely separated. The mental condition of the subject is below the normal standard whenever the pressure of the fluid has induced changes in the brain-substance.

Instances have been reported where twenty or more pounds of fluid have been found to exist within the skull. The circumferential measurement of the head has been known to exceed forty inches.

The fluid formed during the progress of this disease consists of water, albumen, floceuli of lymph, salts of lime, soda, and potash, epithelial and blood cells, and urea.

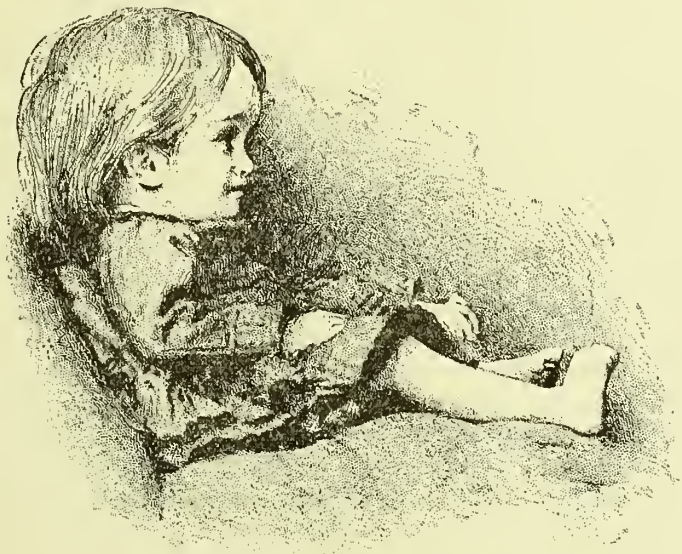


FIG. 84.—CHRONIC HYDROCEPHALUS. (After a photograph.)

The condition of the brain-substance found after death depends upon the seat and extent of the dropsical effusion. It may be softened and atrophied, when degeneration has been produced by a pressure which has impaired its blood-supply. Occasionally it is rendered abnormally resistant and of firm consistence.

Etiology.—The hereditary nature of this affection is proven by the fact that several children of the same parentage have been successively attacked in many recorded instances.

The acquired form may be independent of any hereditary taint. It has been observed to accompany congenital defects of the walls of the blood-vessels, chronic passive hyperæmia, occlusion of the cerebral sinuses, rickets, syphilis, alcoholism, some of the eruptive fevers, and

atrophy or imperfect development of the brain. Aged subjects may sometimes be attacked. In the young, it is frequently developed during or previous to dentition.

Symptoms.—The deformity of the head, which is characteristic of this disease, may occasionally be present at birth. As a rule, however, the increase in the size of the head does not manifest itself until the child is a month or two old. If it exists at birth, the child is apt to die soon after delivery.

Whenever the deformity becomes well developed, it seems to me impossible for a medical man to err in diagnosis.

The countenance of chronic hydrocephalus (see fig. 84) is perhaps the most typical of any of the conditions to which the attention of the physician or surgeon is directed. In it, the frontal bone is tilted forward so that the forehead, instead of slanting a little backward, rises perpendicularly, or even juts out at its upper part, and overhangs the brow. Watson thus describes it: "The parietal bones bulge, above, toward the sides; the occiput is pushed backward, and the head becomes long, broad, and deep, but flattened on the top. This, at least, is the most ordinary result. In some instances, however, the skull rises up in a conical form like a sugar-loaf. Not unfrequently the whole head is irregularly deformed, the two sides being unsymmetrical. Some of these rarer varieties of form are fixed and connate; others are owing, probably, to the kind of external pressure to which the head has been subjected. While the skull may be rapidly enlarging, the bones of the face grow no faster than usual, perhaps not even so fast, and the disproportion that results gives an odd and peculiar physiognomy to the unhappy subjects of this calamity. They have not the usual round or oval face of childhood. The forehead is broad, and the outline of the features tapers toward the chin. The visage is triangular. The great disproportion in size between the head and the face is diagnostic of the disease, and would serve to distinguish the skull of the hydrocephalic child from that of a giant."

As a result of the compression and atrophy of the brain, the limbs do not develop as in health, the abdomen is usually distended and tympanitic, the child is unable to hold the head erect, and the pupils become more or less dilated. Sometimes the eyes bulge from their sockets. At the fontanelles and open sutures fluctuation may be detected when the dropsical effusion is external to the brain.

The digestive functions are carried on imperfectly during hydrocephalus. Hence these children exhibit inanition, and are prone to develop some form of inter-current disease. I have seen instances where death has occurred from starvation.

In a certain proportion of cases, life may be prolonged for years. It will then be observed that the power of walking is acquired imperfectly

and late. The gait is characterized by tottering, stumbling, and a peculiar awkwardness of movement indicative of idiocy. The mental powers are more or less impaired. These subjects are generally very irritable and peevish, and are liable to have recurring attacks of fever, nausea, and vomiting. Convulsions are frequent, and paralysis of special groups of muscles may be developed during the progress of the disease.

I have personally seen one hydrocephalic subject who lived to be over thirty years of age. It is not common, however, for life to be prolonged to such extreme limits. The majority die within three or four years. Whenever anæmia or asthenia are induced, the patient dies within a period of twelve months, as a rule.

Differential Diagnosis.—Little need be said regarding the diagnosis of this disease. It can hardly be confounded with any other affection. Although the cranial deformity of rickets produces a lack of symmetry in the contour of the head, the open sutures, the sense of fluctuation, and the characteristic deformity of hydrocephalus do not exist, nor are mental and physical derangements similar to those of hydrocephalus induced.

Prognosis.—The large majority of these subjects die within one year. The exciting causes of death include the following complications: Meningitis, apoplexy, ependymitis, anæmia, asthenia, paralysis of the heart, and escape of the fluid by rupture of the aponeurosis covering the skull and open sutures. When recovery takes place, the fluid ceases to form and the sutures gradually close.

Treatment.—Suggestions both of a medical and surgical character have been brought forward from time to time as productive of good in this disease; but I have laid most of them aside, as I have found them of doubtful utility. Occasionally, it is well to strap the head. I doubt if the exudation within the skull was ever arrested by mechanical compression; but it sometimes appears to give ease to the patient, and it invariably gratifies the parents and interested friends.

Tapping of the head may be employed with advantage when fluctuation can be detected at the fontanelles. It is contra-indicated when inflammatory changes are present. The aspirating needle should be introduced at the anterior fontanelle, and a few ounces of fluid only should be withdrawn at one time. The head should be bandaged after the operation, and the child carefully watched for inflammatory sequelæ. Langenbeck has employed aspiration of the lateral ventricle (by passing an aspirating needle through the roof of the orbit behind the upper eyelid) in the internal variety of chronic hydrocephalus. I have never resorted to this method of treatment, because I regard it as a dangerous one and of questionable benefit to the patient.

The medicinal treatment of this disease must be purely symptomatic. It is well to administer tonics, such as cod-liver oil, the iodide of iron,

and quinine, at intervals throughout the course of the disease. Some authorities recommend the iodide of potash. Calomel in minute doses (gr. $\frac{1}{4}$–$\frac{1}{2}$ daily) may be employed until severe purging occurs. Personally, I have little faith in the curative properties of any drug in this disease. Plenty of fresh air, a good nutritious diet, warm clothing, frequent bathing and rubbing of the skin, and a change of residence at intervals will assist Nature more than constant medication. It is perhaps advisable to administer some of the phosphates when the disease coexists with rickets. I have never found the slightest benefit from the use of mercurial inunctions.

ENCEPHALITIS.

The substance of the brain may take on inflammatory action with or without the existence of a complicating meningitis. It is generally circumscribed, although many spots may be simultaneously affected. The latter is sometimes termed the "general" variety. Sometimes the gray matter of the cortex is alone involved. Again, only the medullary substance of the brain may be implicated. Finally, the basal ganglia (the "corpora striata" and "optic thalami"), the cerebellum, the medulla oblongata, the pons Varolii, the crura cerebri, and the floor of the fourth ventricle have been known to be the seat of this condition.

Morbid Anatomy.—The existence of encephalitis may be manifested after death (1) as spots of injection associated with abnormal friability; (2) by the presence of punctate extravasations seen on cross-sections of its substance; (3) as localized indurations; (4) as red softening of the brain-substance; (5) as circumscribed collections of pus; and (6) by gangrene.

Whenever cerebral abscess has occurred, the cavity is usually encapsulated by a new connective-tissue formation.

Rindfleisch divides the stages of cerebral abscess as follow: 1. A stage of hyperæmia; 2. The development of infarction (page 230); 3. Œdema or hemorrhagic extravasation; 4. Proliferation of the cell elements; 5. Hypertrophy and induration (in chronic cases); 6. Softening of the brain-substance; 7. Suppuration; 8. A condition of fœtid suppuration, resembling gangrene, which is occasionally preceded by the development of a false membrane; 9. Atrophy, as a late result of the inflammatory changes.

Etiology.—Among the causes of encephalitis which tend to induce suppuration may be mentioned pyæmic infarction, direct injury to the head, disease of the internal ear or temporal bone, diseases of the nasal cavity, syphilitic disease of the bones of the cranium, diseases of the orbit, cancer, and certain idiopathic causes which are not well understood.

Symptoms.—Encephalitis is so closely allied to red cerebral softening that it will be further discussed under that heading. The symptoms of the disease must of necessity vary with the seat and extent of the lesion. Hence it is difficult to interpret them correctly unless the functions and anatomy of the various component parts of the brain are well understood. Pages 68 to 102 will aid the reader in mastering cerebral localization.

Differential Diagnosis.—The points of value in the discrimination of this disease will be given in those pages which treat of the diagnosis of cerebral softening.

CEREBRAL SOFTENING.

The three forms of this condition which are recognized by most authorities are the white, yellow, and red.

Morbid Anatomy.—The pathology of softening of the brain may thus be summarized:—

The *white variety* is a chronic condition in the great majority of cases, and is usually dependent upon some disease of the small arteries and capillaries which *gradually* deprives the parts of their normal nutrition. There is no hyperæmia. The parts are usually of an opaque dirty white.

White softening may sometimes be acute; in which case it is due to a sudden obstruction of some *artery of large size* by an embolus or a thrombus.

The *yellow variety* is simply an altered state of either the white or the red. Its color is due either to the presence of altered blood-pigments which have arisen from a previous slight extravasation; or to a fine state of division and a close aggregation of particles of fat formed within a mass of the former variety.

The *red variety* is commonly an acute affection. As has been stated, it follows vascular occlusion from an embolus or thrombus; or it may be the result of an attack of encephalitis. A marked extravasation of blood into a mass of white softening may cause a red appearance to the mass, but the microscopical appearances will differ from that of the acute form now under consideration. In the red variety, there is intense hyperæmia from the onset, followed by a rupture of the capillaries and an extravasation of blood. Its pathology is similar to that of "infarction" elsewhere in the body (page 229).

All forms of cerebral softening are liable to be accompanied by disturbances of motion or sensation, aphasia, and mental impairment. The seat and extent of the lesion will govern the type of its external manifestations. The history of the patient will often be indispensable in deciding as to the existence of softening, if in the anterior part of the

frontal, the occipital, or the temporal lobes, where the so-called "motor centres" of the brain are wanting. (See Fig. 5.)

There is more or less œdema into the brain-substance which surrounds a spot of red softening. This often causes the area of softening to appear as an elevation above the plane of the section when the brain-tissue is divided with the knife. In cases where the focus of softening is of the white or yellow variety this elevation is wanting.

The débris which constitutes a focus of cerebral softening will be found to consist of fat granules, altered blood-corpuscles, pigment masses, pus-cells, disintegrated nerve-tissue, caseous matter, and large spherical cells, filled with fat granules (Gluge's corpuscles). Each variety of softening causes variations in the relative proportions of these elements. The red variety will exhibit a large admixture of blood-

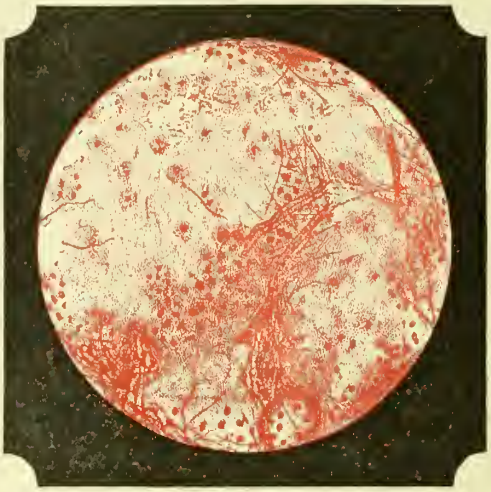


Fig. 85.—Softened Brain-Tissue (After Fox.)

corpuscles, pigment granules, and Gluge's cells; the yellow and white varieties will be accompanied by an excess of fatty matter or caseous substance, and a small admixture of altered blood pigments, corpora amylacea, and crystals.

Etiology.—It is a mistake to suppose that cerebral softening occurs only in the aged. Old age is indeed a prominent factor in exciting cerebral thrombosis; and that condition is followed, as a rule, by softening of the brain in the old. Still, the more common exciting causes of this disease include (in addition to cerebral embolism and thrombosis) pyæmia, syphilis, alcoholism, sun-stroke, and cerebral hemorrhage, all of which may exist independently of old age. I have to-day under my care a young married lady (thirty years old) who has marked cerebral softening that was induced by an embolic attack during the puerperal state, independently of any lesion of the valves of the heart. Another of my

patients developed cerebral softening as a result of gummatous pachymeningitis when but thirty-five years of age. All the exciting causes of abscess of the brain may occasion cerebral softening under favorable circumstances. Among these may be mentioned the exanthematous fevers, glanders, the puerperal state, mercurial- and lead-poisoning, necrosis, osteo-myelitis, pyæmia and septicæmia, abscess of the viscera, and many others.

The *white variety* results from causes that tend to so impair the blood-supply of the softened part as to deprive it of nutrition without creating at the same time a hemorrhage from the surrounding capillaries. It is often designated as the "non-inflammatory form," in contrast to the red, which is commonly of inflammatory origin.

The nature and mode of suppression of the blood-supply to portions of the brain, more or less limited, governs to a great extent the variety of softening which results. As has been stated in a previous page, the arteries, capillaries, or sinuses of the brain may be independently occluded. Thrombosis or embolism may be the immediate cause of such occlusion, or the blood-supply may be arrested by pressure upon the vessels by new growths within the skull, or from without, as in the case of hemorrhage, tumors, ligation, œdematous infiltration, etc.

The rapidity of arrest of the circulation, and the extent of collateral fluxion which immediately follows (within twenty-four or forty-eight hours), are the factors which govern the results which follow. The collateral circulation may be sufficient in some cases to arrest the immediate death of the parts suddenly deprived of blood by an embolus or thrombus, or some quickly-developed and extreme pressure upon the blood-vessels. Again, it may be so great as to cause a capillary hemorrhage, giving the softened area a red appearance (*infarction*) immediately after the arrest of its normal blood-supply. Finally, inflammatory action, as in true encephalitis, may create the red variety of softening.

Symptoms.—It is not always possible to make a positive diagnosis of cerebral softening. The symptoms may be influenced to a greater or less extent (1) by the exciting cause; (2) by the seat of the focus of softening; (3) by the variety of softening which exists; (4) by the development of suppuration; and (5) by the occurrence of hemorrhagic extravasation into the softened brain-tissue.

Notwithstanding these elements of uncertainty in diagnosis, it is generally possible to arrive at some definite conclusions by a careful study of each case and the exclusion of the other conditions of the brain whose symptoms closely resemble those of softening.

Encephalitis or *inflammatory red softening* is accompanied, as a rule, by febrile symptoms of a marked kind. The temperature often rises to 103° F., and the pulse becomes more or less accelerated at first. Later

in the disease, the pulse may be slower than normal. Headache is a marked symptom. It is often accompanied by vertigo, somnolence, hyperæsthesia, formication, pruritus, neuralgic pains, vomiting, and confusion of intellect. Later, it may be attended with delirium, muscular twitchings, general convulsions, a tottering gait, imperfections of speech, motor or sensory paralysis, and coma. Amnesic or ataxic aphasia may be developed. The face frequently becomes flushed, and an irregularity of the pupils may be occasionally observed.

When an apoplectic extravasation occurs into the softened mass, profound coma and paralysis suddenly appear. Other evidences of cerebral disturbance may also exist. Hemianæsthesia will generally coexist with hemiplegia, the functions of sensation and motion being affected upon the same side, if the disease is confined to one cerebral hemisphere. When the hemorrhage is extensive, death occurs within forty-eight hours. In instances of a less severe type life is not destroyed, but the recovery of motility and sensibility is seldom complete.

The ophthalmoscope may often prove of service in the diagnosis of cerebral softening. It enables the physician to detect changes in the size and course of the retinal vessels.

Cerebral softening of the *non-inflammatory variety* may be due to an attack of embolism or thrombosis, or to mechanical compression of some of the cerebral arteries or sinuses. The method of onset will be modified, therefore, by the exciting cause. The symptoms of each of the causes enumerated have been given in preceding pages.

When aged subjects are attacked by cerebral softening, the history of the case is often extremely vague and unsatisfactory. Many instances have been recorded in which extensive foci of softening, abscesses of large size, and direct injuries to the brain have not been suspected during life, although they were encountered at the autopsy. We are justified in attributing diagnostic value, therefore, to certain symptoms when taken collectively, which would be of little importance alone. Thus, for example, when an aged person begins to exhibit an impairment of memory, unnatural peevishness and irritability, an imperfect control over the emotions, despondency, an incapacity for prolonged mental effort, physical weakness, a diminution of motor power in the legs or arms, a monotonous habit of speech or gesture, etc., we are forced to the conclusion that they indicate collectively some serious form of disease. We are strengthened in this conclusion if these symptoms are followed after a lapse of time by an awkwardness of movement, a tottering gait, dementia, or the sudden development of paralysis of sensibility or motility.

Cases of cerebral softening frequently develop a peculiar tendency to clip off words during attempts at conversation or reading. These cases

exhibit also an inability to maintain continuous muscular contraction, an excitability when questioned concerning their ailments, a lack of regard for personal cleanliness and the decencies of life, groundless prejudices, and many other evidences of mental decay.

Paralysis of motion may develop gradually (as well as suddenly) in connection with cerebral softening. In such a case the impairment of motility will generally be perceived in the fingers or toes first, and steadily advance toward the trunk. This method of attack is called "creeping palsy," in contradistinction from paralysis of sudden advent.

When cerebral softening is far advanced, it is not uncommon to encounter bed-sores upon the nates. They resist all methods of treatment, and indicate "trophic disturbances" of the skin.

Respecting the *seat of softening*, some deductions can occasionally be drawn during life from the symptoms presented.

When deglutition becomes difficult, or when the respiratory or circulatory functions are markedly affected, we are justified in suspecting that the basilar artery has probably been occluded, and that softening of the *pons or medulla* exists.

The ocular muscles may be rendered paretic by a focus of softening in the *cerebri crus*.

If the *temporal lobes* of the cerebrum are attacked, we may meet with disturbances of hearing, smell, and taste. Word-deafness may also be encountered, especially if the left hemisphere is involved.

Softening of the *occipital lobes* may give rise to visual disturbances, such as colored spectra, hemianopsia, a loss of the memory of past sight-perceptions, word-blindness, etc.

Evidences of impairment of motility are liable to be developed early when the *motor area of the cortex*, or that part of the *centrum ovale* through which the motor fibres pass to the internal capsule (fig. 6) are attacked.

Lesions of the *centrum ovale* may be diagnosed in some cases by tests mentioned on page 184.

The cerebral cortex, the basal ganglia (corpus striatum and optic thalamus of each cerebral hemisphere), and the white substance of the cerebrum are most frequently attacked. The area of softening seldom crosses the mesial line.

The *causes of death* in this disease are various. Some patients die of slow exhaustion. Others develop asphyxia, on account of a disturbance of respiration. Not infrequently convulsions produce death by interfering with respiration. Instances have been recorded where a patient has been choked to death during an attempt to swallow, or from regurgitation of food when in a convulsion. Finally, deep coma may develop before death.

Differential Diagnosis.—The inflammatory variety may be mistaken for acute meningitis. The non-inflammatory varieties sometimes resemble, in their symptomatology, the conditions of cerebral tumor, cerebral abscess, chronic meningitis, and hæmatoma of the dura.

Acute meningitis is accompanied by a higher range of temperature than red softening. Its stages are distinctly marked. It has a comparatively short duration. Headache is intense at the onset. Vomiting is a prominent symptom. The pulse is also characteristic.

Cerebral tumors do not, as a rule, cause as much embarrassment of speech or impairment of intellect as softening, because they affect the frontal lobes less frequently than some other regions. They are accompanied, in a large proportion of cases, by a pain in the head that is more

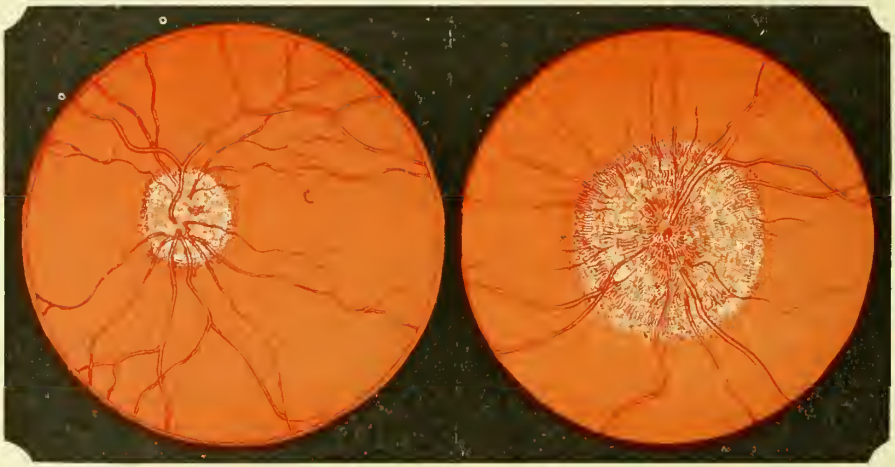


FIG. 86.—THE FUNDUS OF THE NORMAL EYE.—The reader should compare this cut with the next one in order to appreciate the appearance of the so-called "choked disk."

FIG. 87.—THE APPEARANCE OF THE FUNDUS OF THE EYE WHEN AN EXCESS OF INTRA-CRANIAL PRESSURE HAS CAUSED A NEURO-RETINITIS, RESULTING IN THE CONDITION KNOWN AS "CHOKED DISK."

localized than when due to softening of the brain. The cranial nerves are liable to be implicated early. Monoplegia or monospasm is not an infrequent symptom of tumor near the motor centres. Convulsions are more frequently observed in connection with tumors than with softening, and are of the epileptiform type. A history of syphilitic infection is often present, since many cerebral tumors are of the gummatous variety. Choked disk is generally detected when cerebral tumor is present. The thermo-electric differential calorimeter (Fig. 71) will often show a marked variation in the temperature of the scalp over the tumor from that of surrounding parts. This instrument is of great value, therefore, in such cases.

Cerebral abscess usually follows traumatism, or an attack of cerebral embolism or thrombosis. It may be accompanied by a swelling of the

scalp, the so-called "Pott's puffy tumor." It sometimes occurs in connection with pyæmia. Occasionally it follows an attack of cerebral hemorrhage or cerebritis. Rigors may be present when the abscess is forming, and more or less febrile disturbance will generally exist.

Chronic meningitis so closely resembles cerebral softening in some cases that the differential diagnosis is impossible. As a rule, however, the headache of chronic meningitis is more diffused than in softening. The intellectual faculties and speech are not as progressively embarrassed. Spasms of the limbs are frequently developed. The impairment of motility in the limbs is not as diffused as in softening.

Pachymeningitis interna (hæmatoma of the dura) may be confounded with cerebral softening. The history of the patient will generally suffice to clear up existing doubts. (See previous pages, which treat of this condition.)

The following table * will possibly aid the reader still further in making the discrimination between cerebral softening and cerebral tumor :—

CEREBRAL TUMORS.	CEREBRAL SOFTENING.
HEADACHE.	
Headache usually exists in the frontal or temporal region; occasionally in the occipital region. It is either intermittent or paroxysmal at first; but it tends to become continuous and rebellious to treatment. It is not necessarily over the seat of the tumor. This symptom is most marked in those cases where the meninges are subjected to tension by the growth of the tumor.	Headache is less severe, and usually confined to the frontal region. Paroxysms of pain in the head are less frequent. It is more commonly continuous.
VERTIGO.	
Vertigo is quite a frequent symptom in connection with the headache. If the tumor is near the temporal region, the vertigo is apt to be especially severe. The same deduction applies to tumors of the cerebellum.	Vertigo is not specially characteristic of cerebral softening.
EARLY EFFECTS OF LESION.	
Disturbances in both sensation and motion are apt to be developed when the cortex is subjected to irritation or is pressed upon by the tumor. Monoplegia, monospasm, monoanæsthesia, monoparæsthesiæ, aphasia, etc., may be encountered.	The mental faculties exhibit impairment early. Later in the disease the body may be suddenly rendered hemiplegic or the patient may become aphasic.

* This table is modified from one originally published in the Author's work on "Surgical Diagnosis," 3d edition.

Sensory Phenomena.

Late sensory disturbances which may develop include neuralgic attacks; numbness; formication; reflex cramps, etc. These may be followed by more or less general anæsthesia, abolition of the muscular sense, etc.

Sensory symptoms, when present, are not as marked as in tumors. In some situations, cerebral softening does not induce them.

Motor Phenomena.

The motor phenomena which may be induced comprise a stiffness of the muscles; a relaxed condition of one member or limb; spasms of the tonic or clonic type; tremor; permanent contraction of muscles; paresis; monoplegia; hemiplegia; paraplegia; unilateral ataxia, etc.

Crossed paralysis in any of its more common forms may occur.

Double or alternate paralysis, when present, is especially characteristic of tumors.

Hemiplegia (which is usually of sudden origin and complex) is more common than in tumors.

Aphasia is often developed with the hemiplegia.

Double or alternate paralysis is rare.

Special Senses.

Choked disk, anosmia, amblyopia, amaurosis, auditory disturbances, and loss of taste have all been observed to follow the development of cerebral tumors.

Disorders of the special senses are less frequent than in tumors; when present, they do not tend to progressively involve one special sense after another.

Speech

Is infrequently affected by motor or sensory aphasia. The speech is often embarrassed, however, by sputtering, imperfect utterance, etc.

Is frequently affected. When so, the condition of aphasia is commonly present.

Mental Faculties

Impaired late, if at all.

Impaired early.

Etiology.

Tuberculosis, cancerous cachexia, syphilis, and diseases which induce changes in the cranial bones, are frequent causes of tumors within the skull.

Frequently follows disease of the temporal bone; if so, it is preceded by aural disturbances.

Embolism, cerebral thrombosis, and apoplexy, are frequent causes of cerebral softening.

Symptoms in Common.

Both may be associated with headache.
" " " " impairment of mental faculties.
" " " " " " motion.
" " " " " " sensation.
" " " " " " speech.
" " " " " " special senses.

Prognosis.—The duration of life depends upon the cause, extent, and variety of softening. While it may be said that death usually results from cerebral softening sooner or later, I believe that recovery may occur. Many of the reported cases of cure are undoubtedly those of error in diagnosis; but small foci of softening may, to my mind, become encapsulated in young and healthy subjects, and do no more harm than extravasations of a limited extent.

Red softening is liable to lead to cerebral abscess. It frequently destroys life within two weeks. The non-inflammatory varieties may cause progressive symptoms for years. When death occurs in these cases, it is usually the result of some complication, such as those of the lung, meningitis, apoplexy, diarrhœa, interference with the centres of the medulla, bed-sores, anæmia, etc.

Treatment.—The fact that instances of apparent cure of this affection are occasionally reported (two of which I have personally observed) should justify us in not discarding all remedial measures.

The treatment of each individual case will be modified somewhat by the history of the patient. If the symptoms of cerebral softening follow an attack of cerebral embolism or thrombosis, the suggestions previously offered regarding the treatment of those conditions will be indicated. If it be due to some form of tumor, or the pressure of inflammatory exudation, efforts should be made to create absorption and to arrest its development. (See treatment of meningitis and tumors.) When cerebral softening follows any of the varieties of blood-poisoning enumerated in previous pages, the indications for treatment are to aid nutrition by all possible means, and to systematically promote physical vigor. In young and previously vigorous subjects, this can be done more satisfactorily than in aged or enfeebled constitutions.

The treatment of the acute variety should differ from that of the chronic form, since enforced confinement to bed, mental quietude, cold to the head, the internal administration of stimulants in some cases, and the employment of heat to the extremities are frequently indicated in the former.

When the disease is inclined toward chronicity the management of each case must be modified by the symptoms presented. It is always well to guard against any mental anxiety or fatigue of mind or body; hence pleasant surroundings, cheerful companionship, regular exercise, abstinence from business, cessation from reading or writing, driving or riding in the open air, etc., are important factors in the treatment. I prefer the milk diet to any other form of administering nutrition in these cases. One of my patients consumed three quarts of milk daily for over three months without solid food, and experienced the greatest benefit. The bowels should be carefully watched. It is my custom to give from

a teaspoonful to a tablespoonful of ungrouud white mustard-seed before breakfast and at bed-time to patients who suffer from habitual constipation. It acts charmingly in many eases.

As a tonic I often give to these patients a pill recommended by Hammond, consisting of a half-grain of the extract of nux vomica and one-tenth grain of the phosphide of zinc. One of these pills should be taken after each meal. Combinations of phosphorus, iron, strychnia, and quinine, are usually of benefit to these subjects. Cod-liver oil is of the greatest service in some cases.

The insomnia and delirium which often accompanies the disease can be overcome by the bromides of soda or potash (thirty grain dose), chloral (ten to twenty grain dose), cannabis indica (one-quarter grain dose), and by the judicious use of alcoholic stimulants.

Electricity forms a valuable adjunct to the other methods of treatment already mentioned. It is my custom to use the faradaic current daily upon the extremities when paralyzed, and the galvanic current (about fifteen or eighteen cells) transmitted from the forehead to the back of the head for about five minutes every other day, irrespective of paralysis. When the paralyzed muscles begin to exhibit evidences of defective nutrition, I employ the galvanic current in place of the faradaic.

The employment of the actual cautery to the nape of the neck, and of croton oil to the shaven scalp has been recommended. I have never tried the latter because I can see no indication for its use. The former I have tried with the most unsatisfactory results. The experience of most of the prominent neurologists seems to be in accord with my own. Hammond states that the speech was rendered more embarrassed and the mind weaker than before its application in his experiments with it.

Finally, the development of bed-sores should be regarded as a serious complication. They should be treated by a fenestrated air-cushion, or some form of dressing that will relieve the seat of ulceration from pressure.

The use of alcoholic stimulants is indicated when the vital forces are below par. To aged subjects I frequently give a liberal supply of wine with the meals. The effect of stimulation upon the patient is the guide to the quantity which should be prescribed in each case.

CEREBRAL ABSCESS.

Suppuration of the brain-substance may be due to a variety of causes. Cerebral abscesses may be single or multiple, and acute or chronic.

Morbid Anatomy.—Collections of pus within the brain are most frequently found in the centrum ovale of the cerebral hemispheres and next in the cerebellum. Occasionally they are developed in the substance of the basal ganglia, the crus, and the pons. Their size varies from that

of a small walnut to a goose-egg. A limiting membrane may or may not exist.

The contents of the abscess consist of a creamy fluid, which is usually of a greenish hue, and inodorous except when pyæmic. It is composed of pus corpuscles, the débris of broken down brain-tissue, fatty matter, crystals, and salts. The periphery of the abscess is often surrounded by a zone of yellow softening (Rokitansky). Sometimes a well-marked encapsulation is detected, due to a fibrinous wall which may reach a quarter of an inch in thickness.

Embolic abscesses are multiple, as a rule. They are usually of small size.

Abscesses of the brain sometimes rupture into the ventricles—more rarely into the tympanum, the nostril, or the cavity of the orbit. They often create a complicating meningitis by pointing toward the convexity of the cerebral hemispheres. After an extensive meningitis, the pus has been known to perforate the calvaria.

The reaction of the fluid contents may be alkaline or acid. If mucine be present, the pus may appear gelatinous and ropy. If the process of absorption takes place the contents of the cavity may disappear, and be followed by a retraction of the wall of the cyst and the formation of calcareous deposits or of cheesy masses.

The pressure created by the accumulation of pus tends to distort and flatten neighboring convolutions, and to interfere more or less with their nutrition.

Etiology.—Among the most frequent causes of cerebral abscess the following may be mentioned: Suppuration of the middle ear; blows received upon the convexity of the skull; erysipelas of the face or scalp; suppuration of the orbit or nasal fossa; caries or necrosis of the skull; cerebritis; embolism; pyæmia; glanders; some of the eruptive fevers; and other blood-poisons.

Symptoms.—These will be modified (1) by the seat and extent of the abscess; (2) by the causes which induced it; and (3) by the complications which are developed. The presence of rigors, with paroxysms of fever at irregular intervals, should lead us to suspect suppuration when in doubt. The fact that abscess has been known to exist for many months in some parts of the cerebrum without inducing any symptoms, should be remembered by those who are inclined to be hasty in their clinical deductions. I would again impress upon the mind of the reader in this connection the necessity of a familiarity with the deductions embodied in the first two sections of this work, as a basis for all clinical deductions respecting the localization of cerebral lesions during life.

Differential Diagnosis.—The symptoms of cerebral abscess are apt to be confounded with those of cerebral tumor and softening.

From *cerebral tumor*, the rapid emaciation and short duration of life, the presence of rigors and paroxysms of fever, the history of the case, the infrequency of paralysis of special cranial nerves, and the possible escape of pus from the ear, nose, or orbit, would make the diagnosis of abscess possible in many cases.

From *cerebral softening* the presence of intra-cranial pain, rigors, and paroxysms of fever, a healthy state of the superficial blood-vessels, and other symptoms mentioned above would point toward the diagnosis of abscess.

The table on the opposite page may prove of assistance to the reader in making a diagnosis.

Prognosis.—If suppuration occur in connection with acute encephalitis, death may follow rapidly (from three days to three weeks).

Chronic abscess of the brain may exist for years. If it excites complications of a serious kind, such as diffuse meningitis, thrombosis of the sinuses, œdema, extensive softening, effusion into the ventricles, pressure upon vital centres, pulmonary congestion, etc., death may be indirectly produced by it.

Treatment.—Trephining for well-defined collections of pus within or upon the brain has lately assumed a prominent place among the modern surgical procedures. The recent discoveries made respecting the cortical centres of the cerebrum enable us to interpret the clinical evidences of circumscribed pressure upon distinct areas of the brain, and to take steps for their relief which were not dreamed of until within the last decade. It is hoped that the first two sections of this work will aid the reader to take such a step when necessary with confidence and judgment.

SCLEROSIS OF THE BRAIN.

The term "sclerosis" is used to designate a condition characterized by an *increase in the connective tissue of an organ.*

This newly-formed connective tissue subsequently contracts, and induces atrophy of those parts which are thus subjected to pressure, because the blood-supply is gradually diminished.

Morbid Anatomy.—In the nerve-centres, this condition may assume different forms: 1. It may constitute the so-called "general" or "diffuse sclerosis;" seldom involving the brain, but not infrequently affecting large tracts of the spinal cord. 2. It may be disseminated throughout the brain and spinal cord, constituting the "sclérose en plaques" of the French authors, or "multiple sclerosis" of English and American writers 3. A variety of the second form, termed "miliary sclerosis," has also been described.

Sclerosis of the brain probably starts as a *chronic congestion*, which

DIAGNOSTIC SYMPTOMS.	IN CEREBRAL SOFTENING.	IN CEREBRAL TUMOR.	IN CEREBRAL ABSCESS.
HEADACHE	Usually frontal. Generally continuous. May be absent throughout the disease.	Seat variable, but pain is seldom absent. Is most severe when the cerebellum is diseased. Is apt to exhibit distinct paroxysms. Is caused by tension of the meninges.	May be absent, but is usually very well marked and localized.
VERTIGO	May be absent.	Is particularly apt to occur when the cerebellum or temporal lobe is affected.	May be absent.
MENTAL IMPAIRMENT	Apt to be a marked symptom. Develops gradually (as a rule).	Often absent.	Develops rapidly if at all.
SENSORY PHENOMENA	May be normal.	The sensory conducting tracts are sometimes very markedly disturbed.	If sensation is disturbed, anæsthesia of a unilateral type may be detected. Sensation may be unaffected.
MOTOR PHENOMENA	Paralysis of motion may occur suddenly or progressively. It is sometimes not developed.	Motility is not infrequently affected by the growth of the lesion.	Paralysis of motion may or may not be developed.
SPECIAL SENSES	May be normal.	Liable to be progressively affected. "Choked disk" is pathognomonic.	May escape impairment if the abscess be of moderate size and circumscribed.
RIGORS	Seldom observed.	Generally absent.	A strong diagnostic symptom.
EMACIATION	Progressive or wanting.	Progressive or absent.	Rapid, as a rule.
COURSE OF THE DISEASE.	Chronic.	Usually chronic.	Rapidly fatal, as a rule.
TEMPERATURE	Normal.	Usually normal.	Markedly elevated.
CLINICAL HISTORY	Cause may be obscure.	If non-syphilitic, the history will be of a negative kind.	The history will usually point to an exciting cause of the symptoms.
SPEECH	Is apt to be impaired.	Usually normal.	May be impaired.

leads to an exudation of an albuminous fluid, and subsequently to cell-proliferation in the neuroglia.

It is closely allied to inflammatory processes, if not strictly dependent upon them.

Injuries to the convolutions of the so-called "motor area," or a severance of the "motor tracts" of the brain (complete or partial) seem to act as an exciting cause of a so-called "descending sclerosis" which confines itself to the tract of fibres that are functionally associated with the parts injured. In this way it eventually reaches the spinal cord. Similar changes may involve the cranial nerves, chiefly the optic.

In chronic insanity, sclerosis of the brain is not infrequently detected; and the same may be said of general paralysis, some cases of epilepsy, Duchenne's malady, paresis, paralytic tremor, and idiocy.

The diffused or general variety is described by some authors under the term "*induration of the brain.*" This is because an abnormal hardness of the cerebral tissue can be detected, and the brain cuts with greater resistance than when healthy.

In all forms of cerebral sclerosis, the medullary substance is most often involved.

Sections of a sclerotic patch reveal to the eye a moistened surface, more or less transparency, and small bluish or reddish spots. The microscope enables us to detect newly-formed cells in abundance, an excess of connective tissue, atrophy of the nerve-fibres, fatty granules, scattered amyloid corpuscles, and a thickening of the coats of the cerebral capillaries.

Etiology.—Little is definitely known regarding the causes of cerebral sclerosis. It is supposed to accompany chronic cerebral congestion and morbid changes in the walls of the capillary vessels. It may be developed in connection with epilepsy, insanity, idiocy, bulbar paralysis, tremor, destructive lesions of the brain, and old age. Diffused cerebral sclerosis is most commonly observed during infancy.

Symptoms.—The variety of sclerosis which exists will tend to modify the symptomatology of this disease.

The *diffused form* occurs, as a rule, during infancy. It is usually accompanied by imperfections in development of both mind and body, and by paralysis, epileptic convulsions, and post-paralytic contractures. The imperfections in development are generally most marked upon one side. One leg or arm will occasionally fail to grow, or it may grow slower than its fellow.

The mental faculties exhibit an abnormal dullness (approaching to idiocy). Articulate speech is acquired imperfectly and late; again, it may never be developed. In some recorded instances, where the disease

became manifest after the child had learned to talk, serious imperfections of utterance were occasioned by it.

Later in the disease, the limbs begin to show symptoms of paralysis. Still later, contractures of the paralyzed muscles occur, and produce deformities of the limbs. All intuitive or acquired sense of cleanliness of habit, or attention even to the requirements of Nature, seems to be lost or wanting in many of these subjects. The urine and fæces are passed not infrequently in the clothing or bed.

The sensibility of the limbs is apt to be impaired at the time when paralysis of motion is developed. It is most marked upon one side, as a rule.

The so-called "*multiple sclerosis*" seldom occurs except in the adult or the aged. The period of onset is frequently marked by the development of numbness, hyperæsthesia, dysæsthesia, and other sensory disturbances, in one or more of the extremities, for many months before the characteristic trembling appears. Sometimes these patients complain only of shooting pains, which are paroxysmal and of short duration, resembling an electric shock. Again, an epileptic attack may occur. In one of my patients the disease began with a slight attack of facial paralysis, which followed a heated discussion over business affairs with his partners. In another, attacks of "petit mal" were first observed. These were followed, after the lapse of a few years, by tremor of an aggravated type.

The *most characteristic symptom of this disease is tremor.* It develops gradually, as a rule. At first, the patient may notice only a slight tendency of some limited part of the body to rhythmical twitching. It may be so slight in the beginning as to attract the attention of the patient only when quiet and unoccupied; as, for example, during the moments preceding sleep. I have known it to be limited to a single muscle of the leg or arm for many months. One of the most typical cases which I have ever encountered observed it for over a year in the exterior part of the thigh.

Gradually these convulsive and involuntary twitchings become more aggravated, and are diffused over a larger area.

If the hand be affected, its movements become uncertain. Such patients often become afraid to dine with strangers, on account of their liability to accident during the meal. They cannot write as well as before the trembling commenced, and are forced after a while to discontinue all attempts to carry on correspondence or book-keeping. The constant movements of the extremity render them the objects of painful scrutiny when out-of-doors or among strangers. Dressing and undressing become extremely difficult. One of my patients requires the aid of a body servant for that reason alone. Finally, dynamographic traeings

will show an inability to maintain continuous contraction of the muscles.

When the legs become attacked the gait closely resembles that of paralysis agitans (page 163). The knee and foot are apt to move involuntarily when the patient is sitting or reclining. These subjects frequently acquire the habit of constantly changing the position of the affected limb when not walking, because it seems to produce a temporary respite from trembling. During sleep, the shaking ceases for a greater or less period of time; but, finally, the movements continue day and night. Excitement of any kind and muscular effort invariably causes the spasmodic movements to become markedly intensified.

Occasionally, the eyes and face become affected with this disordered condition of motility. The tongue may also participate in the tremor.

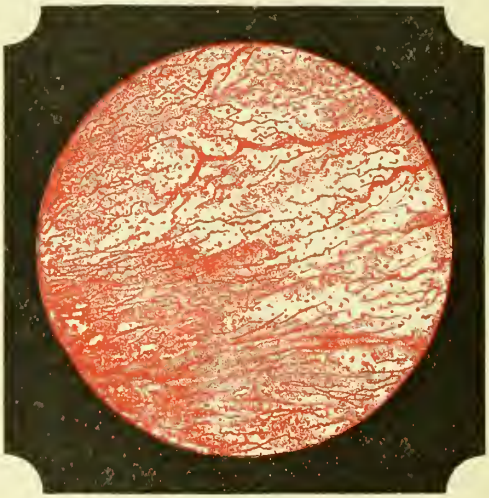


FIG. 88.—CEREBRAL SCLEROSIS. (After Fox)

When symptoms of ataxia are induced, we have reason to believe that the *fillet-tract of the brain*, or the *posterior columns of the spinal cord* are involved. I once saw a case where the patient could not respond to any of the tests for coördinated movements of one arm (page 180), but could use the opposite arm and the lower limbs perfectly.

Paralysis of motion or sensation is apt to follow the development of tremor. The limbs are not usually completely paralyzed. They are more or less paretic.

The sense of touch and the conscious appreciation of pain, temperature, pressure and the muscular sense may be disturbed to a greater or less degree.

The *special senses* may be impaired late in the disease. Smell, sight, taste, and hearing have been known to be individually destroyed.

The condition known as the "choked disk" (page 322) may be detected by the ophthalmoscope in some cases.

Finally, these patients gradually become unable to perform any mental or physical effort. They give evidences of mental decay by their inability to retain urine or fæces, a loss of memory and of mental control, dementia, a disregard of surroundings, etc. Death may occur from general convulsions, exhaustion, coma, or some complicating disease. The duration of this disease may vary between the limits of a few months to ten years. It seldom exceeds five or six years.

Differential Diagnosis.—This condition may be confounded with chorea, paralysis agitans, the tremor which follows cerebral hemorrhage, and spinal sclerosis.

Chorea is occasionally developed in adults. It is never associated, however, with the head-symptoms of sclerosis due to cerebral congestion, such as vertigo, pain in the head, etc. The peculiar gait of cerebral sclerosis is never encountered in chorea. The spasmodic movements of chorea are irregular, and differ markedly from the rhythmical movements of tremor. Impairment of sensation or motility is not developed in connection with chorea. Feebleness of intellect, when present, appears during the early stages of chorea.

On the other hand, the diffused variety of cerebral sclerosis, although confined almost exclusively to children, is characterized by symptoms of idiocy, convulsions, hemiplegia, etc. It could, therefore, hardly be confounded with chorea.

Paralysis agitans is not accompanied by evidences of impairment of sensibility or motility in the limbs; nor are the "head-symptoms" of cerebral sclerosis developed whenever the tremor is due to a purely functional disturbance. Patients with functional paralysis agitans can usually cause the dynamograph to exhibit a tracing of continuous muscular contraction. This is impossible in subjects affected with multiple cerebral sclerosis.

To my mind, however, many cases of sclerosis are diagnosed as paralysis agitans. It is easy to understand why this should be so. Both are liable to exist after middle life; both cause marked tremor; and the gait of the two diseases are nearly identical.

Post-paralytic tremor is to be diagnosed by the history of the case. Unlike that of cerebral sclerosis, the tremor follows the development of paralysis. Post-paralytic tremor is more liable to follow lesions of the internal capsule of the cerebrum than of the cortex; hence the history will point probably to a simultaneous impairment of sensation as well as of motion in one lateral half of the body at the time of the attack, and possibly, also, to a disturbance of the function of smell, sight, hearing, or taste. The ophthalmoscope may reveal the condition

known as a "choked disk." This has been described on a previous page.

Spinal sclerosis can be excluded by the history of the case. Of course, all "head-symptoms" would be absent. The evidences of mental decay would not be developed. The cranial nerves would escape. The evidences of impairment of sensibility or motility would be irregularly manifested in the limbs, in accord with the seat and extent of the sclerotic patches.

Prognosis.—The diffused variety, when occurring in infants, is almost invariably fatal. When it is developed in the adult the symptoms may sometimes be ameliorated by judicious treatment, and I doubt if a cure was ever complete.

The multiple variety perhaps offers a little more hope of cure. Still, it must be confessed, I think, by all who have experimented in these cases, that a very large proportion grow steadily worse and die in spite of all remedial measures. One thing is certain to my mind, viz., that all benefit which can be expected must be derived from active treatment in its early stages. Hence a prompt diagnosis is essential if a hope of relief is to be extended to the patient with any prospect of its realization.

Treatment.—Among the remedial agents which have been suggested for this disease, may be mentioned the chloride of barium combined with hyoscyamus (Hammond), the bichloride of mercury (Mitchell), the phosphate of zinc, the chloride of iron, the nitrate of silver, strychnia, cod-liver oil, and electricity. I would add to these the internal administration of hot water and the removal of all recognized sources of reflex irritation.

Hammond claims that great benefit may be derived from the administration of a *grain of the barium salt* three times a day, when employed in conjunction with the *tincture of hyoscyamus* in doses of one or two drachms three times a day. His suggestion that the chloride of barium be fresh and properly prepared seems to me particularly well taken. It is an unstable salt, and is difficult to obtain. If not a reliable preparation, it is inert.

I have found that it acts favorably (as does also the nitrate of silver and strychnia) upon tremor. It also tends to diminish disorders of sensation and motility in some cases. On account of these effects, it is indicated in cerebral sclerosis.

The *mercurial treatment*, in the form of baths, hypodermic injection (page 290), the bichloride, etc., is indicated when a syphilitic history can be obtained. I do not believe that small doses of mercurial salts arrest the new connective-tissue formation of nerve-centres.

Strychnia, *nitrate of silver*, *iron*, and *cod-liver oil* may be employed either as substitutes for the chloride of barium or as tonics. The first

two have a decided anti-spasmodic action. A moderate quantity of alcoholic stimulants with meals, and exercise in the open air (if tempered with judgment) are often beneficial, and should be employed as adjuncts to the other methods of treatment mentioned.

Electricity is employed by me in all cases where tremor exists. It is my custom to apply the galvanic current (derived from five or ten milliamperes of current to the head, passing it from the forehead to the occiput. Hammond recommends the stimulation of the main sympathetic cords. This can be best accomplished by placing the poles at the neck. I am in the habit also of using from ten to twenty milliamperes upon the tremulous muscles, one pole being placed at the nape of the neck. Further directions relating to the electrical treatment of tremor will be given later.

When paralysis has been developed the faradaic current should be substituted for the galvanic, as a remedial agent to the paralyzed muscles, or the static spark should be administered.

For reasons previously given, I believe that the *internal administration of hot water* (page 248) exerts a marked effect upon all diseases where the sympathetic system is at fault. I should advise that it be tried upon these subjects.

Finally, all mental efforts of a laborious kind should be forbidden. Emotional excitement of any kind should also be carefully guarded against.

CEREBRAL ATROPHY.

This condition may be of two varieties,—the infantile and senile.

Morbid Anatomy.—In the *infantile form*, the characteristic lesions include (1) obliquity of the skull, one lateral half being shrunken and deformed; (2) a premature closure of the sutures; and (3) atrophy of the corresponding cerebral hemisphere, involving its convolutions and basal ganglia.

The atrophic changes may extend to the pedicles of the brain, the pyramids of the medulla, and the columns of the spinal cord.

This form is due chiefly to fœtal apoplexy, encephalitis, hydrocephalus, and physical shocks or violent emotions on the part of the mother during pregnancy.

In the *senile variety*, the atrophic changes may be due to any cause which tends to slowly impair the nutrition of the brain. Among such may be mentioned embolism, thrombosis, hemorrhage, tumors, encephalitis, inflammations of the pia mater, alcoholic, opium- or lead-poisoning, syphilis, and excessive venery (?).

Etiology.—Something has already been said respecting the probable causes of the two varieties. The fact that pathological changes in the meninges are generally found to coexist with cerebral atrophy—

especially in that form which occurs in connection with the so-called "*general paralysis of the insane*"—is worthy of remark as shedding some light upon the causation of the disease in individual cases. Senile marasmus is one of the chief causes of this condition when occurring in the aged. Males are more frequently attacked than females.

Symptoms.—The symptoms of the *infantile variety* vary with the extent of the atrophy. Weakness of intellect, deaf-mutism, abolition of some of the special senses, incomplete paralysis, muscular contractures, and impairment of the sensibility of the paralyzed parts may be present, in addition to the cranial deformity. The bones, muscles, nerves, etc., of the side opposite to the cerebral atrophy may be imperfectly developed. Ptosis and strabismus often occur.

The peculiarities in the appearance of the cranial bones (enumerated in the preceding lines) would naturally suggest the presence of this morbid condition. Not only is the skull misshapen, but the bones are apt to become altered in their relative size and thickness.

The symptoms of the *senile form* include many manifestations of enfeebled mental powers. The memory and intelligence are affected early; apathy and somnolence develop; the power of motion is slowly but gradually lost; tremor makes its appearance; finally, the patients take to their beds and pass into the condition of childishness, accompanied by the symptoms of bulbar paralysis, from which they die. Bed-sores, bronchitis, and acute pulmonary œdema are frequent complications.

Differential Diagnosis.—The senile form might possibly be confounded with cerebral softening and apoplexy. The history of the case and a careful study of the symptoms would rapidly dispel all doubts.

The infantile variety can hardly be confounded with any other condition. The spasmodic attacks and the paralysis which occur, not to speak of the cranial distortion, are sufficient for a diagnosis.

Prognosis.—The infantile variety usually results in death before the fifth year. The senile form is apt to be associated with intercurrent diseases which hastens death, among which may be mentioned various pulmonary complications, bed-sores, and renal or vesical diseases. When the condition exists in connection with the "general paralysis of the insane," the duration of life seldom exceeds one year.

Treatment.—General hygienic measures are indicated in order to increase the physical vigor. Massage may be substituted for active physical exercise, when the patient is unable to walk with ease. Galvanism is sometimes of benefit. The bowels and bladder should be looked after if the patient be old. I do not believe in the curative effects of the iodides, calabar bean, cold douches, tepid baths, etc., although they have been recommended.

CEREBRAL HYPERTROPHY.

This disease is comparatively rare. We owe most of our knowledge concerning it to Virchow, who published the results of autopsies and deductions concerning its pathological changes. It is not a true hypertrophy, because the connective-tissue elements are alone increased.

Morbid Anatomy.—The cranial bones are abnormally thin, and the brain protrudes when the calvaria is removed. An enormous increase in the circumference of the head is present when the disease attacks a child, and the sutures fail to close. In these respects it resembles chronic hydrocephalus. The brain is abnormally heavy, and is rendered abnormally tough and elastic. It is very anæmic; the ventricles are found to be empty; the membranes are dry and attenuated; the convolutions are compressed and flattened; and the dura may be firmly adherent to the skull. The cerebral hemispheres are more often attacked than the basal ganglia, the pons, the cerebellum, or the medulla.

Etiology.—When not congenital, it usually attacks a child before the third year. It seems to be hereditary in some instances. It accompanies idiocy, and may follow plumbism, traumatism, rickets, alcoholism, and epilepsy. It sometimes occurs in adults. Dwarfs seem to be particularly predisposed to its development. It may accompany insanity.

Symptoms.—When present in the child, the head is apt to lean toward one side, and a tottering gait or tremor may be developed. Sometimes there are convulsions, strabismus, feebleness of mind or idiocy, somnolence, protrusion of the tongue, and severe headache. The thymus gland may become enlarged and cause spasm of the larynx. In the final stage coma develops. It is generally preceded by dilatation of the pupils, vomiting, convulsions, and a slow pulse.

When adults are attacked, vomiting, dyspnœa, and difficulty in swallowing are developed in connection with delirium, vertigo, epileptiform attacks, headache, and abnormalities of the heart's action.

Differential Diagnosis.—It is impossible, in some cases, to distinguish between this disease in a child and chronic hydrocephalus. The presence of a cerebral souffle, pulsation of the fontanelles, and a previous precocity of the child point to cerebral hypertrophy.

Prognosis.—It is always fatal. In the child progressive stupor develops. In adults, complications are more liable to shorten the course of the disease.

TUMORS OF THE BRAIN AND ITS ENVELOPES.

The various forms of new growths which may be encountered in the brain have been enumerated in a tabulated form on page 218. All of the

attempts which have been made to classify tumors of the nervous system, from that of Jaccoud to the present time, are more or less illogical. Every classification must be open to some objection, but attempts of that kind unquestionably serve to assist memory and to systematize description.

We have already touched upon aneurisms as one of the lesions of the vascular apparatus. Parasites of the brain (which are enumerated by Jaccoud) are discarded by Fox, because they can hardly be said to constitute a tumor. Exostoses should be discussed among the tumors of bone, rather than in this connection, although they may develop in the brain and its coverings.

Morbid Anatomy.—Among the entire list of cerebral neoplasms, *gummata* (*syphilitic tumors*) possess more clinical interest than any of the others. This is because they are more frequent than the rest, and also because the prognosis is favorable,—often after the most severe effects to the brain are manifested.

1. We owe much of our knowledge of *syphilitic tumors* to Broadbent, who has studied their effects upon the nerve-centres. They start from the membranes or attack the surface of the brain directly. They are strictly localized and grow slowly. They usually affect only small portions of the organ. Gradually they tend to induce adhesions of the membranes, both to each other and to the brain itself; and, by pressure, they cause local softening of the brain-substance. The effects of pressure upon the cranial nerves which lie adjacent to these tumors are apt to be also exhibited early, and thus the diagnostician is enabled to locate the tumor. This statement applies, however, with equal force to all tumors of the brain.

Gummata appear as reddish-gray, jelly-like masses, which are infiltrated, as it were, into the brain-tissue.

2. Perhaps the most common form of cerebral tumor is *tubercle*. The pathology of this form of deposit has been discussed already at some length in the pages devoted to tuberculous meningitis. It tends, as a rule, to invade several regions at the same time,—when deposited within the substance of the brain. Tubercular masses in the aggregated form may be of different sizes. They vary from that of a small pea to the dimensions of a cherry, or even of a small hen's egg. They are sometimes encapsulated, being separated entirely from the cerebral substance. Again, the line of demarkation of the deposit may be indistinctly defined from that of the gray or white matter. Caseous degeneration is not infrequently encountered in the central portions of these tubercular masses. Similar deposits are apt to be found simultaneously in other viscera and tissues. Tubercle of the brain, when softened, might easily be mistaken for a gummatous deposit. The examination of the viscera, the history

of the case, and a microscopical examination, would dispel any such elements of doubt.

3. The brain or its envelopes may become the seat of *carcinoma* or *cancerous growths*. Scirrhus and encephaloid are the more common types. Occasionally the melanotic variety is detected. Old age seems to be a factor in the development of cancer of the brain in most cases. It may start either in the brain-substance, the meninges (chiefly in the pia), or in the bony skull-cap.

The size and rapidity of progress of carcinomata of this region depends upon the type. The encephaloid variety grows rapidly, is very vascular, and may attain an immense size. The scirrhous type is of slower growth, and is less vascular.

4. Regarding other varieties of cerebral tumors which have been enumerated in the preceding table (page 218), it does not seem to me

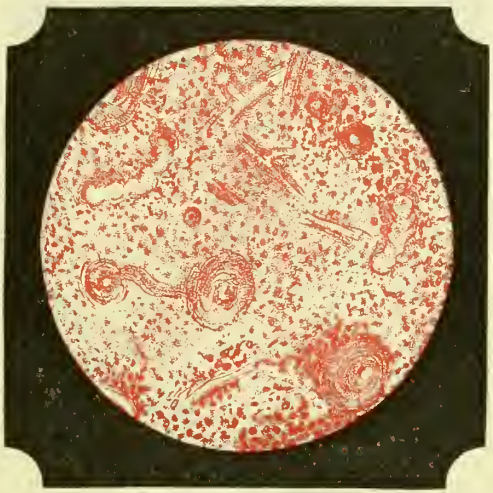


FIG. 89.—CEREBRAL GLIOMA.

necessary to describe the microscopical appearances of each, because most of the works on pathology afford all necessary information respecting them. It may be well, however, to mention a few facts pertaining to each which possess a clinical value.

Cysts of the brain are generally the result of an old apoplectic clot which has undergone certain morbid changes already described.

Glioma are formed of connective-tissue elements of the brain; hence they are usually developed in the substance of the organ.

Epithelial growths may spring from the cerebral vessels or the walls of the ventricles, as well as from the cells of the pia and arachnoid. This statement does not comprise those epithelial tumors which are properly classed as cancerous.

Psammoma or *sand-tumors* consist of granules of carbonate of lime held in a matrix of connective-tissue, in whose meshes concentric layers of epithelial cells are also found. (Virchow.) They are encountered chiefly in the dura (particularly in that part which covers the parietal lobe) and in the choroid plexus of the fourth ventricle.

Cholesteatomata, or *pearl-tumors*, are composed of cholesterine and stearine. They are destitute of vessels. Their size is, as a rule, small. They are known to arise from the bones, meninges, and in the brain itself.

Tumors of the osseous type spring, as a rule, from the calvaria. In rare instances, however, they do not do so. The falx is sometimes more or less osseous. True ossific deposits have been encountered within the brain-substance. I lately discovered one in the brain of an epileptic patient.

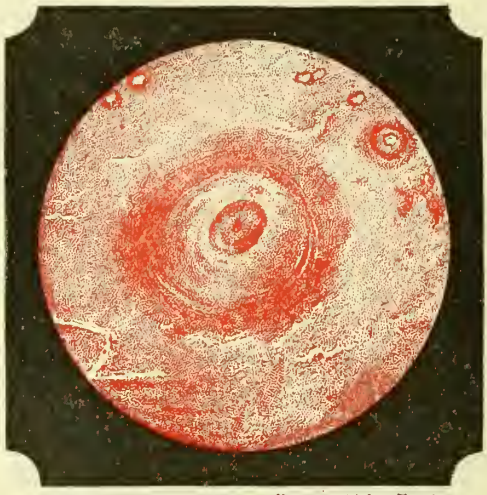


FIG. 90.—SYPHILIS OF THE BRAIN. (After Fox.)

Fibrous, *fibro-plastic*, and *fatty growths* within the skull are to be classed as accidental neoplasms. The fibro-plastic variety has been known to attain the size of an orange. (Hammond.) The consistence of these tumors is subject to extreme variations. Fibrous tumors are rare, but they may arise from the ependyma of the ventricles. Lebert reports seventeen to have developed simultaneously within the lateral ventricle.

Hydatids (when present) are generally detected within the cerebral hemispheres. The parasite (*echinococcus*) is enveloped in a cyst, which may be as large as an orange. The number of such cysts is usually small. They are often solitary.

Another form of *parasitic tumor* (due to the *cysticercus*) may be encountered within the brain-substance. The surrounding cyst is usually wanting in this variety. They lie near to the surface of the brain, as a

rule. Sometimes they are found in the ventricles and pia. They are seldom solitary. Cruveilhier reports a case where over one hundred were found.

Vascular tumors of the miliary type have been discussed in connection with the morbid anatomy of cerebral hemorrhage. It may be well, however, to mention the statistics of Gouguenheim (as quoted by Hammond) respecting the relative frequency of aneurismal dilatations of the more important vessels. This author found that out of 69 cases, seventeen affected the basilar; fourteen attacked the middle carotid; twelve involved the internal carotid; eight, the anterior cerebral; five, the posterior, or communicating; four, the cerebellar; three, the posterior cerebral; and two, the anterior communicating and the middle meningeal.

M. P. Jacobi has lately contributed a classical article on the subject of cerebral tumors,* a study of which will well repay the reader.

Etiology.—Much that has been said already respecting the morbid anatomy of cerebral tumors relates to their causation as well.

Large *aneurismal tumors* usually occur after the age of fifty, and are preceded by textural changes in the arterial walls (atheroma, fatty degeneration, etc.). In such cases, the exciting causes of the aneurism may include all forms of traumatism to the head or body, cardiac hypertrophy, prolonged anxiety or emotional excitement, excessive mental labor, embolism, etc.

Cancerous growths may occur at any age, but they are most commonly encountered in male adults. It may be excited by traumatism (according to Hammond).

Tubercular deposits within the skull are most frequently encountered in the young. They may occasionally be developed during adult life, as a sequel of similar deposits within the lungs or other viscera, and after infection from caseous masses in the viscera, joints, glands, etc.

Gummata are invariably due to syphilitic infection. They are developed late in the disease, as a rule.

Parasitic tumors are due to the transportation of the embryos from some distant part.

Symptoms.—It must be evident to the reader that the symptoms of a cerebral tumor necessarily depend upon three factors, viz., its seat, its rapidity of growth, and the amount of pressure or injury created by it upon the cortical cells or nerve-tracts which lie in close proximity to it. I would refer the reader, therefore, to a preceding section which treats of the localization of lesions of special areas of the cortex and the more important component parts of the cerebral architecture; as well as to those pages also which deal with cerebral thermometry, the use of the ophthalmoscope, and the symptomatology of cerebral hemorrhage.

* Reference Handbook of Medical Sciences, vol. i. p. 668.

It should be remembered, however, that the slow growth of a cerebral tumor does not allow of pressure-effects upon neighboring parts as rapidly as would an apoplectic extravasation, although in time it might create even more serious damage.

Again, the presence of a tumor of the meninges or bone might at first cause symptoms of irritation of the cortex (Jacksonian epilepsy, frequent convulsions, etc.). Later on, other symptoms, which would be apt to appear, would indicate a destruction of the functions of parts which at first were only irritated by the slight pressure upon them exerted from the growth of the neoplasm.

Third, the vascularity of the tumor and the activity of the circulation required to insure its development would be apt to create modifications in the temperature of the scalp over the seat of the tumor, provided it was superficially situated.

Fourth, we would be apt to notice that the steady increase in size of the growth would progressively impair one or more of the cranial nerves, and possibly some of the tracts of nerve-fibres which help to form the cerebral hemispheres.

Fifth, tumors of the brain, and in fact any lesion which tends slowly to increase intra-cranial pressure, tend to manifest their existence by development of a double optic neuritis,—the so-called "*choked disk*" (Fig. 87).

This condition is only apparent when the ophthalmoscope is employed, but it possesses a decided clinical value. I have described the condition in detail in a brochure, from which I quote as follows:—

"When the radiating fibres of the internal capsule are involved in a lesion which creates a gradually-increasing pressure (as in the case of tumors which grow slowly), the *fundus of the eye* exhibits morbid changes in the region of entrance of the optic nerve which are of value in diagnosis. The condition so produced is commonly known as the 'choked disk.' It is nearly always bilateral, but often most marked in one eye. It may be considered as one of the most positive signs of an extensive intra-cerebral lesion, and especially of tumors of the brain. When the eye is examined with an ophthalmoscope, this condition is characterized by a swollen appearance of the optic nerve, which projects appreciably above the level of the surrounding retina; the margin of the disk is either obscured or entirely lost; the arteries appear small, and the veins large and tortuous; finally, small hemorrhagic spots may often be detected in the retina near the margins of the disk. In spite of this condition, the power of vision may be little impaired; so that the existence of 'choked disk' may be unsuspected unless the ophthalmoscope be used before the diagnosis is considered final. After a number of weeks, and very much longer if a tumor is the exciting cause of the condition, the

appearance of the disk changes. An unnatural bluish-white color, which denotes atrophic changes, develops; the outline of the disk becomes sharply defined; the retinal vessels become small, and vision becomes markedly interfered with."

Differential Diagnosis.—The chief points by which the existence of a cerebral tumor is indicated are as follow: (1) Severe and persistent pain of a more or less localized type; (2) the occurrence of convulsive attacks, which may or may not be followed by transient paralysis and unconsciousness; (3) the retinal changes of optic neuritis; (4) the presence of monoplegia or the paralysis of some (one or more) of the cranial nerves; (5) the history of the case when syphilis, tuberculosis, or the development of parasitic cysts are suspected.

Since *cerebral softening* often accompanies the development of a cerebral tumor, it is possible that all the symptoms of that condition (except the choked disk) may exist and render the diagnosis more or less obscure.

From *epilepsy* the diagnosis is to be made by the presence of intracranial pain; by the age and history of the patient; by the fact that the convulsion is apt to be more or less unilateral, and to start in that particular part of the body whose motor centres are subjected to the greatest amount of irritation; by the presence of consciousness during the convulsive attack, or of incomplete coma; by occasional development of transient paralysis after the fit; and by the fact that the mental powers of the patient are seldom weakened.

From the *multitude of causes* that may induce *hemiplegia*, *monoplegia*, or *paralysis of some cranial nerve*, the diagnosis must be made by exclusion, after the facts revealed by a clinical examination of the patient (according to Section II) have been thoroughly and accurately determined.

One of the most remarkable cases of supposed hysteria that ever came under my observation was found after death to be due to a widely diffused form of enchondroma of the falx cerebri.

Prognosis.—Unless of the syphilitic type, cerebral tumors are liable to cause death in spite of treatment. Lately, cases of successful removal of a cerebral neoplasm have been reported. In the light of modern cerebral localization it is possible that surgical measures may occasionally be employed with benefit to the patient when the tumor is non-malignant and superficially situated.

Gummata are unquestionably absorbed in many cases when the iodides are pushed to extreme limits.

Treatment.—The remarkable successes in treatment of *syphilitic tumors* of the brain or its envelopes, which are frequently reported, should lead us to carefully investigate the question of syphilitic infec-

tion in every case. I have personally witnessed in four of my own patients a perfect cure, when the prognosis appeared to be extremely grave. In one, hemiplegia and homonymous hemianopsia existed; in another, aphasia, strabismus, and extensive paresis had been produced; in a third, almost total blindness existed when the patient was first seen by me; in the fourth, complete coma suddenly occurred after an attack of ocular paralysis, which was accompanied by very marked mental disturbance. Another of my patients had several convulsions within a week, and is to-day in perfect health.

As early as 1872, I began experiments with enormous doses of the iodide of potash and the iodide of calcium in these cases. To one patient, seen by Prof. A. L. Loomis with me in consultation, I gave an ounce of the former salt daily for three days. I have frequently given half of that amount daily for a week at a time, or until the effects of iodism became very marked. With some patients, who bear the potash salt badly, I employ the calcium preparation in about the same doses, as I have found that it creates less gastric disturbance. The objection to it is that the salt decomposes easily and free iodine is liberated, causing the solution to become of a brown color. Care should therefore be exercised in using the pure drug and in exposing a solution of it to light.

My experience is strongly in favor of combining mercurialization with the internal administration of the iodides in the tertiary as well as the secondary stages of syphilis. I prefer the mercurial bath and the hypodermic injection method to the administration by the stomach, when it is practicable to employ them.

When the tumor is of the *aneurismal type* the use of the iodide of potash in moderate doses seems to exert a curative influence in some cases. It is well to keep such patients in a recumbent position, and to avoid all excitement and other possible causes of an excessive action of the heart.

Regarding the treatment of the other varieties of tumors mentioned as liable to exist within the skull, I can offer but little encouragement. If it be possible, in any given case, to decide positively respecting the character and size of a tumor as well as its exact seat, and if none of the contra-indications to trephining existed, I might be tempted to employ the trephine. So far, however, I have never encountered a case that justified such a measure in my opinion.

A SUMMARY OF THE SYMPTOMS OF CEREBRAL DISEASES.

As an aid to the reader, I have deemed it wise to add to this chapter a differential table of the more common symptoms of cerebral diseases. Tables of this kind are particularly of service to the busy practitioner in reviewing the prominent features of the separate diseases which have been previously described.

A TABLE DESIGNED TO SHOW THE DIAGNOSTIC VALUE OF CERTAIN SYMPTOMS IN BRAIN-CONDITIONS.

	ADVENT.	COMA.	MOTOR PARALYSIS.	SENSORY PARALYSIS.	CONVULSIONS.	MUSCULAR CONTRACTURE	ELECTRO-CONTRACTILITY.	CRANIAL PAIN.	DISTURBANCE OF SPECIAL SENSES.	DEFECTS IN SPEECH.	IMPAIRMENT OF INTELLECT.	TREMOR.
CEREBRAL EMBOLISM.	Sudden.	*Incomplete or absent.*	*Hemiplegia.*	Rare.	Infrequent	Absent.	Normal.	Infrcquent	Very uncommon.	*Marked.*	Uncommon.	Absent.
CEREBRAL THROMBOSIS.	Variable.	*Not infrequent.*	Not infrequent.	Depends on seat of clot.	*Very common.*	Absent.	Normal.	Common.	*Ear often suppurates*	Rather infrequent.	Delirium, etc	Usually absent.
CEREBRAL APOPLEXY.	*Sudden.*	*Usual.*	*Seldom absent.*	Not infrequent.	Not infrequent.	*May follow the attack.*	Normal at first.	Not infrequent.	*Some may be impaired.*	May be induced.	May follow attack.	May be developed.
CEREBRAL TUMORS.	Gradual.	Infrequent	May be developed.	May be developed.	Very often occur.	Infrequent	Normal.	Very common.	Common.	May be developed.	Not marked, as a rule.	May be developed.
PACHYMENINGITIS.	Gradual.	May occur.	Not infrequent.	Very infrequent in limbs.	Often occur.	Infrequent	Normal.	Common.	Frequent.	May be developed.	May be developed.	Usually absent.
SIMPLE MENINGITIS.	Moderately sudden.	Not infrequent.	Uncommon.	Infrequent	May occur.	May be developed.	Normal.	Severe.	Common.	Infrequent	Delirium.	Usually absent.
HYDROCEPHALUS (ACUTE)	Gradual.	Late in disease.	Late in disease.	Rather infrequent.	Marked.	Marked.	Normal.	Severe.	Marked.	Infrequent	Delirium, etc.	Absent.
HYDROCEPHALUS (CHRONIC).	Usually congenital.	Rare.	Not infrequent.	Uncommon.	Common.	Frequent (with deformity).	Modified.	Moderate or absent.	Not infrequent.	Common.	Imbecility, etc.	May be developed.
CEREBRAL SCLEROSIS.	Gradual.	Infrequent	May be developed.	May be developed.	May occur.	May be developed.	Often modified.	Not uncommon.	May occur.	May be developed.	May be developed.	*Very common.*
CEREBRAL ANÆMIA (Gen'l variety)	Modified by its exciting cause.	May occur.	Absent.	Infrequent	May occur.	Absent.	Normal.	Common.	Frequent.	May be developed.	Transient or absent.	Absent.
CEREBRAL HYPERÆMIA.	Modified by its exciting cause.	May occur.	Very infrequent.	Infrequent	May occur.	Absent.	Normal.	Common.	Frequent.	May be developed.	May be developed.	Usually absent.
CEREBRAL SOFTENING.	Gradual.	May occur.	Common.	Not infrequent.	May occur.	May occur.	Normal.	Dull.	May occur.	Common.	Usually developed early.	May occur.
CEREBRAL ABSCESS.	Gradual.	Occurs early.	Common.	Not infrequent.	May occur.	May occur.	Normal.	Severe.	Not infrequent.	May occur.	Precedes coma.	Infrequent

The preceding pages of this section will aid the reader in obtaining further information respecting any symptom of disease mentioned in this table.

SECTION IV.

DISEASES OF THE SPINAL CORD AND ITS ENVELOPES.

SECTION IV.

DISEASES OF THE SPINAL CORD.

In the first section of this volume, certain anatomical, physiological, and clinical deductions have been given respecting various component parts of the spinal cord. It is very important that the reader familiarize himself thoroughly with these before he attempts to master the symptomatology of spinal lesions.

One of the first distinctions that must be drawn respecting spinal lesions (in order to make accurate diagnosis) is that which exists between what is known as "systematic" and "non-systematic" or "focal" lesions of the cord.

By "*systematic*" *lesions*, we mean any pathological condition which tends to progress along definitely recognized subdivisions of the spinal cord, without spreading laterally to adjacent parts.

By "*non-systematic*" or "*focal*" *lesions*, we mean any pathological condition which tends to spread laterally, and thus to involve adjacent columns of the cord as the lesion progresses.

For example, a systematic lesion of the anterior horn would remain confined to the anterior horn, irrespective of its extent up or down the cord. On the other hand, a focal lesion starting in the anterior horn might spread to any of the various subdivisions which lie adjacent to the horn, viz., the column of Türck, the crossed pyramidal column, the anterior root-zone, the spinal commissure, etc. (See Fig. 91.)

Clinically, this distinction is very important. A systematic lesion of a motor column would, for example, yield exclusively motor symptoms during the life of the patient, while a focal lesion starting in a motor column might subsequently spread to a sensory column, and thus occasion both motor and sensory symptoms during life.

The table on the next page will give the reader a knowledge of the more important spinal diseases which are to-day clinically recognized.

It must be remembered that the spinal cord, like the brain, consists of two anatomical elements, viz., nerve-cells and nerve-fibres.

The SPINAL FIBRES are connected with the spinal cells in such a way as to allow of a communication (1) between the spinal cells and the periphery of the body (*the spinal nerves*); (2) between the cells of the cord and those of the various gray masses of the brain (*the conducting tracts*); and (3) between the cells of the different spinal segments themselves (the *associating fibres* of the cord).

A TABLE OF THE ABNORMAL STATES OF THE SPINAL CORD.

A. Systematic Lesions of the Motor Portions of the Cord—or of the so-called "*Kinesodic System.*"

- Sclerosis of the Direct Pyramidal Column (usually secondary to a lesion of the brain or cord).
- Sclerosis of the Crossed Pyramidal Columns.
  - *Primary*—Usually bilateral—(*tetanoid paraplegia*).
  - *Secondary*—Usually unilateral—(*descending variety*).
- Inflammation of the Cells of the Anterior Horns of the Spinal Gray Substance (*poliomyelitis anterior*), 3 varieties.
  - Acute.
  - Sub-acute.
  - Chronic.
- Degeneration of the Cells of the Anterior Horns (*progressive muscular atrophy*).
- Progressive Facial Atrophy.
- Pseudo-Hypertrophic Paralysis.
- Amyotrophic Lateral Sclerosis.
- Central Myelitis.

B. Systematic Lesions of the Sensory Parts of the Cord—or of the so-called "*Æsthesodic system.*"

- Primary Bilateral Sclerosis of the Postero-External and Postero-Internal Columns (*locomotor ataxia*).
- Secondary Unilateral or Bilateral Degeneration of the Sensory Columns (*ascending variety*).

C. Non-Systematic or "Focal" Lesions of the Cord.

- Spinal Meningitis
  - Acute leptomeningitis spinalis.
  - Chronic " "
  - External pachymeningitis spinalis.
  - Internal " "
- Spinal Tumors
  - Of the bones.
  - Of the meninges.
  - Of the spinal cord.
- Spinal Hemorrhage
  - *Hæmatomyelia.*
  - *Hæmatorrhachis.*
- Myelitis
  - Acute.
  - Chronic.
- Syringomyelia and Hydromyelia.

D. Functional Diseases of the Cord.

- Spinal Irritation.
- Functional Paraplegia, Thomson's Disease.
  - From Hysteria.
  - " Poisons.
    - Lead.
    - Arsenic.
    - Phosphorus.
    - Ergot.
    - Alcohol.
  - " Anæmia.
- Spinal Neurasthenia.
- Acute Ascending Paralysis.
- Writer's Cramp.
- Tetany.

E. Vascular Changes (of a diffused or circumscribed character).

- Spinal Congestion or Hyperæmia.
- Spinal Anæmia.
- Spinal Embolism.
- Atheroma of the Vessels.
- Fatty or Amyloid Degeneration of Arterial Coats.
- Aneurismal Dilatations.

F. Congenital Abnormalities of the Cord.

- Spina Bifida (with alterations in the cord).
- Absence of Spinal Cord.
- Incomplete Development of Spinal Cord.
- Congenital Cavities of the Cord (Syringomyelia).

Thus we have within the spinal cord the following groups of fibres:—

1. Those which constitute the *anterior* and posterior nerve-roots.*†

* The fibres of the anterior nerve-roots may be said to have the following connections (indirectly, of course, through the multipolar cells of the anterior horn of the corresponding side): 1, with the lateral motor column of the corresponding side of the cord; 2, with the anterior motor column of the corresponding side of the cord; 3, with the anterior motor column of the opposite side of the cord, by means of fibres which decussate in the white commissure.

† A direct continuity of some of the fibres of the posterior nerve-roots in the column of Goll has been asserted to exist by Singer. This observer detected a tract of degeneration in these columns extending to the medulla after section of the posterior roots in dogs.

They pass, of necessity, through the white substance of the cord to reach the spinal gray matter.

2. The *paths of motor and sensory conduction.* These are prolonged to the brain, and probably do not enter directly into the formation of the spinal nerve-roots.

3. The *associating fibres.* These do not extend to the cerebrum; nor do they leave the substance of the cord. They simply join the various spinal segments with each other.

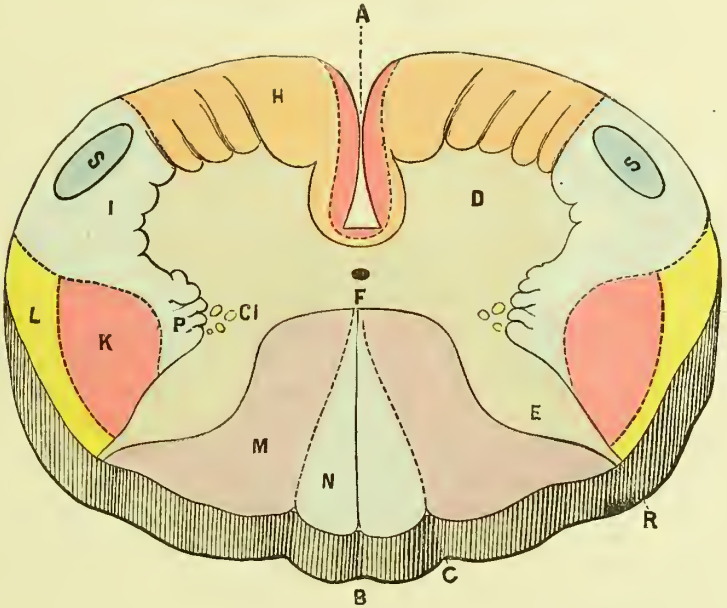


Fig. 91.—Diagram Illustrating the Relations of the Nerve-Fibre Tracts in the Spinal Cord.—The section is supposed to be taken transversely through the lower part of the cervical enlargement (slightly modified from Flechsig): A, Anterior median fissure; B, posterior median fissure; C, intermediate fissure; D, anterior gray cornu; E, posterior gray cornu; F, gray commissure, with central canal; G, uncrossed pyramidal tract (Flechsig), or column of Türck; H, fundamental part of the anterior column (anterior root-zones of Charcot and his pupils); I, anterior part of lateral column; K, crossed pyramidal tract of lateral column; L, direct tract from lateral column to cerebellum; M, column of Burdach, posterior root-zones of Charcot and his pupils; N, column of Goll; S, sensory tract of Gowers. The posterior columns of descriptive anatomy include the fields M and N extending on the surface from B to R. The antero-lateral columns extend on the surface from R to A. Their anterior division includes the fields G and H; their lateral division, the fields K, L, and I. Similar colors are supposed to indicate in this plate a similarity of function.

4. *Trophic and vaso-motor filaments.* These connect the cells of the cord (by means of the spinal nerve-roots) with the blood-vessels and the organs related to motion and sensation.

We can therefore draw the following conclusions, which bear upon diagnosis:—

Interference with the function of the first and second of these

groups of spinal fibres will result in a disturbance (more or less profound) of the patient's capabilities either of motion or of perceiving and recording sensory impressions of various kinds (those of touch, pain, temperature, muscular sense, and electrical stimulation).

Destruction of the third group of fibres will cause symptoms of incoördination of movement.

Impairment of the functions of the fourth group may create abnormalities in the calibre of blood-vessels, and an unhealthy state of the skin, hair, nails, muscles, etc. The effects of spinal lesions upon the pupil (p. 411) are probably attributable to the vaso-motor fibres.

The arrangement of the cells and fibres of the spinal cord are very clearly shown, from a physiological standpoint, in a table which I have prepared (p. 355). It is somewhat similar to one lately published by M. A. Starr.

It is well to know that there are *certain symptoms* which are peculiarly apt to be encountered in connection with spinal diseases. These may be separately discussed with advantage to the reader prior to the description of the separate diseases.

It is also important that a beginner in this field of diagnosis should grasp certain *general axioms* that will materially aid him in discriminating between focal or systematic spinal lesions which may be creating an impairment of the functions of one or more of the groups of fibres just described or the horns of the spinal gray matter.

The following paragraphs and table may possibly shed some light upon the diagnosis of spinal diseases:—

1. Contracture of muscles, when present in a case afflicted with paresis or paralysis, points strongly to a lesion of the motor fibres in the *lateral column of the same side* (the "crossed pyramidal fibres").

2. Exaggeration of the tendon-reflexes is a symptom which points to the same conclusion.

3. Rapid atrophy of muscles (either as an independent affection or as a sequel to paralysis) points to a diseased condition of the cells of the anterior horn of the spinal gray substance. A piece of muscle (when bitten out by means of Duchenne's trocha and subjected to a microscopical examination) will quickly show whether atrophy is occurring as a result simply of disuse or of organic disease of the nervous mechanism.

4. Abnormal sensory phenomena (such for example as pain, hyperæsthesia, anæsthesia, analgesia, formication, numbness, tingling, etc.) point to the existence of a lesion which affects either the posterior nerve-roots or the æsthesodic portions of the cord (p. 93).

5. Diminution or abolition of the reflexes (p. 96) points to lesion which affects a reflex-arc (Fig. 95).

A Table of Some of the More Important Diagnostic Symptoms of Spinal Lesions.

	Part of Spinal Cord Affected.	Onset.
Contracture (tonic shortening of muscle of a persistent type).	(1) Generally due to an implication of the "*crossed-pyramidal fasciculi*" (Fig. 32). (2) May possibly follow (?) implication of the fibres of Türck's column (Fig. 32).	(1) May occur simultaneous with paresis or paralysis (*primary contracture*). (2) May follow paralysis of motion (*post-paralytic contracture*), if the lateral sclerosis is a secondary affection.
Atrophy of Muscles (due to fatty degeneration of the sarcous elements).	(1) Is generally due to a lesion confined to the *cells of the anterior horn.* (2) It may follow a *severance of the motor fibres* which compose the anterior nerve-roots.	(1) *Rapid*, and *preceded by motor paralysis*, if the lesion be an inflammatory or traumatic one. (2) *Slow*, and *not associated with motor paralysis*, if the lesion be of a degenerative kind (progressive muscular atrophy).
Exaggerated Reflexes.	Occurs from implication of the motor bundles of the *lateral column*, as a rule.	When complete paralysis of motion exists in a limb, this test cannot be employed.
Diminution or Abolition of Spinal Reflexes.	(1) Usually occurs with lesions of the *posterior columns* of the cord (locomotor ataxia). (2) Lesion of the *posterior nerve-roots* may also cause this symptom.	(1) Usually occurs *independently* of *motor impairment.* (2) Abnormal sensory phenomena generally coexist with it.
Trophic Disturbances.	Lesions of the *gray substance* of the cord are particularly liable to cause symptoms of this variety.	Are apt to accompany symptoms of vesical or rectal impairment (myelitis).
Abnormal Sensory Phenomena.	(1) May indicate either an *irritative* or a *destructive* lesion of the cord. (2) The *posterior columns* or *posterior nerve-roots* are generally involved. (3) The *posterior horns* of spinal gray substance may be implicated.	(1) May develop *slowly* or *rapidly.* (2) Are often accompanied by *inco-ordination of movement*, or *trophic disturbances*, or *impairment* of the *bladder or rectum.* (3) Spinal reflexes are apt to be *diminished* or *abolished.*

Let us now examine some of the symptoms, which have been already referred to, more in detail.

Motor Paralysis (*of spinal origin*) may assume one of four varieties:

(1) *Hemiplegia*—where one *lateral* half of the body is affected with motor paralysis.

(2) *Paraplegia*—where the *lower half* of the body is affected with motor paralysis.

(3) *Hemi-paraplegia*—where the *lower half* of *one lateral half* of the body is affected with paralysis of motion.

(4) *Paralysis of special nerve-roots* (spinal-nerve paralysis).

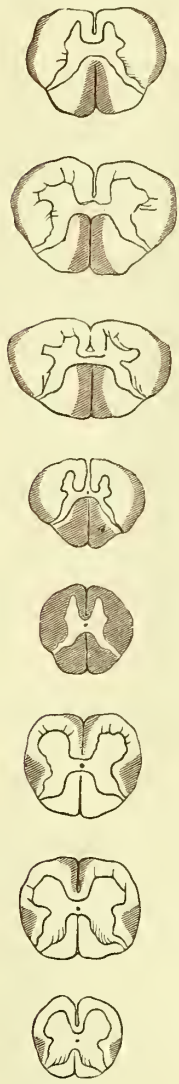


FIG. 92.—A DIAGRAMMATIC REPRESENTATION OF THE SECONDARY EFFECTS OF A LESION OF THE ENTIRE SPINAL CORD AT (5). (After Erb.) Note the *ascending degeneration* of the *sensory tracts* in sections 4, 3, 2 and 1; and the *descending degeneration* in the motor tracts in sections 6, 7 and 8.

The SENSORY PHENOMENA, which may be produced by lesions of the spinal cord, or of the posterior nerve-roots, include the following:—

(1) *Pain*—usually of a peculiar kind (see locomotor ataxia, and the various focal lesions of the cord).

(2) *Hyperæsthesia*, or increased sensibility of parts.

(3) *Numbness*, or a *sense of tingling* (as if "the part were asleep").

(4) *Sense of coldness* or *of heat* in some part of the body.

(5) *Anæsthesia*, or loss of sensibility. It may be complete or partial and be limited to the appreciation of *pain*, *touch*, or *temperature* by the patient.

(6) *Delayed sensation* (see locomotor ataxia).

(7) *Formication*, or a feeling likened to the crawling of ants over the body.

Among the remaining symptoms which are of value in the diagnosis of spinal lesions may be mentioned:—

(1) Incoördination of muscular movements.

(2) *Diminution, abolition, or increase* of the *spinal reflexes* (see Section II of this volume).

(3) *Abnormal electro-muscular reactions* (see Section II).

(4) *Contracture of muscles*—often preceded by stiffness (see lateral spinal sclerosis).

(5) *Atrophy of muscles* (see poliomyelitis and progressive muscular atrophy).

(6) *Vaso-motor phenomena* (see myelitis, ataxia, etc.).

(7) Symptoms which are indicative of *destruction or irritation* of some of the *special physiological centres of the spinal cord* (see focal lesions of the cord).

(8) *Tremor* or some other form of spasmodic movement.

Fig. 67 exhibits in a diagrammatic way several of the above-mentioned abnormal conditions which may coexist as a result of an unilateral lesion of the dorsal segments of the spinal cord. It will serve to aid the reader in mastering the statements made in subsequent pages,—chiefly those which refer to the symptomatology of focal spinal lesions.

The methods which should be followed in *inves-*

tigating each of the above-mentioned symptoms (prior to a diagnosis) have been fully described in Section II., to which the reader is referred.

Before we pass to the consideration of the separate spinal diseases, I would call attention to a carefully prepared summary of the functions of special spinal segments, which differs but slightly from one compiled and tabulated by Starr.* It should be compared with the diagram and table of Gowers (p. 90), as each will explain the other.

A TABLE SHOWING THE ARCHITECTURE AND FUNCTIONS OF THE VARIOUS COMPONENT PARTS OF A SPINAL SEGMENT.

A. THE WHITE MATTER OF THE CORD.	1. "ANTERIOR MEDIAN COLUMN." ("*Türck's column*"—"*direct pyramidal column.*")		*Motor fibres* from the "motor area" of the *cerebral hemisphere of the same side* (figs. 5 and 29).
	2. "ANTERIOR ROOT-ZONE." (*Anterior column.*)		(1) *Fibres of association* between different segments of the spinal cord (vertical in direction). (2) *Motor fibres* passing from the cells of the anterior horn of the spinal gray matter into the anterior nerve-roots (horizontal in direction).
	3. LATERAL COLUMN (consisting of three subdivisions).	(a) *Un-named portion.*	(1) *Associating fibres* between spinal segments. (2) *Fibres of the sensory tract* of Gowers (?). (3) *Vaso-motor fibres* (?).
		(b) "*Crossed pyramidal column.*"	(1) *Motor fibres* from the "motor area" of the *opposite cerebral hemisphere* (vertical in direction). (2) Fibres passing from the cells of the column of Clarke to form the direct cerebellar column (horizontal in direction).
		(c) "*Direct cerebellar column.*"	Fibres passing from the cells of Clarke's column to the cerebellum. The "*vegetative-tract*" *of Starr.*
	4. "POSTERO-LATERAL COLUMN." ("*Column of Burdach*"—"*posterior root-zone*"—"*fasciculus cuneatus*"—postero-external column.)		(1) *Sensory fibres* from posterior nerve-roots to spinal cells (except those associated with the "superficial" or "skin reflexes") (horizontal in direction). (2) *Associating* fibres between spinal segments (vertical in direction). (3) Fibres of conduction of *sensations of touch* and *the muscular sense*, from the arms and neck, upward (vertical in direction).
	5. "POSTERO-MEDIAN COLUMN." ("*Column of Goll*"—"*fasciculus gracilis*"—"postero-internal column.")		Fibres of conduction of *sensations of touch* and the *muscular sense*, from the legs and lower half of the trunk, upward.

* *Am. Jour. Neurol. and Psychiatry*, November, 1884.

A TABLE SHOWING THE ARCHITECTURE AND FUNCTIONS OF THE VARIOUS COMPONENT PARTS OF A SPINAL SEGMENT (*continued*).

B. THE GRAY MATTER OF THE CORD.

Cells of the Anterior Horn.

- a. (1) *Mesial group* of cells. (2) *Lateral groups* in the cervical and lumbar enlargements. — Presiding over flexion and extension of the limbs (possessed by all vertebrates).
- b. (1) *Middle group* of cells. (2) *Central group* of cells in the cervical and lumbar enlargements. — Presiding over movements of the hand and fingers (peculiar to man) and the act of walking erect.
- c. *Motor cells*, whose peculiar functions are not determined.
- d. *Trophic centres* for the *motor nerves* and the *muscles supplied by them.*
- e. *Motor mechanism* necessary to *spinal automatism* and *reflex spinal action.*

Cells of the Central Gray Matter.

- a. *Anterior part.* Trophic centres for the skeleton.
- b. *Posterior part.* (1) Trophic centres for the skin, nails, bladder, joints. (2) Vaso-motor centres.
- c. Automatic centres of a complex nature, and the associating fibres necessary to their peculiar functions (sexual, vesical, rectal, cilio-spinal, etc).

Cells of the Posterior Horns.

- a. *Trophic centres* for sensory conducting-tracts.
- b. *Paths of conduction of sensations of pain, and temperature* from all parts below.
- c. *Clarke's column of cells* (vesicular column) which are apparently associated with the fibres of the "direct cerebellar column."
- d. Posterior group of cells; related to sensations of all kinds.
- e. The *sensory mechanism* necessary to *spinal automatism* and *spinal reflex action.*

The size of the multipolar cells of the anterior horns seems to depend upon two factors: (1) the size of the muscle supplied by the cell, and (2) the length of the nerve-fibre which connects the cell with the muscle (Spitzka).

We are now prepared to discuss the separate lesions enumerated in the table of diseases of the spinal cord. The scattered hints which have been already given in a previous section will possibly help us to grasp the salient features of each, and their physiological interpretation.

In examining a case of paralysis of spinal origin, the following points should be ascertained with great care:—

(1) The *area of distribution* of the paralysis (be it sensory or motor in character).

(2) The *degree of the paralysis;* by separately testing the motor power of different sets of muscles, and also the skin for sensory paralysis by means of the æsthesiometer.

(3) The *state of nutrition* of the paralyzed muscles (see pages which treat of poliomyelitis and progressive muscular atrophy).

(4) The *electrical reactions* of the paralyzed muscles; noting all abnormal formulæ and the intensity of the current required to produce muscular contraction (see section on electricity).

(5) The presence or absence of *rigidity* in the paralyzed muscles (see pages which discuss sclerosis of lateral columns).

(6) The condition of the *superficial* and *deep spinal reflexes* of the two sides. These have been discussed in Section II.

(7) The presence or absence of symptoms of *incoördination of muscular movements* (see locomotor ataxia).

SCLEROSIS OF THE ANTERIOR COLUMNS.

The anterior columns of the cord are frequently called "*the columns of Türck*" and "*the direct pyramidal fasciculi.*"

The first of these names was given in honor of a distinguished pioneer in pathological research relating to spinal lesions. The latter is employed because the bundles which compose these columns pass directly from the hemisphere of the cerebrum to the ultimate spinal segments without decussating in the medulla,—the *anterior pyramids* of which they help to form.

Several diagrams have been introduced in Section I of this volume to illustrate the formation of these columns; as well as their physiological association with the motor bundles of the opposite lateral column of the cord (see Figs. 29 and 32).

Morbid Anatomy.—Sclerosis of these bundles of nerve fibres usually coexists with similar changes in those which compose a part of the lateral column of the cord,—the so-called "crossed pyramidal fasciculi." It may occasionally exist as an independent lesion; but it usually follows the development of some brain or cord lesion and travels downward. It is then unilateral. The symptoms which are peculiarly characteristic of its development are unknown. Its existence is to be inferred when secondary sclerosis of the postero-lateral columns is manifested by symptoms which are clinically well determined. The pathological changes of sclerosis of the cord do not differ from those of that condition elsewhere. They have been described in the preceding chapter, in connection with the brain.

The discovery, made by Flechsig, that the *relative proportion* of the direct and decussating pyramidal fibres *differs in individuals*, helps us to properly interpret those rare cases where a lesion of the cerebral hemisphere has been known to produce a hemiplegia of the same side (instead of the opposed side); as well as those cases of greater frequency where a paresis of the corresponding side coexists with a hemiplegia of the side opposed to the cerebral lesion. In one case in sixty, no decussation of the pyramidal tracts occurs.

SCLEROSIS OF THE MOTOR FIBRES OF THE LATERAL COLUMN.

(*Lateral Spinal Sclerosis—Tetanoid Paraplegia—Spastic Paralysis—Spasmodic Tabes.*)

Within the lateral columns of the cord, we encounter a bundle of motor fibres which decussate anteriorly in the medulla at its lowest part. They are, therefore, associated with the opposite cerebral hemis-

phere. These fibres occupy only a portion of each lateral column, and lie adjacent to the posterior horn of the spinal gray matter. They are known as the "crossed pyramidal" fibres. They are separated from the periphery of the spinal cord (in some of the spinal segments, although

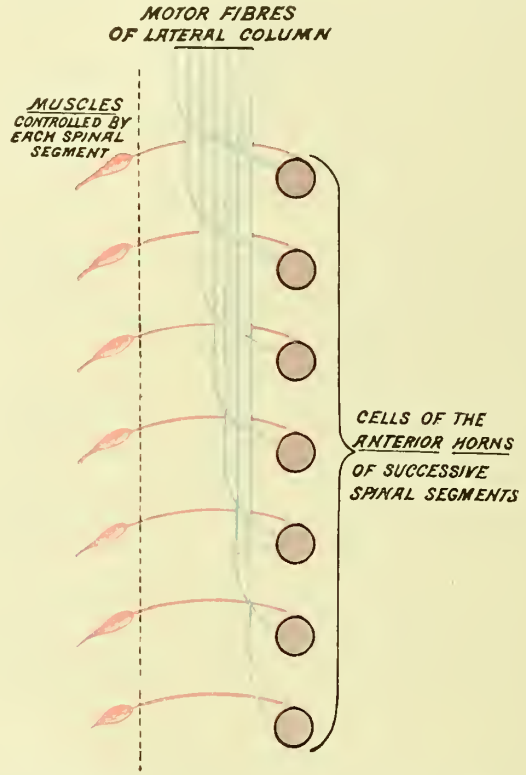


FIG. 93.—A DIAGRAM DESIGNED BY THE AUTHOR TO SHOW THE DISTRIBUTION OF THE "CROSSED PYRAMIDAL FIBRES" (OF ONE SIDE) TO THE CELLS OF THE ANTERIOR HORN OF SUCCESSIVE SPINAL SEGMENTS.—Note that the motor fibres of the lateral column can act upon the muscles only indirectly (through the cells of the anterior horn); also that each segment of the cord receives from the lateral column certain fibres which put the muscles associated with that particular segment in the circuit of cerebral influence (volition). In this diagram, the circles represent groups of cells, and not a single cell. Each red fibre represents a *bundle of fibres* having similar termination. The blue lines represent bundles of motor fibres, which form the anterior nerve-root of successive spinal segments. The terminal muscles (in red) represent the *entire group* controlled by each spinal segment,—not individual muscles. Figs. 19 and 32 will help to further interpret this diagram. This diagram illustrates the reason why the motor columns of the spinal cord grow smaller in size as they reach the terminal segment.

not so in all) by the so-called "direct cerebellar column." (Fig. 19.) The size of this motor bundle decreases gradually (by the giving off of fibres to the various spinal segments) from the cervical enlargement of the cord till it ends in the lumbar enlargement.

Morbid Anatomy.—Sclerosis of this tract may exist as a *primary disease;* and also as a *secondary result*, occasioned by the development of a lesion higher up in a cerebro-spinal axis.

When the disease is of the primary variety, sclerosis is usually found on *both sides of the cord* in the *lateral column.* When it is of the secondary variety, the sclerotic process in the cord (if due to a cerebral lesion) is usually detected in the *lateral column of one side* and the *anterior column of the opposite side.* Fig. 32 will make the reason of this fact apparent to the reader.

In some cases, sclerosis of the posterior columns and the morbid changes of poliomyelitis anterior may coexist with lateral spinal sclerosis. Whether this is due to an extension of the morbid process or not is as yet not thoroughly determined.

Etiology.—Sclerosis of this tract of fibres, when it occurs as a *primary* affection, is rarely encountered before the twentieth or after the fiftieth year of age. It may follow exposure to cold or dampness and injuries of various kinds. In many cases, its causation is very obscure. Some authors believe that it starts as a transverse myelitis.

The *secondary variety* occurs as the result of any morbid process which tends to cut off the fibres of the so-called "motor tracts" from their trophic centres in the motor area of the brain. It is commonly known, therefore, as "secondary degeneration" of the spinal cord. We are apt to encounter this condition as a sequel to any form of cerebral disease which affects the motor fibres or "will-tract." It may also be due to any spinal lesion which has involved the motor fibres of the cord above the seat of the sclerosis.

Secondary degeneration of nerve fibres travels, as a rule, in the direction of the impulses conveyed by the fibres affected (downward in the motor, and upward in the sensory bundles).

PRIMARY LATERAL SCLEROSIS.

(*Primary Spastic Paraplegia—Tetanoid Paraplegia.*)

This condition is usually present upon both sides of the spinal cord. It is most frequently encountered in adults between the ages of thirty and fifty, although it may exist in children. It seems to be more frequent among males than females, and to attack individuals in apparently robust health. It has been suggested that any excessive muscular exercise or strain may predispose to its development. Among children, this form of paralysis may be traced, apparently, in some instances, to an injury received upon the spinal column, to the head during birth, or to some congenital defect in the development of the motor apparatus.

In the opinion of Bramwell, lateral sclerosis of the primary variety is very rarely observed. He attributes the frequency of those cases (which are usually considered as of the primary type) to a transverse myelitis unassociated with disturbances of sensation, and usually present

in the dorsal segments of the spinal cord. In this view he is supported by Leyden.

Irrespective of the relative frequency of this condition as a primary disease or its etiology, the bilateral character of its symptoms is in marked contrast to the unilateral character of the form which is commonly regarded as secondary.

Symptoms.—In this form of spinal sclerosis, the patient is attacked by a paresis of a progressive character. This develops slowly and attacks, as a rule, the lower limb of both sides simultaneously. Subsequently the upper limbs may exhibit similar symptoms. There is in almost every case a marked increase of the spinal reflexes. The paralyzed muscles tend to become rigid to a greater or less extent when sitting, rising or walking. This accounts for peculiarities in the gait of these patients.

No evidences of atrophy in the paralyzed muscles, more than would be accounted for by disuse, are observed. Sensation is preserved in the affected limbs, and there are few if any well-pronounced clinical evidences of disturbed sensory functions. Pain is infrequent, there is little numbness, tingling, or other subjective phenomena, and the viscera of the pelvis are not usually affected, as they are liable to be in myelitis.

The *stiffness in the legs* which accompanies the development of paresis compels the patient to use two canes early in the disease when attempts at walking are made. Subsequently crutches, and possibly an attendant, are rendered necessary. The patient moves with the most extreme difficulty. When an advance step is made the feet appear to be glued to the ground, and are scraped or dragged along rather than lifted. The pelvis and the limb as a whole is lifted in order to allow of the scraping of the foot forward, because little if any flexion is made at the knee.

The *knees frequently tend to become locked together* during the act of walking, because the foot is apt to cross its fellow as it is brought forward. This is an evidence of spasm of the adductor muscles.

As a rule, these patients are inclined to stand upon the toes, rather than on the entire sole of the foot, when walking.

Occasionally the *muscles of the calf are affected with spasm* during attempts at walking, and the foot is then suddenly raised from the ground irrespective of the will of the patient (*hopping gait*). This peculiarity in gait closely resembles that of a horse when affected with what is known as the "string-halt." Again, the patient may be lifted suddenly upon the toes when endeavoring to walk, by contraction of the extensor muscles acting upon the foot.

A peculiar attitude of the back and chest is observed as the patient leans heavily first on one cane and then on the other in order to raise the weight of his body by the arms. The *back is strongly arched* and the *chest is thrown very much forward.*

Whenever the muscles are manipulated they become more or less tense and rigid. This is due to the fact that the *spinal reflexes are very much exaggerated* in this disease.

The knee-jerk is markedly intensified and an "ankle-clonus" can generally be elicited. Sometimes a blow upon the patella-tendon causes a response in the opposite limb. This is known as "*radiation of the reflex.*"

The superficial reflexes are sometimes decreased or abolished; but in exceptional instances they may be exaggerated.

The tests employed to determine the condition of the spinal reflexes have been already described in the second section of this work.

After a lapse of several months or years, these patients are obliged to remain in bed from an inability to walk. The legs then tend to remain stiffly extended; and the thighs are closely approximated, as the result of spasm of the adductor muscles. The feet are usually inverted. Ultimately, the upper extremities may become affected with contracture, in which case flexion predominates over extension.

The *electrical reactions* of the affected muscle are normal or slightly decreased. Those of the nerves may be diminished, both to the faradaic and galvanic currents.

Cerebral complications are rarely if ever developed; the viscera are apparently healthy; and the pelvic organs are not, as a rule, affected.

In somewhat rare instances, one leg alone, or one leg and one arm may be attacked. Such cases are to be diagnosed from hemiplegia or monoplegia of cerebral origin.

Diagnosis.—This condition is to be distinguished from the secondary form of lateral sclerosis; from poliomyelitis; from amyotrophic lateral sclerosis; and from focal lesions of the spinal cord. A subsequent table (p. 364) will make the points of discrimination more apparent than a verbal description.

SECONDARY LATERAL SCLEROSIS.

(*Descending Spinal Sclerosis.*)

This morbid condition is due to a degeneration of the motor fibres of the cord from any cause which tends to separate them from their trophic centres (which are situated within the cortical motor centres of the cerebrum). Whenever a nerve fibre is separated from its trophic cell, it tends, with few exceptions, to degenerate in the direction of the impulses which it is designed to transmit. Hence *motor fibres* usually exhibit under such circumstances a progressive degeneration *downward*, and the *sensory fibres* a similar alteration in an *upward* direction.

The CEREBRAL LESIONS which are liable to produce this form of spinal disease may affect one of the following parts: (1) those convolutions of the brain which are chiefly associated with motion; (2) the motor

bundles of the "corona radiata" or of the "internal capsule;" (3) the caudate nucleus or lenticular nucleus; (4) the crusta cruris; (5) the motor fibres of the pons; or (6) the anterior pyramids of the medulla, above the point of decussation of the pyramidal fibres. Figs. 36 and 79 will make this apparent to the reader.

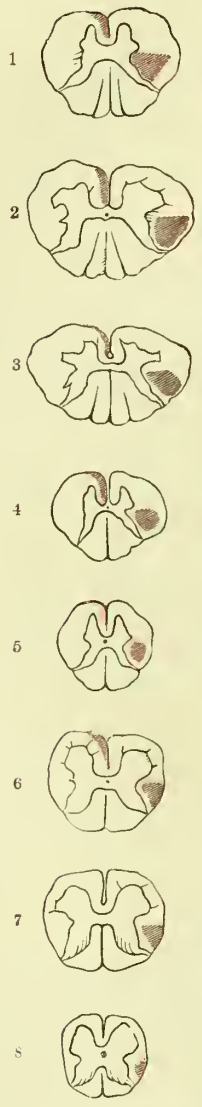


FIG. 94.—A DIAGRAM DESIGNED TO ILLUSTRATE THE SECONDARY SCLEROSIS WHICH WOULD FOLLOW A LESION OF THE LEFT CEREBRAL HEMISPHERE. (After Erb.) Note the involvement of the column of Türck is seen on the left side in sections 1, 2, 3, 4, 5 and 6 (where its fibres end). That of the crossed pyramidal tracts extends throughout the entire length of the spinal cord.

Secondary degeneration of the spinal fibres, which occurs after cerebral disease, tends, as a rule, to progress downward both in the column of Türck on the same side, and in the lateral column of the opposite side of the cord.

Although, in the majority of cases, this results in a bilateral spinal lesion, the preponderance of the symptoms are due to the sclerosis of the lateral columns. They are, therefore, most marked upon the side of the body which is opposed to the cerebral hemisphere primarily attacked.

If, on the other hand, the *primary lesion* is confined *to one side of the cord*, secondary sclerosis of the lateral column will occur only below the level of the primary lesion on the corresponding side of the cord.

Finally, when a transverse spinal lesion which affects both sides of the cord exists, or when a lesion of both cerebral hemispheres or one which crosses the median line cuts off both motor tracts, it may induce secondary sclerosis of a descending character in both of the lateral columns. In the latter case, the symptoms exhibited by the patient during life would be of a markedly bilateral type.

Secondary sclerosis produces, as a rule, about the same train of symptoms as the primary form, with the exception that the symptoms are most marked upon one side; provided they are not exclusively confined to it.

Symptoms.—In this disease, *paresis or paralysis*, *contracture* of muscle, and *ex-*

aggeration of the tendon reflexes are the chief symptoms which are to be expected.

The paralysis or paresis *precedes the development of contracture.* The paralytic symptoms are usually of a very marked character; and they may have developed suddenly. The pelvic organs are liable, moreover, to be affected. The skin, hair and nails may also exhibit trophic disturbances.

All of these symptoms are usually observed either upon one side only; or, if on both sides, one will be more markedly affected than the other. Whenever the exciting lesion is of a bilateral type, the symptoms will be identical with those described under the primary variety.

Diagnosis.—This form of sclerosis is to be distinguished chiefly from a chronic myelitis which involves one lateral half of the spinal cord in its anterior and lateral portions; and also from those diseases which tend to produce a gradual compression of the spinal cord, such as meningitis, tumors, etc. It is far more frequent than the primary variety; and cannot, as a rule, be confounded with it. Moreover, the history of the case will usually point toward some cerebral or spinal lesion as its exciting cause. A subsequent table will aid the reader in making the necessary discriminations between it and other spinal lesions which might be mistaken for it.

Something has already been said regarding the discrimination of the primary and secondary forms of this disease from other spinal affections. The chief diagnostic points by which lateral spinal sclerosis can be distinguished from all other spinal diseases (provided it be itself uncomplicated) are: (1) the development of paralysis with *contractures and rigidity* of the muscles; (2) the *absence of atrophy*; and (3) the *marked increase in the tendon reflexes.*

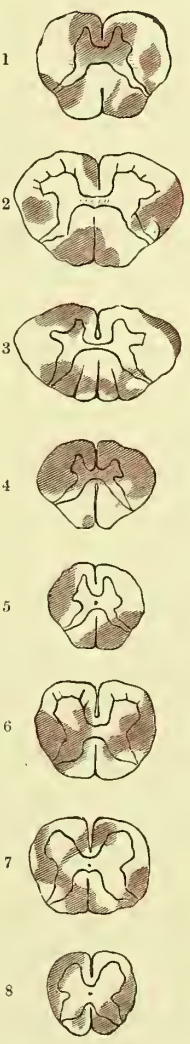


FIG. 95.—A DIAGRAMMATIC REPRESENTATION OF THE CORD IN MULTIPLE SPINAL SCLEROSIS. (After Erb.) This condition, as will be seen, affords a marked contrast with systematic sclerosis shown in Figs. 92 and 94.

When no cerebral symptoms or those of a lesion of the upper part of the cord (bulbar symptoms) have preceded the development of the contractures and paresis, it is safe to infer that the primary variety exists.

	LATERAL SPINAL SCLEROSIS.		POLIOMYELITIS.		AMYOTROPHIC LATERAL SCLEROSIS.	FOCAL LESIONS.
	Primary.	*Secondary.*	*Acute.*	*Chronic.*		*Multiple Spinal Sclerosis, Tumors, Meningitis, etc.*
CONTRACTURE OF MUSCLES	Not preceded by paralysis. Is usually *bilateral.*	*Preceded by paralysis.* May be *unilateral.*	Absent.	Absent.	Causes a characteristic deformity of the limbs, especially in the upper extremity.	May be unilateral (if present).
ATROPHY OF MUSCLES.....	Absent.	Absent.	Develops after the paralysis.	Develops after paralysis, or independently of paralysis.	Present.	May or may not exist. May be unilateral or bilateral.
TENDON REFLEXES.....	*Exaggerated on both sides.*	*Exaggerated on affected side.*	Unaffected.	Unaffected.	*Exaggerated.*	Will be exaggerated if the *lateral column* is involved.
HISTORY OF CASE.....	No previous brain lesion has existed.	Some previous lesion of brain or spinal cord causing paralysis of motion has existed.	*Febrile symptoms* and *paralysis* precede the atrophy.	*Whole groups* of muscles undergo atrophy simultaneously.	The nuclei of the motor cranial nerves of the medulla become affected.	Clinical history will often give valuable information respecting the morbid condition which exists.
SENSORY FUNCTIONS.....	Normal.	Normal.	Normal.	*Pain precedes the Atrophy.* No anæsthesia.	Normal.	Pain, anæsthesia, hyperæsthesia, motor paralysis, etc., may exist, showing *that the lesion is non-systematic.*
ELECTRICAL CONTRACTILITY.....	Not markedly affected.	Normal.	Abolition of faradaic contractility early in the case. "Reaction of degeneration."	"Reaction of degeneration."	Faradaic contractility is never abolished.	Normal.

Prognosis —Cases of apparent recovery from this affection have been recorded; and in many instances a period of indefinite duration, in which no advance of the symptoms takes place, seems to be developed. I believe that a few cases may be cured by proper treatment, and that almost all can be materially helped if seen sufficiently early.

When it results fatally, the nuclei of the medulla are usually first affected, thus producing the symptoms of the so-called "glosso-labio-laryngeal paralysis," or Duchenne's disease.

Treatment.—It is my custom to administer large doses of ergot to these patients very early in the disease,—before the paresis or contractures become very apparent. This drug alone will occasionally arrest the disease, in my opinion.

To syphilitic patients in the active stage, or those who have been infected with that disease at any time, I administer the treatment suggested on page 291.

I usually employ the galvanic and static currents to the paralyzed muscles from the onset; and, as a tonic, I am in the habit of giving cod-liver oil and quinine. The nitrate of silver in combination with the extract of belladonna may prove of service. Hot water (as recommended on page 248) has helped some of my cases.

I am satisfied that I have obtained marked and permanent improvement by the use of the *cautery* and *severe static sparks* when applied on each side of the spinous process of the vertebræ and the contractured muscles every other day, or less often, for several weeks consecutively.

The spasms may be relieved by daily hypodermic injections of gr. $\frac{1}{120}$ of atropine to begin with, and gradually increasing the dose.

The efficacy of *static electricity* (administered chiefly by the "spark") in relieving contracture and spasm of muscles is now quite well established to my mind. I have had quite an extended experience with this agent; and I know of nothing that will give such immediate relief to patients so afflicted.

The great disadvantage which most static machines labor under (in the hands of those who own them) is their lack of power. A machine which gives but a feeble and thin spark is practically useless for medical purposes. Personally, I cannot recommend revolving plates of less than 24 inches in diameter; and several such plates are needed to generate the quantity which is requisite to a satisfactory use of the induction machine.

INFLAMMATION OF THE CELLS OF THE ANTERIOR HORNS.

(*Poliomyelitis Anterior, of Infants and Adults—Atrophic Spinal Paralysis—Infantile Paralysis.*)

This condition sometimes coexists with lateral sclerosis. It frequently occurs, however, as an independent affection, especially during childhood.

The cells of the anterior horns are affected by an inflammatory process in this disease; which, if sufficiently severe, leads to their *atrophy* or *destruction*.

Morbid Anatomy.—When these cells become inflamed, their function is at once arrested; hence *sudden paralysis* is developed, provided the inflammatory action be of the acute type. If the gray matter be so affected beyond the possibility of recovery, acute pigmentary degeneration of the cells so attacked apparently follows. The name poliomyelitis

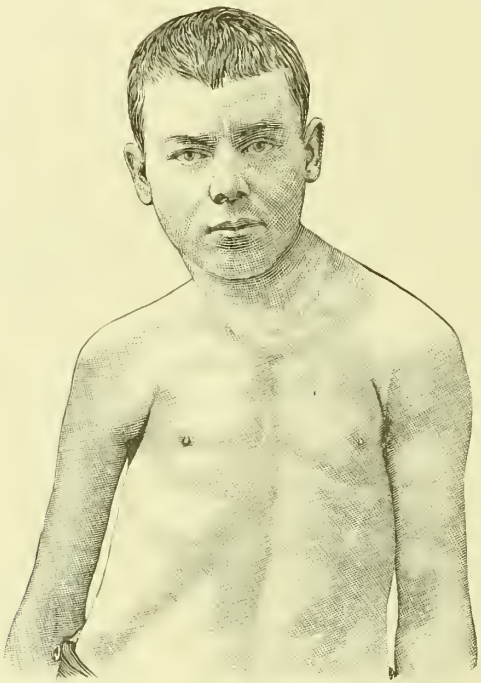


Fig. 96.—Poliomyelitis Anterior (Acute) followed by Extensive Atrophy, chiefly of the Right Side. (From a photograph in the possession of the author.)

(πολιός, *gray*, and μυελός, *marrow*) expresses the seat of the lesion, as well as its inflammatory character.

As a result of inflammatory and degenerative changes within the cells of the anterior horns, the spinal nerve-fibres (which serve to connect the inflamed cells with the muscles) degenerate as a result of defective nutrition; and the muscles connected with those fibres also undergo rapid fatty degeneration and atrophy. The symptoms of this disease tend to confirm the view that the ganglion-cells of the anterior horns preside not only over muscular movement, but that they also serve to regulate the nutrition of the muscles connected with them by means of the fibres which

compose the anterior nerve-roots. It is believed by some observers, however, that some of the cells found in this locality have a peculiar trophic function.

The inflammatory changes observed in the spinal cells during an attack of poliomyelitis must not be confounded with a non-inflammatory degeneration of the cells in the anterior horns. This is probably the spinal cause of "progressive muscular atrophy" (in which there is no paralysis).

The changes observed in poliomyelitis anterior are commonly confined to a *few spinal segments*. It is not uncommon for the horn of one

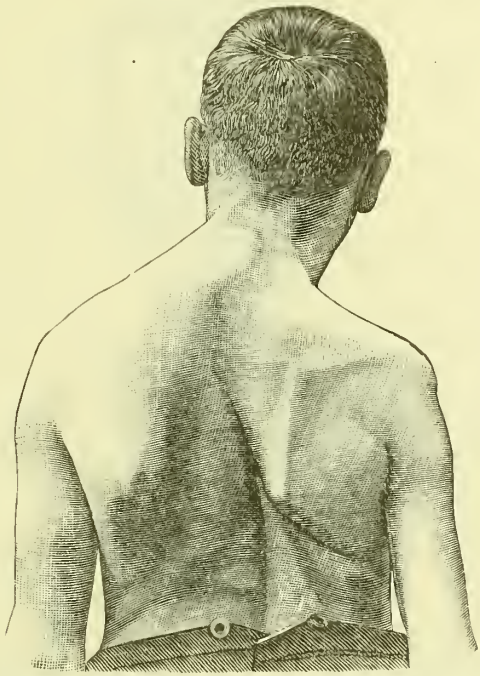


FIG. 97.—BACK VIEW OF SAME CASE, SHOWING THE SO-CALLED "WING-SCAPULA" (ESPECIALLY ON RIGHT SIDE) FROM ATROPHY OF THE MUSCLES.

side to escape while the other is seriously involved. Whenever the attack has been a severe one, the anterior horn of the affected segments will appear after death to be more or less distorted from atrophy of its cells and the development of newly formed connective-tissue.

The *form of paralysis* which develops in any given case will be modified by the spinal segments attacked; and also by the character of the attack, whether unilateral or bilateral. One of my cases exhibited an attack confined to the right horn of the cervical segments and the left horn of the lumbar segments. He had paralysis and extensive atrophy

of the right arm and paralysis of the left leg, which passed away after a lapse of a few weeks.

If the inflammatory process is not sufficiently severe to prevent the recovery of the cells attacked, the spinal cord may exhibit no evidences after death of destructive processes.

Etiology.—This disease is commonly described as of three varieties,—the acute, subacute, and chronic. It is more common in children than in adults, although the chronic variety is less frequently observed during childhood than the acute.

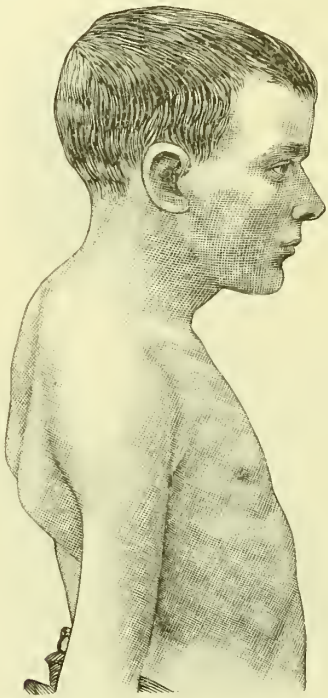


FIG. 98.—PROFILE VIEW OF SAME CASE, SHOWING ATROPHY OF DELTOID REGION, THE ALTERED POSITION OF THE RIGHT SCAPULA, AND THE WASTING OF THE RIGHT ARM.

The *infantile variety* has been known to follow exposure to cold or dampness, overfatigue of the muscles, some forms of blood-poisoning (such as eruptive fevers, diphtheria, lead-poisoning, etc.), dentition, and traumatism. Some cases develop from imperfectly understood causes. It generally occurs before the second year,—seldom later than the seventh year. It is most common among boys.

The *adult variety* seems to be excited chiefly by exposure to cold or dampness and overexertion. Lead-poisoning is said to sometimes excite it. Debility, convalescence from fevers, malaria, pneumonia, etc., are

mentioned by authors as among its factors of causation. It may develop between the ages of twenty and fifty years. One of my patients (Figs. 99 and 100) was so affected (after severe exertion and excessive indulgence in alcohol) from sleeping on the ground during a summer shower. The paralysis in this case attacked the muscles which were chiefly employed by him in his occupation.

Symptoms.—The three forms of this disease will be described separately, as they should be distinguished at the bedside.

Acute Form. (*Infantile Spinal Paralysis—Acute Spinal Paralysis of Adults.*)—The onset of this form is usually marked by a *sudden elevation of*

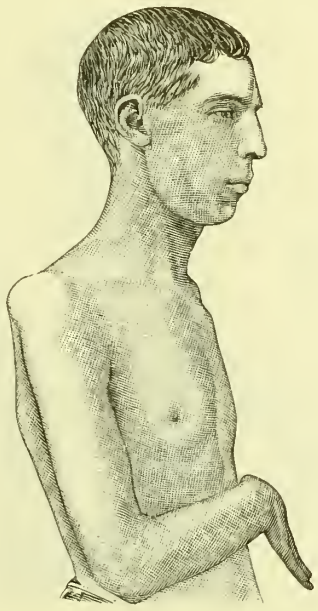


Fig. 99.—Poliomyelitis Anterior Acuta, occurring in the Adult from Sleeping on Wet Ground. (From a photograph in the possession of the Author.) Note the extensive atrophy of deltoid region, forearm, and hand.

temperature. The febrile symptoms may be either continued or remittent in type. The fever may last from twenty-four hours to several days. It is not uncommon to observe pains in the limbs, muscular twitchings, tremors, convulsions, delirium, and occasionally a sense of numbness in connection with the stage of fever.

Sudden paralysis of a marked character soon follows, and with its appearance the febrile symptoms disappear. The paralysis usually attains its height at the onset.

The *seat and type* of the paralysis vary with the spinal segments affected and with the character of the attack,—whether unilateral or

bilateral. All the limbs may be affected in some cases. (*Complete paralysis.*) Again, it may be confined to one lateral half of the body. (*Hemiplegia.*) When the lesion is bilateral and confined to the dorsal or lumbar segments, *paraplegia* may develop. Finally, if the lesion be unilateral and confined to the cervical or lumbar segments, *monoplegia* may occur. I have personally reported an unique case, where the right upper and left lower limb were simultaneously paralyzed.

The paralysis of motion is usually *quite complete* at the onset. It is not accompanied by any disturbances of the sensory function as a rule,

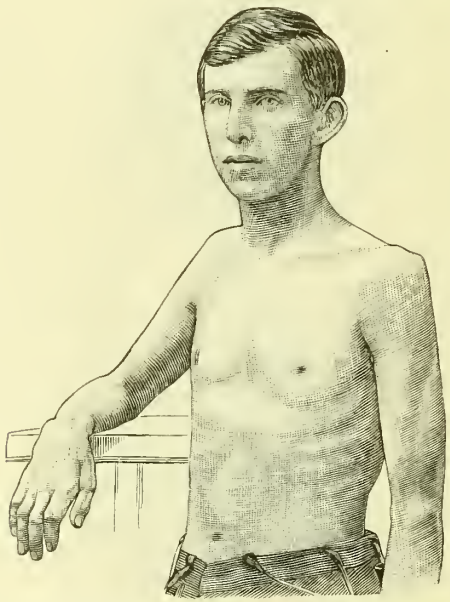


FIG. 100.—FULL VIEW OF SAME CASE, SHOWING THE DEFORMITY AND ATROPHY OF HAND.
(From a photograph in the possession of the Author.)

although a slight numbness may be complained of by the patient. The pelvic organs are not affected.

In infants, the existence of paralysis may be overlooked. Sooner or later, the nurse or mother may notice that the child does not move its arm or leg. When this disease is suspected during infancy, the movements of the limbs should be very carefully observed. Nurses are often unjustly blamed by physicians as well as parents for the development of paralysis in infants intrusted to their charge. The misfortune is in many cases erroneously attributed to some blow or fall which the child may have received. The fever which precedes the development of the

paralysis is, therefore, a very important and valuable point in the diagnosis of this affection.

Soon after the onset of the paralysis, the affected muscles of the limb (some usually escape) cease to respond to the faradaic current. They also contract slowly and with *abnormal formulæ* when the galvanic current is used (thus exhibiting evidences of nerve degeneration and muscular degeneration. (Fig. 58.) Later on, they exhibit a marked increase of galvanic excitability, with abnormal formulæ.

Whenever the cells are not sufficiently impaired to produce a *permanent arrest of their function*, the paralyzed muscles begin in a short time to show a return of power. In such a case, the normal formulæ of muscular reaction to galvanism returns, and faradaic currents begin to cause muscular contraction.

After several weeks have elapsed, *signs of atrophy* will begin to be apparent in those muscles whose cells have been most seriously injured. In all such cases, the muscular atrophy is markedly progressive and more or less permanent. If a piece of such a muscle be removed by Duchenne's trocha, evidences of extensive fatty degeneration could be discerned under a microscope. This step is sometimes a valuable one in making a diagnosis or a prognosis.

This variety is commonly described as "*infantile spinal paralysis*," because children are more often affected than adults. Nevertheless, it is still encountered in adult life, but rarely in old age. Prévost and Charcot were the first observers to discover the exact morbid changes which occur in the anterior horn in this type of disease.

Subacute Form.—This is a rare type of disease, and never affects children. It differs from the acute variety in the gradual onset of the paralysis, the total absence of all cerebral symptoms, the presence of only slight febrile symptoms, and the fact that it attacks adults exclusively. It may closely resemble lead-paralysis and progressive muscular atrophy.

Chronic Form.—A chronic type of inflammation confined to the anterior horns causes symptoms which may closely resemble those of multiple neuritis and "progressive muscular atrophy." It is a comparatively rare form of disease. It may attack children or adults.

This condition may be recognized from the other forms of poliomyelitis chiefly by its chronicity. The presence of severe neuralgic pains and other abnormal sensory phenomena, which exist for weeks or months prior to the development of atrophic changes in the muscles, points rather to multiple neuritis. The type of muscular atrophy which occurs in this affection differs from that observed in progressive muscular atrophy, in that it affects entire groups of muscles simultaneously. In the latter disease, separate bundles in the affected muscles may be

destroyed, while others may remain unchanged. The reactions of the affected muscles to the faradaic and galvanic currents are similar to those of the acute variety.

Diagnosis.—Acute poliomyelitis in the infant may be mistaken for *toxic neuritis*, *progressive muscular atrophy*, *rickets*, the *wasting diseases of childhood*, *pseudo-hypertrophic paralysis*, *hemiplegia* from cerebral or spinal lesions, and (during its initial stage) with the *exanthemata* or some of the *inflammatory disorders*.

In the adult, the acute form might be confounded with *multiple neuritis*, *progressive muscular atrophy*, *amyotrophic lateral sclerosis*, *hemiplegia*, *monoplegia*, *myelitis*, and the *subacute variety of poliomyelitis*.

It does not seem to me possible for the reader to err in the recognition of this affection, provided the essential facts pertaining to this disease are firmly fixed in his memory.

It should be remembered that the acute form of the disease begins with a stage of febrile excitement, which ceases in a short time; that paralysis develops suddenly, and reaches its height at once; that the paralysis begins to improve almost immediately after its appearance; that atrophy of some of the muscles previously paralyzed also begins soon after the attack; that no cerebral symptoms will have existed previous to, during, after the attack; and that the child or adult has usually been in perfect health up to the commencement of the disease.

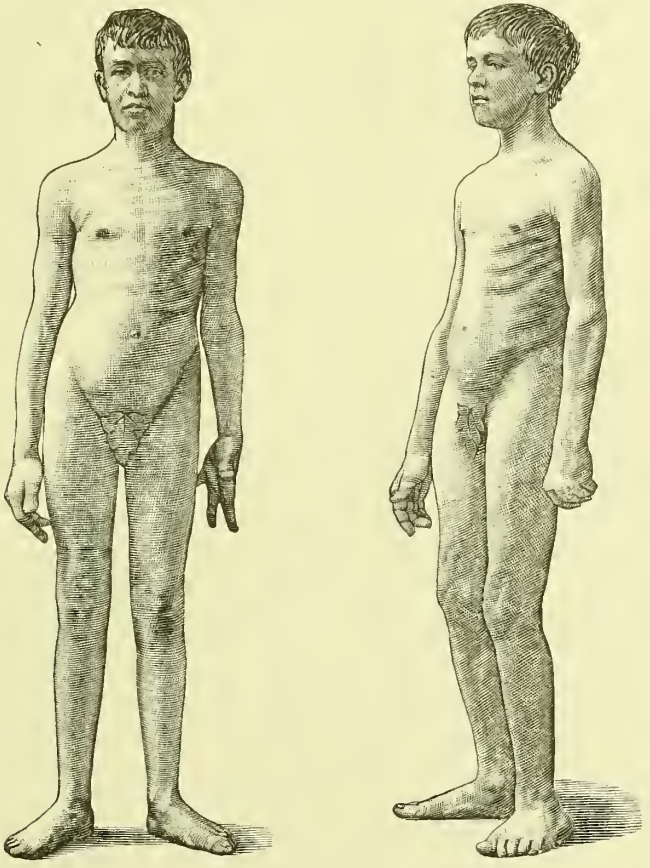
The paralysis is at first generally extensive; but it soon becomes limited to a greater or less extent. Rare exceptions to this rule have been recorded, however, where the reverse has occurred. Atrophy of the muscles follows the paralysis. There is usually a diminution of reflex spinal irritability. Bed-sores do not occur, nor are the bladder and rectum affected. Disturbances of sensibility are absent, as a rule.

The distinctions between the acute, subacute, and chronic types of poliomyelitis are those of degree rather than of kind. The respiratory muscles are never involved in true spinal paralysis, according to Seguin. In this respect he draws a line of distinction between the so-called "acute ascending paralysis" and poliomyelitis. To my mind this clinical distinction is questionable.

From *progressive muscular atrophy* acute poliomyelitis is to be distinguished by the rapid development of the paralysis, the febrile stage which precedes its development, its appearance before the seventh year of age, and the fact that the faradaic current fails to create a response in the paralyzed muscles; whereas in progressive muscular atrophy the uninvolved fibres of the affected muscles respond to faradaism. The insidious advent of progressive muscular atrophy and the effects of electric tests (p. 189) would decide between it and

poliomyelitis in the adult. Furthermore, by means of Duchenne's trochar (p. 212), the fibres of the muscles attacked may be examined microscopically.

Pseudo-hypertrophic paralysis can be easily distinguished from poliomyelitis by the absence of a febrile stage, the increase in size of



FIGS. 101 and 102.—A CASE OF INFANTILE PARALYSIS, WITH INVOLVEMENT OF THE MEDULLARY NUCLEI. (From two photographs in the possession of the Author). The deformity of the left side of the patient and the facial atrophy of the left side is well shown. Several years had elapsed between the date of the attack and the taking of the photographs.

the muscles, the locality affected, the normal electro-muscular phenomena, the characteristic gait (p. 164), the late development of symptoms in the extremities, the peculiar curve of the vertebral column, and the microscopical examination of the muscles.

Rickets never produce paralysis, alterations in the normal electro-muscular reactions, nor a stage of well-marked fever.

Hemiplegia of cerebral origin can be diagnosed from poliomyelitis by the history of the case, the presence of symptoms of impairment of the intellect or speech, by paralysis of some of the cranial nerves, the development of hemiplegia and hemianæsthesia upon the same side [if the lesion be non-cortical (p. 72)], the presence of normal electro-muscular formulæ, the absence of a febrile stage, and abnormalities in the pupil.

Myelitis commonly causes more or less trophic disturbances; but it is very liable to create symptoms referable to the genito-urinary tract as complications, and to present all forms of combinations of motor and sensory symptoms, which do not exist in poliomyelitis.

Some other points in the differential diagnosis of this affection have been given in a table when discussing sclerosis of the lateral column of the cord. (P. 364).

The condition of *multiple neuritis* has been very frequently mistaken for poliomyelitis. There seems to be little, if any, doubt that many cases reported in the past by authors of note as those of poliomyelitis, were improperly classed.

The development of abnormal sensory phenomena, such as pain, anæsthesia, parasthesiæ, etc., in connection with motor disturbances and muscular atrophy, should always lead to the suspicion of the existence of multiple neuritis.

Prognosis.—In cases afflicted with poliomyelitis, partial or complete recovery usually takes place. Some of the muscles may undergo permanent atrophy. Deformities may ensue from post-paralytic contracture, in some cases. As a rule, the electro-muscular phenomena return to the normal standard in the muscles which are the least affected. The power of motion is regained with greater or less rapidity and completeness; and the reflexes tend to return to the condition of health. If the muscles continue to respond at all to the faradaic current during the height of the attack, it is safe to predict a total recovery. I have never seen a muscle undergo permanent atrophy when it constantly preserved even a trace of faradaic excitability. If the disease creates interference with the action of the respiratory nerves, it is possible for a fatal termination to take place. Happily, such instances are uncommon.

Treatment.—There exists experimental as well as clinical evidence to show that a regeneration of the cord may sometimes take place after a serious injury. Hence we are justified in devoting particular care and attention to the medicinal and mechanical treatment of the peripheral manifestations of spinal disease with the hope that the cord itself may be stimulated and eventually regain its functions.

The administration of large doses of *ergot* early in the acute variety of poliomyelitis (as first suggested by Hammond) often tends to check

the inflammatory process. Ten drops of the fluid extract may be given with safety three times a day to a six-months-old infant. It should not be given after evidences of muscular atrophy appear.

Strychnia by the mouth, or by injection into the paralyzed muscles, is sometimes of benefit to these subjects later in the disease. The dose must be graded to the age of the patient. I never give over one-hundredth of a grain at a dose to a child under one year of age. Personally, I prefer the hypodermic method of administration.

Daily immersion of the limbs in hot salt water (110°–120°) for thirty minutes, *friction* (made by the hand or a rough towel several times a day), *massage*, and *passive movements* all tend to excite a determination of blood to the paralyzed muscles, and are of great utility in these cases. I do not believe in the use of Jounod's boot, as I have known of serious harm being done by it. It is a dangerous instrument in the hands of inexperienced persons.

To adults I often recommend the *internal administration of hot-water* drinking. If administered to children, a competent nurse must supervise its use. I have given the rules for its administration in a preceding section (p. 248).

Electricity is an extremely valuable adjunct to treatment in these cases. It must be kept up for a long period of time, and the parents or the patient must be prepared for slow results. When the faradaic current fails to create responsive contractions of the muscles, the galvanic or static current must be substituted for it. The strength of the current must be sufficient to create muscular contractions. It should not be used oftener than on alternate days, as a rule. Months, and even years, may elapse before the muscles are brought back to the standard of health.* Pieces of the affected muscles may be removed from time to time (through the aid of Duchenne's trochar) and examined microscopically. In this way we can decide regarding the progress of the muscular atrophy. If the disease is progressing favorably, the percentage of oil-globules scattered throughout the muscular fibres will show a decrease.

During the acute stage, the patient should be kept in bed. After all febrile symptoms have disappeared this is not necessary.

The tonic plan of treatment—iron, quinine, cod-liver oil, arsenic, phosphorus, etc.—may be combined with hypodermics of strychnia when deemed necessary. The diet should be nutritious and adapted to easy digestion and assimilation.

* One of my cases made a perfect recovery under electrical treatment and massage, in spite of the fact that the lower limbs had been almost completely paralyzed for over three months previous to my examination of the child. She moved about the room by the aid of her hands only when I made my first examination of the patient.

AMYOTROPHIC LATERAL SCLEROSIS.

In connection with two systematic spinal conditions which have been already described, viz., lateral sclerosis and changes in the anterior horns, it may be well to consider another systematic affection of the spinal cord where the two are combined. This has been named by Charcot, who first recognized the pathological changes which tended to produce it, "amyotrophic sclerosis." The term "amyotrophic" (signifying an absence of muscular nutrition) expresses well the chief morbid change which characterizes this form of spinal sclerosis.

Morbid Anatomy.—The lesion is not confined necessarily to the spinal cord, for it tends to extend throughout the medulla oblongata and even into the peduncle of the cerebrum. Hence the nuclei of the hypoglossal, spinal accessory, and facial nerves are involved, as a rule, late in the disease. The changes in the anterior horns in this disease are apparently identical with those which exist in connection with progressive muscular atrophy. The morbid process seems to start first in the cervical enlargement of the spinal cord; for that reason the muscles of the upper extremity are first attacked.

From these segments the sclerotic and degenerative processes generally extend both upward and downward. Bands of dense, newly-formed, connective tissue are often detected between the sclerosed lateral columns and those portions of the anterior horns which are involved.

In the atrophied muscles, the perimysium undergoes a marked hyperplasia. Inflammatory changes are more apparent than in progressive muscular atrophy.

When the medulla becomes involved, the cells which constitute the motor nuclei within the gray matter of the floor of the fourth ventricle undergo a rapid degeneration.

The deformities of the limbs which result from muscular contracture are extreme in this type of spinal sclerosis.

Etiology.—The causes which conduce to the development of this condition are apparently similar to those mentioned in connection with poliomyelitis and primary sclerosis of the lateral columns. Exposure to cold or dampness seems to be a prominent cause. In one of my cases, it developed after prolonged and intense mental anguish following the death of a child.

Charcot, who has investigated this disease, divides its manifestations into three distinct phases. These are as follow:—

1. The first stage is manifested only in the muscles of the upper extremities. 2. During the second stage, the muscles of the lower extremities are attacked. 3. In the third stage, the morbid process extends to the medulla oblongata.

Although this clinical distinction is generally true, there may be exceptional instances in which the disease attacks the medulla first, and gradually extends downward. Again, cases in which the disease first attacks the lower limbs and gradually extends upward, have been reported.

First Stage.—The duration of this stage varies from four months to a year. During this time tremors of the upper limbs appear early, and paresis or paralysis subsequently develops. There is no alteration in the electric tests of the muscles. Fibrillary twitchings are commonly observed in the muscles of these patients.

An *extensive form of atrophy* follows the paralysis, and the muscles tend to develop a state of *rigidity and contracture* which creates permanent deformities. The characteristic deformity of this disease is chiefly observed in the hand, the wrist and fingers being permanently

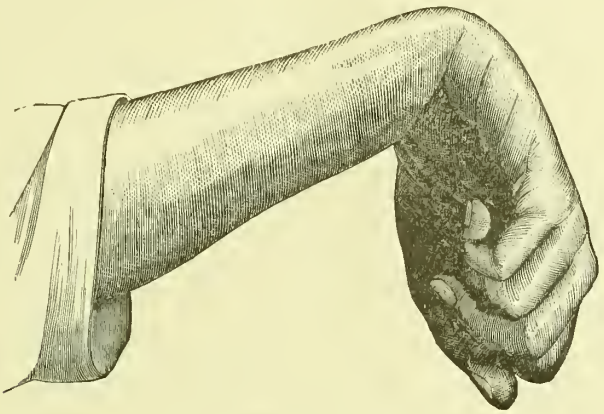


Fig. 103.—Hand in Amyotrophic Lateral Sclerosis. (Charcot.)

flexed to a greater or less extent, and more or less rigid. Fig. 103 illustrates this attitude.

Sometimes the muscles of the neck and jaw are thrown into a state of spasm, which is more or less persistent. After atrophy has progressed to a marked extent in the forearm, this rigidity of the neck and jaw usually tends to disappear. Several months usually elapse between the first and second stages, during which time the disease appears to remain stationary.

Second Stage.—As this disease advances, after it has apparently remained stationary for a time, the muscles of the lower limbs begin to exhibit *evidences of paralysis*, and, at the same time, *tonic or clonic spasms* (or both forms) may simultaneously develop. Gradually the state of permanent rigidity or contracture appears in some of the affected muscles.

The *spinal reflexes*, chiefly the knee-jerk, are very much increased, and in some cases an ankle-clonus may be detected.

After a considerable lapse of time has occurred, the muscles of the lower limbs tend to become less rigid and give place to *atrophy* and *fibrillary twitchings*.

The *pelvic organs* are not usually disturbed, nor is there any tendency toward the development of bed-sores which are so commonly observed in connection with myelitis.

The *muscular atrophy* in the upper limbs increases to a very marked extent during this stage.

Third Stage.—In many patients afflicted with this disease, the development of "*bulbar*" *symptoms* are superadded to the symptoms of the second stage.

When these occur the disease has extended to the medulla, and has involved the nuclei of origin of the cranial nerves which arise from the medulla. These nuclei are situated in the gray matter of the fourth ventricle. (Fig. 16.) In this stage, we are liable to encounter evidences of paralysis in the muscles of the tongue, lips, larynx, and pharynx. The patient experiences difficulty in articulation, in swallowing, and in controlling the escape of saliva from the mouth. During the act of swallowing the food is liable to be expelled in part through the nose, and it is with great difficulty that some patients are able to get the bolus of food into the pharynx. This distressing condition is commonly known as Duchenne's disease. Its physiognomy is shown in Figs. 108 and 109.

Serious disturbances of the *circulation and respiration* are apt to occur during this stage from paralysis of the pneumogastric nuclei. These symptoms may prove the cause of death.

Amyotrophic lateral sclerosis usually proves fatal within two years after its initial symptoms make their appearance.

Diagnosis.—This disease can hardly be confounded with any other spinal affection, in spite of the fact that some of its manifestations closely resemble those of progressive muscular atrophy and poliomyelitis. When we review the symptoms of the three we may easily make the necessary discrimination. (See table on p. 364.)

From *progressive muscular atrophy* this disease can be distinguished by the following facts: The atrophy follows the paralysis and attacks groups of muscles, rather than individual fibres. Contractures develop, resulting in characteristic deformities of the limbs. The progress of the disease is rapid. The legs are attacked soon after the arms. The medulla becomes implicated in almost every case. The disease is rapidly fatal.

From *poliomyelitis* (adult variety) the diagnosis is made by the fact

that the reflexes are impaired, but the farado-muscular excitability not decreased. Moreover, the atrophy is more rapid and permanent in this form of spinal sclerosis. The contractures of muscles are conducive to greater deformity; the medulla is implicated; fibrillations are present; improvement in the symptoms is rare; and the disease is always fatal.

Prognosis.—I am not aware that a case of cure has ever been reported. Death usually results from an embarrassment of the circulatory and respiratory functions, in consequence of an extension of the morbid changes to the nuclei of the medulla. It is uncommon for these patients to live over two or three years.

Treatment.—Nothing can be said under this head which will aid the reader in curing this disease. Its advent deprives the patient of all hope of cure, and places the fatal termination at no very distant date. I have, however, apparently succeeded, by employing static sparks daily to the spine and limbs, in greatly relieving the contracture of the muscles and holding the progress of the disease in check for many months.

PROGRESSIVE MUSCULAR ATROPHY.

The close analogy which this disease bears in some instances to "poliomyelitis anterior" in the adult as regards its symptoms will help to explain the fact that such cases are sometimes erroneously regarded as a variety of progressive muscular atrophy.

This disease is essentially one of adult life, although the so-called "pseudo-hypertrophic paralysis" of children bears some resemblance to it. It affects males more frequently than females, and usually appears between thirty and fifty years of age.

Morbid Anatomy.—*A slow degeneration of the ganglion cells of the anterior horns* of the spinal gray matter, probably independent of inflammatory changes, exists in this disease. It is one of the most chronic and incurable of all spinal affections, and is comparatively common. By some authors, this disease is believed to start in the muscular tissue, in some instances,—and possibly in all.

The *vessels* of the cord are often abnormally dilated. The arterioles may be sclerosed, and an inflammatory exudation sometimes surrounds the larger arterial trunks.

The *neuroglia* is excessively developed. Sometimes the anterior roots of the spinal nerves exhibit atrophic changes and discoloration.

The *anterior horns* of gray matter are altered both in their size and appearance. The *ganglionic cells* within them are atrophied or completely obliterated.

The *affected muscles* are shrunken and pale in color to the naked eye. The microscope reveals a disappearance of the transverse striæ in

the fibrillæ; and in those bundles most diseased all traces of muscular tissue may have disappeared. The muscular tissue has been replaced by oil-globules. This process of muscular disintegration does not appear to affect whole muscles at once; it seems to attack only individual bundles, or even fibrillæ. Eventually, an entire muscle may thus be destroyed, bundle by bundle, or fibre by fibre.

I am personally inclined to believe that this disease is primarily one of the spinal cord, and that the muscular changes are a result of trophic disturbances dependent upon the morbid process within the spinal gray matter.

Etiology.—Among the exciting causes of this affection, which have been reported by authors of note, may be mentioned: (1) a hereditary

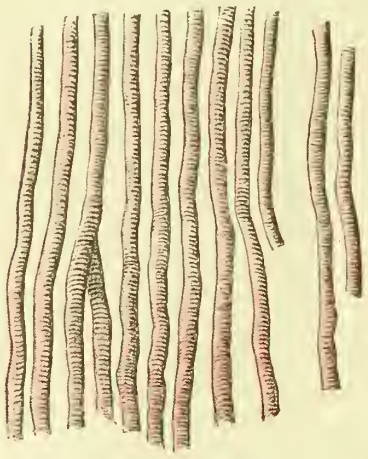


FIG. 104.—FIBRES FROM THE DIAPHRAGM IN HEALTH. (Charcot.)

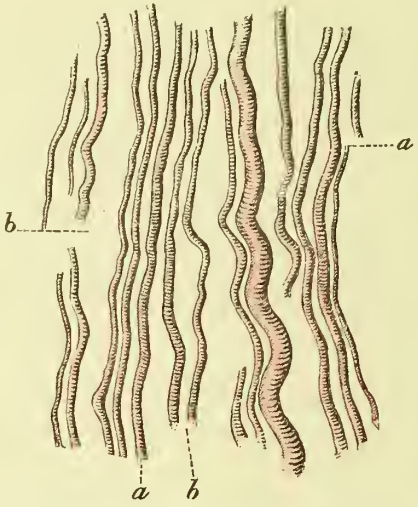


FIG. 105.—SAME TAKEN FROM A CASE OF PROGRESSIVE MUSCULAR ATROPHY AFFECTING THE DIAPHRAGM. (Charcot.) The muscular fibres (*a*) are greatly atrophied but preserve their transverse striæ. The connective tissue intervals (*b*) are enlarged.

tendency; (2) excessive muscular efforts; (3) traumatic injuries of peripheral parts of the body; (4) lead poisoning; (5) certain blood conditions, such as rheumatism, measles, typhoid, etc.; (6) exposure to cold and dampness; (7) excessive venery and masturbation.

There seems to be no doubt regarding an etiological relationship between *certain occupations* (demanding an excessive and continual use of the fingers and hand) and progressive muscular atrophy in some of the cases reported.

Symptoms.—The chief clinical feature of this disease is the development of *extensive and progressive atrophy* of certain muscles.

The wasting of the muscle may not be detected by the patient for some time after its onset. It is unattended with any symptoms of paralysis, and there are no febrile manifestations to mark its advent.

The patient usually first perceives that certain muscles are apparently wasting, and that some loss of power has occurred in the diseased part in consequence of the diminution in size of the affected muscles. This loss of power is always proportionate to the extent of the atrophy. In this respect, this disease differs from those spinal affections in which paresis occurs independently of alteration in the muscular structure.

As a rule, progressive muscular atrophy commonly affects the *upper extremities first;* and, in many cases, homologous regions on both sides are successively attacked.

Patients commonly first observe a wasting of the muscles of the hand and of the shoulder. A considerable lapse of time usually occurs before any evidence of atrophic changes in the lower limbs can be detected.

In the *hand*, certain muscular groups are generally attacked,—chiefly those of the thenar and hypothenar eminences, and the interossei muscles. These patients complain early of more or less stiffness in the fingers and an inability to perform delicate manipulation with the hand. Cold tends to increase this difficulty, and warmth to diminish it. The ball of the little finger and thumb becomes very much wasted as the disease progresses, and the bones of the metacarpus tend to become unduly prominent.

Sooner or later the hand assumes a characteristic deformity on account of a predominance of power in the extensors and abductors of the thumb (the so-called "*ape-hand*"). Marked atrophy of the interossei, combined with the unopposed action of the lumbricales, may give rise to a condition commonly known as the "*claw-hand.*" Fig. 106 well illustrates this deformity.

Flattening of the palm is occasionally observed as the result of atrophy of the lumbricales.

When the *muscles of the shoulder* become involved, the movements of the arm are more or less interfered with, and the deltoid region is markedly flattened. Atrophy of the scapular muscles may also occur; in which case movements of the arm are still more seriously embarrassed.

In the *forearm*, the extensor muscles undergo atrophy more frequently than the flexors. The supinators usually escape atrophy.

As a rule, the *right hand is attacked before the left.* The muscles of the scapula and trunk are not generally attacked until the arms exhibit very marked atrophic changes.

When the muscles of the back or abdomen undergo atrophy, a

characteristic posture during the erect attitude is developed. (See Figs. 54 and 55.)

In very rare instances the diaphragm may undergo atrophy, and create difficulties in respiration.

The *lower limbs* are usually attacked late in the disease. I have observed one very striking case, in which the muscles of the thigh were very extensively wasted, in spite of the fact that the arms and trunk remained unaffected. Generally, the flexors of the legs are the first to exhibit atrophy when the disease has extended to the lower extremities.

The *electrical reactions* of the muscles undergoing atrophy are normal in their formulæ. They are, however, impaired (as is the power of the muscle) in a direct proportion to the number of fibres which are

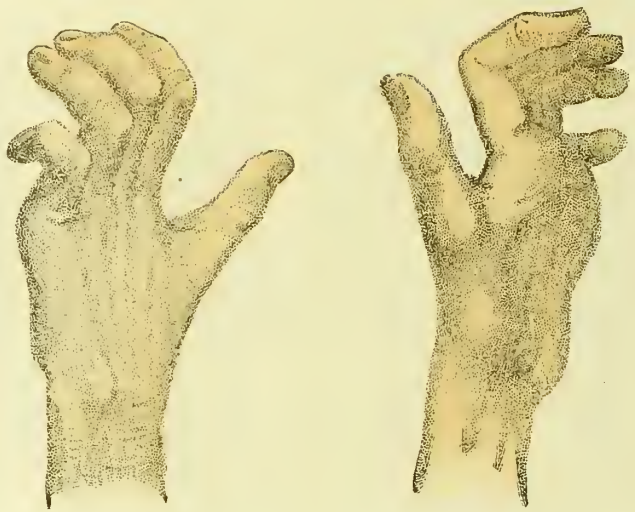


Fig. 106.—Two Views of the Hand of a Patient suffering from Progressive Muscular Atrophy.

involved in the muscle tested. The faradaic current, as well as the galvanic, will produce contractions of the affected muscle so long as any of its individual fibres escape atrophy. The "reaction of degeneration" is not observed in this disease.

In the early stages of this disease the *spinal reflexes* may be more or less increased, this being the rule for many of the so-called "wasting diseases." They are of course abolished whenever all the fibres are destroyed, and they tend to diminish proportionately to the extent of the atrophy whenever it becomes established.

Some diagnostic symptoms are frequently observed in connection with the clinical evidence of muscular atrophy. Among these the following may be mentioned as prominent:—

1. *Fibrillary Twitchings.*—These are more apparent perhaps in this disease than in any other. They are confined to the atrophied muscles. They consist in repeated and brief contractions of individual parts of the muscles. They are apt to be most marked when the muscles are tapped with the finger, subjected to a current of cold air, or faradized. They are often observed by the patient while disrobing. Occasionally, involuntary movement of the fingers, arm, or leg, may be caused by them. It is difficult to detect them whenever the integument is fatty.

FIG. 107.—PROGRESSIVE MUSCULAR ATROPHY OF ALL THE LIMBS. (After Friedreich.) The age of the patient was 45 years.

2. *Diminished Temperature in the Affected Parts.*—A peculiar sensitiveness to cold on the part of the patient is perhaps attributable to this fact.
3. *Pains* in the muscles and in the neighboring joints are occasionally observed.
4. *Deformities.*—These are due to shrinking of the muscles and the unantagonized action of unaffected muscles. The joints of the fingers may become enlarged.
5. The *skin* may appear mottled or of a bluish-red color over the wasted muscles. The epidermis may become scaly, the nails may

thicken, the growth of hair diminish, the secretion of perspiration become unnaturally excessive, and eruptions may occasionally be detected.

6. *Changes in the Pupils.*—The pupils may be unnaturally small on one or both sides, dilate imperfectly, and react slowly to light.
7. *Bulbar Symptoms.*—These indicate an extension of the spinal lesion to the medulla. Whenever the pneumogastric nerve becomes involved, death may occur from a disturbance of the heart and the function of respiration.

Diagnosis.—This disease cannot well be confounded with any other spinal affection, except perhaps one of three forms of poliomyelitis which have been described. A lesion of the ulnar nerve might create a deformity which could be easily mistaken for a progressive muscular

FIG. 108.—EXPRESSION DUE TO IMPLICATION OF THE NUCLEI OF THE MEDULLA GOVERNING THE MOUTH, TONGUE, AND THROAT. (After Hammond.)

atrophy of one extremity. Amyotrophic lateral sclerosis and lead-poisoning present points of difference which should be readily recognized.

The *deformity of the hand* which ensues from progressive muscular atrophy can be distinguished from that due to paralysis of the ulnar, musculo-spiral or median nerves by the following points: (1) the groups of muscles attacked are not supplied by one nerve, as a rule, but often by several; (2) the thenar eminence is usually attacked first, then the hypothenar eminence, and finally the interossei muscles; (3) the muscles of the arm and shoulder are liable to be attacked simultaneously with those of the hand; (4) a microscopical examination of the muscles affected reveals extensive fatty changes; (5) the so-called "bird-claw hand" is produced, which differs from that of any post-paralytic deformity.

The reader is referred to page 156 for information as to other forms of hand-distortion or the varieties of paralysis exhibited in the hand.

From *poliomyelitis* (adult variety), this disease is distinguished by the fact that no paralysis precedes the atrophy. The wasting muscles respond to faradaism as long as a fibre remains unaffected. We do not have the febrile stage nor the galvanic "reaction of degeneration," both of which are characteristic of poliomyelitis. The deformity of the hand may be identical in both diseases. For that reason the history of

FIG. 109.—PROFILE OF PATIENT SIMILARLY AFFECTED. (After Hammond.)

the case is very important, provided the patient had not been seen until the hand-deformity became well marked.

From *amyotrophic lateral sclerosis*, the diagnosis may be made by points already given on page 378.

From *lead-poisoning*, this disease is to be told by the fact that the extensor muscles of the wrist and fingers are invariably paralyzed by lead, causing the so-called "drop-wrist." Furthermore, the blue-line about the gums, the existence of attacks of lead-colic, the marked decrease in the electro-muscular contractility, and the history of exposure to lead, would be wanting in a patient affected with progressive muscular atrophy.

Progressive Facial Atrophy.—I am inclined to agree with Hammond in considering this rare condition as one of the manifestations of a condition closely allied to progressive muscular atrophy. Hence it may be well to mention it in the same connection, before the indications for treatment of the more common malady is considered.

It is more common in females than in males. Its causes are unknown. It affects adults. The pathological lesion which is associated with atrophic changes in the facial, palatine, and lingual muscles seem to be a degeneration of some of the cells which constitute the various nuclei of the medulla.

The cuts introduced here show the effects produced in a remarkable case of facial hemiatrophy. The face, tongue, and palate show atrophic changes upon the same side.

Fig. 110.—Hemiatrophy of the Face. (From a photograph.)

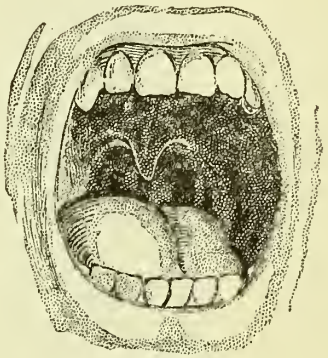


Fig. 111.—Hemiatrophy of Tongue and Palate. (Exhibited by the preceding case.)

The left side of the face is generally attacked. Why this is so I am unable to explain satisfactorily to my own mind. It is not an unvarying rule, however, since over a dozen cases have been reported where the right side was involved.

It is to be diagnosed from facial paralysis by its gradual onset, its progressive character, unimpaired electro-muscular reactions, and the incurability of the affection.

Prognosis.—Progressive muscular atrophy is, as a rule, incurable. It may run its course very slowly in some cases, and apparently remain stationary for long periods of time, but there is little, if any, hope of permanently arresting its development. Complete recovery is extremely rare. The disease usually runs its course in about five years, although

it may last indefinitely. When a well-marked hereditary predisposition to the disease is present, the prognosis is particularly grave. This disease may cause death by attacking the muscles of deglutition or respiration, and also by exciting fatal complications, such, for example, as bronchitis, pneumonia, pulmonary congestion, etc.

Treatment.—Among the various remedial agents employed in the treatment of this disease may be mentioned the following: galvanism, faradaism, franklinism, the actual cautery, phosphorus, arsenic, massage, inunctions, warm baths at the natural springs, and tonics.

In employing electricity, it is advisable to use a strong galvanic current or static sparks to the spine, and to treat the atrophied muscles with the faradaic or static current, or with alternating applications of the three. The polar method (p. 186) is commonly employed in case the spine is to be stimulated by galvanism, the neutral point being placed at the nape of the neck or over the sternum. Each application should not exceed ten minutes. They should not be given oftener than on alternate days. The secondary or induced current is preferable to the primary when the muscles are being stimulated by faradaism. The interruptions should be slow, and the current strong enough to throw each muscle into firm contractions. If the induced current fails to produce contractions, then employ the galvanic or static current. I have seen very marked benefit follow a judicious use of electricity in these cases, but I cannot say that I have ever seen a case where I believed a perfect recovery took place. Many cases of reported cures have, in my opinion, been wrongly diagnosed, or watched for too short a period. Error in diagnosis, in the case of this serious malady, is not uncommon with general practitioners, and even with some neurologists.

When a history of syphilitic infection can be discovered, it is well to put the patient upon specific treatment (according to the rules suggested on page 291).

I usually advise moderate exercise, friction of the skin, massage of the atrophied parts, and a highly nutritious diet to these patients. They should clothe their bodies warmly in silk or flannels, and avoid over-fatigue. Arsenic, phosphorus, cod-liver oil, and the bitter tonics, are often indicated. My experience with hydropathy is limited. It is claimed that great benefit has been afforded by it in some cases of this malady. The employment of baths in warm natural-spring waters affords relief to the pains and cramps in many cases, and cases of reported cures have been recorded by enthusiastic advocates of their therapeutical efficacy.

The employment of static electricity by the "direct" or "indirect spark" methods to the affected spinal segments and to the atrophied muscles has, in my experience, so far surpassed galvanism and faradaism

in its beneficial effects, that I always use it in the treatment of these cases. The machine employed for this purpose must, however, be of considerable power. (See subsequent pages which treat of this therapeutical agent.)

PSEUDO-HYPERTROPHIC PARALYSIS.

This term is applied to a rare disease of childhood which differs from progressive muscular atrophy in the following respects:—

(1) In the fact that the muscles of the lower limbs are primarily affected with atrophic changes; (2) in that the small muscles of the hand are not affected; and (3) in the circumstance that some muscles become increased in size when involved. It has been named "pseudo-hypertrophic paralysis," because the increase in the size of the affected muscles is not a genuine hypertrophy, but rather an increase in the interstitial fibrous tissue and fat of the muscles.

Morbid Anatomy.—The question of the exact pathological changes which exist in this form of disease is not yet decided, observers of equal note having differed in their examinations of the nerve-centres of patients so afflicted. Some authors hold that the disease is primarily one of the muscles, while others claim to have established the fact that the muscular changes are secondary to spinal lesions. When spinal changes exist, the motor and trophic cells of the anterior horns are usually involved.

Etiology.—An *hereditary tendency* to the disease on the maternal side seems to be more clearly proven than the exact pathological processes which cause it. Boys are more frequently attacked than girls, and the changes in the muscles usually commence before the tenth year.

Symptoms.—The symptoms of onset are very gradual, and are characterized by a weakness of the legs and a clumsiness in walking which is exhibited by frequent stumbling and falls. Gradually the patient assumes a characteristic attitude and gait. These will require a separate description.

The *attitude* is very peculiar. In the standing posture the back is thrown beyond the proper position, so that a vertical line dropped from the shoulders frequently falls behind the sacrum. (Fig. 112.) This antero-posterior curvature entirely disappears, however, when the patient is in the sitting posture. The feet are placed wide apart so as to increase the base of support. The heels are usually drawn upward by a contraction of the tendo-Achilles. In the effort to preserve the balance, the arms are held at the side with the hands extended, and the slightest touch may cause the patient to fall.

Another remarkable feature of the disease is the difficulty which is experienced in rising from the recumbent, or even the sitting posture.

The sufferer uses surrounding objects as a means of rising, drawing the body upward by the hands. When unable to reach such assistance the steps which are taken to rise are thus described by Gowers: "If laid, for example, on his back upon the floor and told to rise, he would first with great difficulty turn on his face; he would next get on his knees, his head being almost between his thighs; from this position he would gradually extend himself, so that he stands upon his feet and hands with all his limbs extended; finally he would extend the hip-joint by grasping the thigh with the hand, and pushing up the body, as it were, by the

FIG. 112.—SIDE VIEW OF ATTITUDE OF PSEUDO-HYPERTROPHIC PARALYSIS. (Duchenne.)

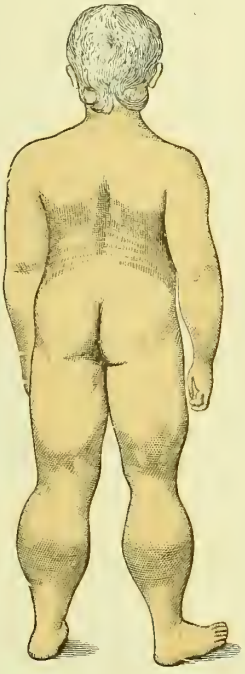


FIG 113.—REAR VIEW OF ATTITUDE OF PSEUDO-HYPERTROPHIC PARALYSIS. (Duchenne.)

arm." This movement of "climbing up the thighs," as it has been termed, is an indication of weakness in the muscles which straighten the knee, and also those which extend the trunk upon the thigh,—the extensors of the hip-joint. (See Figs. 52 and 53.)

The *gait* of these patients is associated with an oscillation of the body from side to side, or a waddling movement. The advance made at each step is very small, and a difficulty seems to be experienced in flexing the thigh upon the abdomen.

The *muscles of the calf* exhibit early a firmness and increase in size which is not proportionate to their motor force,—as that is far below

normal. Soon they become excessively developed, as do also those of the buttock, while the other muscles of the leg commonly grow smaller from atrophic changes.

The *latissimus dorsi* and the lower part of the *pectoralis major* muscles exhibit marked wasting in a very large percentage of cases. In some instances all the striated muscular fibres of the body, including even the heart, may become affected.

The *electric reactions* of the muscles to faradaism are markedly diminished.

The *spinal reflexes* are diminished in the early stages, and, later on, are abolished. The bladder, rectum, and the sensory functions of the skin are not interfered with as a rule.

Diagnosis.—The diagnosis of this affection cannot easily be confounded with that of any other disease. A simple hypertrophy of muscles, as a result of increased vascular supply, could easily be distinguished from it by the aid of Duchenne's trochar and a microscope The symptoms of muscular weakness and the characteristic gait and attitude would be wanting, moreover, in case the muscles had undergone a genuine hypertrophy.

Prognosis.—Although cases of recovery have been reported, the prognosis of this disease is unfavorable. Life may, however, be prolonged for many years, and death usually occurs as the result of some intercurrent affection.

Treatment.—Authorities agree in the statement that electricity forms the chief therapeutical agent in the treatment of this malady. Duchenne employed the faradaic current in the two cases of cure reported by him. They were subjected to its influence from the incipiency of the disease. Hammond recommends the use of galvanism to the spinal segments which are related to the muscles affected, and faradaism of the muscles themselves with as strong a current as the patient can bear. Massage and the external use of heat have proved beneficial. Strychnia, phosphorus, and iron may assist, on account of their tonic properties, in promoting the general vigor. Static electricity may be substituted for faradization of the muscles. It is best applied in the form of the "spark" and the "static-induced" current. (See subsequent chapter.)

SCLEROSIS OF THE POSTERIOR COLUMNS.

(*Progressive Locomotor Ataxia—Tabes Dorsalis—Gray Degeneration of the Posterior Columns.*

This is an extremely common spinal affection of a chronic type. It is characterized, when fully developed, by a difficulty in walking, and, in some cases, by an imperfect use of the upper extremities, defects in the

visual apparatus, and marked disturbances of the sensory functions and visceral reflexes. There is no actual loss of muscular power; but, because the muscles of the limbs are imperfectly coördinated, the existence of paralysis is often erroneously suspected.

Morbid Anatomy.—The spinal lesion which is characteristic of this disease is confined chiefly, but not exclusively, to the sensory columns of the cord. (See Fig. 115.) For this reason we should expect to encounter during life well-marked disturbances of sensation, and also

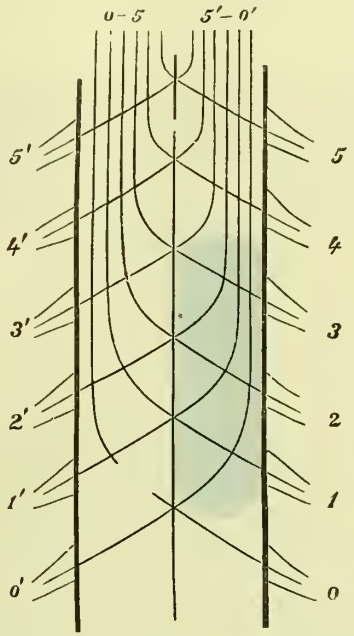


Fig. 114.—A Diagrammatic Representation of the Lesion of the Sensitive Tracts in an Unilateral Lesion of the Right Side, to the Extent of the Colored Space. (After Erb.) All sensitive tracts of corresponding height, as well as those coming from farther back, on the left side, are interrupted. On the right side only those are interrupted which enter by the roots from 1 to 3. Numbers 0 to 5 represent the sensitive tracts of the right side, entering with the posterior roots; 0′ to 5′ the same on the left side.

incoördination of movement. The clinical history of this disease fully sustains such conclusion.

To the naked eye the spinal cord may, in exceptional cases, appear normal, although the microscope may reveal marked pathological changes. As a rule, the posterior part of the cord is more or less flattened and somewhat narrower than normal. The consistency of this part of the cord is usually increased. The pia is generally more or less thickened and opaque over the posterior columns of the cord. It may also be pigmented and adherent both to the cord itself and to the

arachnoid and dura. Calcareous plates may be found in the arachnoid and the dura.

The most marked changes are usually observed in the lower dorsal and upper lumbar segments of the cord. In these localities, the columns of Burdach are usually most affected, but in the cervical segments the columns of Goll are generally very markedly sclerosed.

The posterior nerve-roots are usually more or less atrophied as far as their ganglia.

In exceptional instances, sclerotic changes have been observed in

(Cut Colored.)

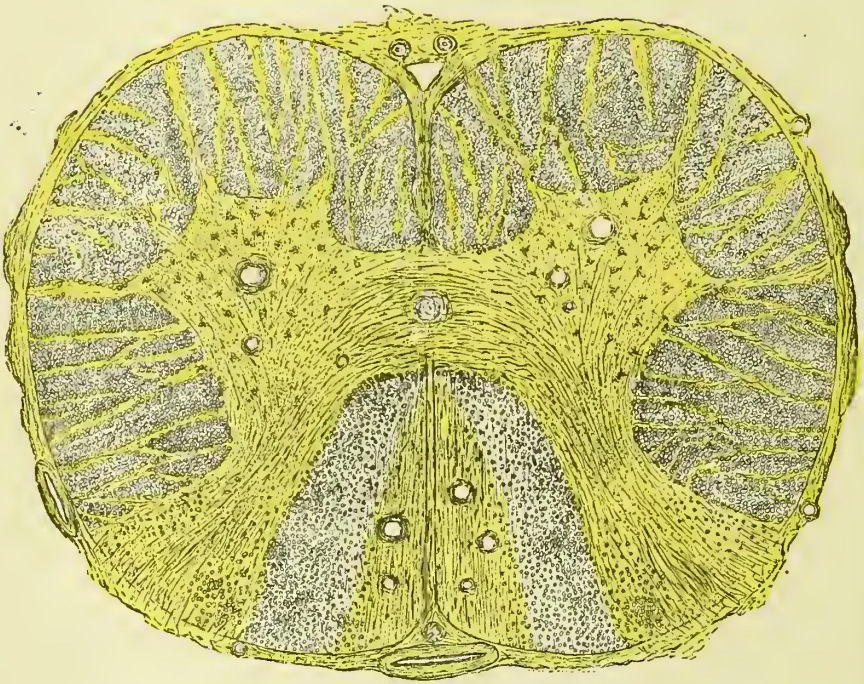


Fig. 115.—A Transverse Section of the Spinal Cord of an Ataxic Patient in the Upper Lumbar Segment. (Weigert's staining—25 diameters.) Attention is called to the thickening of the coats and the expanded lumen of the posterior spinal artery; the thickened coats and contracted lumen of the anterior spinal vessels; the thickened coats and altered lumen of the vessels in the spinal substance; the diffused sclerosis in the anterior and lateral columns of the cord; the extensive sclerosis and disappearance of the nerve-fibres in the postero-internal column; and the very marked hypertrophy of the meninges. This beautiful drawing was made by Dr. Van Schaick, of New York, from a section of a typical ataxic cord.

the medulla, the pons, the corpora quadrigemina, and some of the cerebral nerves. Among the latter, the nerves connected with the eye are more commonly affected.

A microscopical examination of the diseased area in the cord shows a disappearance of the nerve-fibres and an excess of connective tissue

which is poor in cells. The walls of the blood-vessels are usually markedly thickened. Their walls may also contain an excess of nuclei. The medullary sheath of the nerve-fibres may have undergone absorption, leaving naked axis-cylinders.

Occasionally, and perhaps more frequently than most authors seem to believe, an increase of connective-tissue may be detected in the vesicular columns of Clarke and in other parts of the cord; but the cells of this column are usually unaffected.

Respecting the exact nature of these changes and the causes which induce them, authorities of note differ. Some regard them as inflammatory, others as the result of a primary non-inflammatory degeneration of the nerve-fibres; while a few consider this disease as the result of primary changes in the blood-vessels. An inflammation of the pia and arachnoid may possibly act as an exciting cause, according to some published observations.

Respecting the pathology and etiology of locomotor ataxia, my friend, Prof. William H. Porter, has lately called my attention to some pathological facts which his extended investigations lead him to regard as of greater importance than is usually attached to them. The beautiful drawing of Dr. Van Schaick (Fig. 115) is taken from one of Dr. Porter's sections, and serves to illustrate the pathological changes encountered in locomotor ataxia.

Dr. Porter's views may be summarized as follow:—

(1) That in ataxia, there is found after death in every case a *thickening of the coats of the larger vessels with an alteration in their calibre.* This he believes is generally due to syphilis.

(2) That the *lumen of the posterior spinal artery* is always greatly in excess of the normal, while that of the anterior spinal vessels is generally below the normal size.

(3) That a chronic hyperæmia of the posterior columns of the cord is thus primarily induced.

(4) That excessive nutrition induces an abnormal development of connective tissue in the spinal cord.

(5) That the complete organization of this newly-formed tissue is followed by its contraction, which, in turn, causes atrophy and destruction of the nervous elements of the spinal cord.

That this observer's views may be accurately presented I quote from a communication received lately from him, in which he says:—

When we study carefully the anatomical arrangement of the arterial system distributed to the spinal cord, we find that the anterior spinal artery is formed (by a branch from each vertebral artery) near the junction of the pons and medulla. From this point it passes downward upon the anterior surface of the spinal cord, until it reaches the third or fourth cervical vertebra, where it is lost as a distinct and separate vessel. Here it forms

a plexiform net-work with the lateral spinal vessels, which constitute the source of nutritive supply upon the anterior surface of the cord from this point to its termination. On the posterior surface of the cord the posterior spinal artery, arising at nearly the same point, descends as a distinct vessel, as far as the second lumbar vertebra, and then divides into numerous branches to the cauda equina.

From this arrangement it is quite apparent that the nutritive supply of the postero-internal and postero-lateral columns is directly from this posterior spinal artery, while the anterior and lateral columns have a more general nutritive supply.

Physiology teaches that with a high tension and increased rapidity of the blood-current, nutrition is diminished; but with a moderate or low arterial tension a larger volume of blood comes in contact with the tissues, and a longer period occurs in which it is in contact with the tissues. By this alternation in the nutritive supply the various changes are compensated for, and a normal standard is maintained. Whenever, for any reason, we have this dilatation unduly prolonged, and the increased nutritive supply constantly above normal, we are apt to have a development of new connective-tissue cells and basement substances (too often called an inflammation), without the production of serum, fibrin, or pus, but with the development of new histological elements.

With these anatomical and physiological facts clearly in mind, we are in a position to study the vascular changes in locomotor ataxia, and possibly to explain the reason for the major part of the lesion being located in the posterior or postero-lateral columns; also the more or less general sclerosis in the superior portion, and throughout the cord as the disease advances.

None seem to deny the common if not universal vascular changes in this affection. In Cornil and Ranvier's "Pathological History," the following statement is made: "A peculiarity of these scleroses consist in a thickening of the walls of the capillaries and small vessels." But thus far no one of note appears to have attached any special importance to these vascular changes as a causative agent in producing this disease, or as an explanation for the localization of the lesion in the postero-external and postero-internal columns of the spinal cord.

The following anatomical conditions have been found in all the cases examined by me. In some of the cases examined the history was of short duration, while in others the disease had existed from eight to ten years. There was a marked thickening of the pia-mater of the cord, which is well illustrated in the drawing which was kindly made for me by Dr. George G. Van Schaick. This thickened membrane was often firmly adherent to the spinal cord, especially at the sclerotic zone, and occasionally also to the dura-mater.

The vascular changes in all the cases examined by me constituted the first and most apparent lesion. It was in the form of a peculiar or hyaline thickening of the walls of the smaller arteries and arterioles. In some instances this caused a marked thickening of the wall with an increased lumen, while in others a marked decrease in the lumen was found.

A noticeable feature in all the cases examined was the marked changes in the posterior spinal artery. It was found in every instance to have its coats thickened and its lumen expanded. On the other hand, the vessels of the anterior portion of the meninges were thickened and their lumen contracted. [These vascular changes are well illustrated in the accompanying drawing. (Fig. 115).—Author's Note.]

When the arterioles are thickened in this way they undoubtedly lose their power to contract and expand to meet the varying changes of nature, and thus to regulate the nutritive supply to be distributed to the various parts and organs of the body. In this particular lesion the posterior spinal artery being thickened and its lumen expanded, the posterior columns of the cord receive an increased quantity of the nutritive material.

Ultimately this condition results in a hypernutrition, the development of new connective tissue corpuscles, and new basement substance. According to the law of all newly-formed connective tissue, contraction follows and thus causes a compression-atrophy, and ultimately a disappearance of the nerve-fibres in the sclerotic zone.

Owing to the irregular thickening and dilatation of the posterior spinal artery and its small branches (especially the latter), we find in some the sclerotic condition most marked in the posterior external columns; in others, in the posterior internal or the columns of Goll. In fact, in the same cord, the lesion may be also observed most marked in one column at one part, and in another column at another part; but in all cases the vessels immediately supplying the sclerotic patch are invariably found thickened and dilated, while in the unaffected portions they are thickened and contracted, or nearly normal in appearance.

In the cord from which this drawing was made the sclerosis in the inferior lumbar region was in the posterior external columns; but higher up in the cord it was distinctly in the columns of Goll, as represented in the drawing.

As there is an increased rather than a decreased nutritive supply, there is no primary change in the ganglion cells in ataxia. They only suffer whenever the new-formed connective tissue becomes sufficiently abundant to cause a compression of the cells. When this stage is reached, some of the cells may be found in a state of atrophic degeneration.

At the anterior and lateral portions of the cord (the vascular supply being in these regions more general) the increase in new-formed or sclerotic tissue is more surely developed, forming either a general or patchy sclerosis, which is never confined to a distinct column.

In the medulla oblongata this general sclerosis is apt to be found throughout the section. Taking this view of its development, we may say that the disease is caused by these vascular changes. In my opinion, these are primarily due to syphilis in a majority of the cases, if not in all. I believe that the disease is non-inflammatory, but one due to a prolonged and increased nutritive supply. Finally, it is generally conceded that the change in the nerve-fibres and ganglion cells are secondary, and the result of simple compression.

The insidious development and slow progress of this disease argues strongly against the lesion being inflammatory in the generally-accepted sense of acute vascular changes, with an exudation of the liquor sanguinis, and a migration of the blood-corpuscles.

Etiology.—This disease may be congenital or acquired. If acquired, it may follow traumatisms of the spine, exposure to cold or dampness, sexual excesses, syphilis, and some acute blood-diseases. When congenital, it is commonly associated with a syphilitic diathesis, although not necessarily dependent upon it.

The question whether syphilis can or can not be included among the etiological factors of this disease is still an open one. There can be no question regarding the statement that this disease is often encountered in patients who give a positive history of syphilitic infection; but this fact does not in itself prove that the ataxia is a result of the acquired syphilis. The coexistence of the two conditions may be a mere coincidence. Those who combat the idea that a syphilitic origin of ataxia is ever present, advance the following grounds for their belief: (1) that the records from which statistics that support the opposing view are drawn

are very unreliable and often worthless; (2) that the statistics of ataxia published by different compilers show a percentage of previous syphilis that fluctuates between 4 per cent. and 90 per cent. of the collected cases (extremes which cannot be reconciled); (3) that syphilitic lesions of nerve-centres do not tend to follow definite nerve-tracts (systematic lesions), and that the opposing view is in opposition to all established pathological data relating to syphilis.

On the other hand, it is urged: (1) that late statistics (from which all elements of error have been carefully expunged) show a percentage of syphilis too large to be classed as a mere coincidence; (2) that specific treatment often relieves the ataxic symptoms to a marked degree; and (3) that our knowledge of syphilitic processes in the nerve-centres is as yet imperfect.

Personally, I am rather inclined to exclude the recognition of ataxia as a syphilitic disease, *per se*, in any case. I have never seen a cure follow the most active anti-syphilitic treatment. I am, however, open to conviction when the disputed points regarding it are involved in less obscurity than I think they are at the present time.

This disease is more common among males than females. It is usually encountered during middle life. It is very rare during childhood, and after the fiftieth year.

Acute infectious diseases are sometimes followed by the typical spinal changes observed in this condition. It is claimed that ataxia may also occur as a result of chronic ergot-poisoning.

Heredity can unquestionably be shown to exist in certain cases. In one family eighteen cases of well-marked ataxia were observed, according to Carré.

Progressive paralysis of the insane is not infrequently accompanied or preceded by the symptoms of this disease.

Symptoms.—The symptoms of this chronic disease* extend over so long a period of time (five to thirty years) that they have been commonly divided into three stages, viz., those of invasion, incoördination, and the stage of complications.

The STAGE OF INVASION is characterized by the following sensory phenomena: (1) pains of a peculiar character, which will be described later; (2) hyperæsthesia of the skin; (3) anæsthesia of the skin; (4) retarded conduction of sensation; and (5) peculiar subjective sensations in the parts associated with the affected spinal segments. These are commonly observed in the lower extremities, because the sclerosis is confined, as a rule, to the dorsal and lumbar regions of the cord. If the disease has progressed to the cervical region, the upper extremities will

*I quote from my work on "Surgical Diagnosis" the paragraphs relative to the symptoms of this disease, subject to some slight changes.

be similarly affected, and in addition the pupils will be contracted and fail to respond to light. (P. 120.)

In addition to the sensory phenomena described, the *spinal reflexes* (especially the patella reflex or knee-jerk) will as a rule be diminished or abolished.

The pains of this disease are to be differentiated chiefly from those of rheumatism and neuralgia. They are pathognomonic of the first and second stages of ataxia, and may persist even in the third. They are best described as "lightning pains," which are of a stabbing or boring character and extremely severe. They are paroxysmal and of momentary duration. Some patients compare them to an electric shock, which gives a sensation as if a knife were run into the muscles or a joint. Unlike the pains of rheumatism, with which they are often confounded, they attack circumscribed areas which are usually between joints, and successive paroxysms seldom occur in the same spot. After the attack of pain has subsided, the skin over the seat of pain is extremely sensitive to the touch. They differ from the pains of neuralgia in that they do not follow the course of individual nerve-trunks; they are not associated with the so-called "puncta dolorosa" or points of sensitiveness to pressure along the course of the affected nerve, as is the case with neuralgia; they do not radiate in the superficial area of distribution of any special nerve-trunk; they are not commonly referred to the skin, but to the deeper structures. Finally, they are most frequently confined to the lower extremities or to the pelvic organs,—chiefly the bladder and rectum.

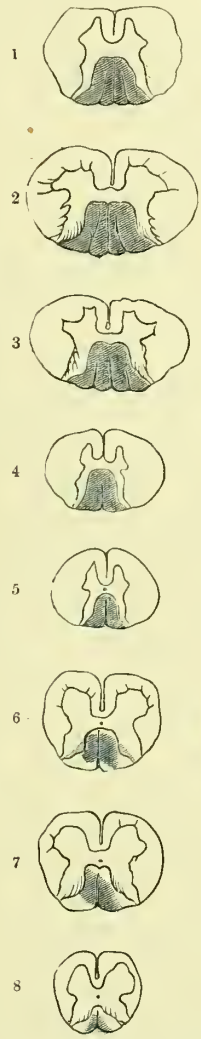


Fig. 116.—A Diagrammatic Representation of the Pathological Lesion of the Cord observed in Locomotor Ataxia (After Erb.) Note that the diseased portions are represented as confined to the posterior columns (those of Goll and Burdach) exclusively.

These lightning pains are so severe and constant in some instances as to induce suicide; while in less severe cases the paroxysms are infrequent and easily controlled by anodynes. They may affect the stomach and be mistaken for those of dyspepsia; or the rectum, and be confounded with hæmorrhoidal disease; or the bladder, and suggest the possible existence of a

calculus or cancer of the bladder. As the paroxysms of pain in some cases not infrequently occur at long intervals for months or years before the evidences of incoördination of movement appear, it is common for the specialist to meet with these patients after an incorrect diagnosis (usually of rheumatism or neuralgia) has resulted in a long-continued and ineffectual course of medication on the part of many medical advisers.

Hyperæsthesia of the skin (especially at the seat of pain after the paroxysm has passed away) is commonly met with in these cases. Like the pain, this increased sensitiveness is fugitive.

Anæsthesia of the skin to the sense of touch, pain, or temperature, is more or less completely developed as the disease progresses. These patients often experienced, as a result of this anæsthesia, subjective feelings which are peculiar; for example, as if the feet were cold or numb; or again, as if the patient was standing upon feathers or wool, or a pebble was in the shoe.

Delayed Conduction of Sensation.—When the sclerosis of Burdach's column has progressed to an extent sufficient to interfere with, but not arrest, the conduction of sensation to the brain through the spinal cord, a symptom is produced which is of great clinical value, viz., the occurrence of a *perceptible interval of time* between the infliction of a wound (a pin-thrust, for example) or a simple tactile impulse and its perception by the patient. In making this test, it is well to have the patient close the eyes so as to obviate all danger of seeing the test applied. I have met with one case where the interval was ten seconds, and a much longer interval than that has been recorded.

The STAGE OF INCOÖRDINATION OF MUSCULAR MOVEMENTS follows after a lapse of time, which varies between the extreme limits of a few months and many years. Incoördination is usually first exhibited in the gait, because the lesion is confined primarily to the dorsal and lumbar regions of the cord in the great majority of cases. When the cervical region becomes involved, the upper extremities also exhibit a marked impairment of coördinated movements,—as manifested, for example, in the efforts to button and unbutton the clothing, to carry the hand with accuracy and rapidity to designated parts of the face or body when the eyes are closed, and in writing letters with continuous curves.

The *gait* of ataxic patients (although walking is extremely difficult in some cases) is not that of paralysis. The two should not be confounded with each other. In this disease, the motor power of any individual muscle is not impaired, nor do the limbs usually show any lack of development. It is only when some act is demanded which involves a coördination of muscular movement, or, in other words, where several muscles must be employed in some special order, that the difficulty

becomes manifest. Too much emphasis can not be laid upon this distinction between incoördination and a loss of muscular power.

Among the early symptoms of incoördination which the patient experiences may be mentioned a difficulty in performing feats of locomotion when suddenly called for, as in hastily crossing a street, climbing a flight of steps, or washing the face with the eyes closed. Under such circumstances a sense of insecurity first dawns upon the patient and causes him to avoid such acts. Gradually he finds it necessary to stand with

Fig. 117.—Extensive Joint-Changes in Connection with Locomotor Ataxia. (Charcot.)

the feet apart to increase his base of support; to keep his eyes upon the ground when walking, so as to use the visual sense in directing his movements, and to employ canes to aid him in preserving his balance. Such patients have great difficulty from the first in placing the feet upon small objects, as in mounting into a saddle by means of the stirrup, climbing ladders, or in getting upon a chair to reach some object.

When incoördination becomes still more impaired, the patient walks slowly and with great deliberation. The feet may be suddenly jerked outward, the heel generally strikes the ground before the sole of the foot,

causing the peculiar "stamping gait;" and sometimes the movements of the legs are so unexpected that the patient falls to the ground.

When the *upper extremities* become similarly involved, the hand cannot be rapidly carried to designated portions of the body, such, for example, as the nose, mouth, ear, shoulder, etc., when the patient is instructed to do so, with the usual accuracy, provided that the eyes are closed. The clothing is buttoned and unbuttoned with great difficulty. When the patient is asked to convey a glass of water to the lips, it is raised to the proper level, held there for an instant with the eyes fixed upon it, and then darted suddenly toward the mouth. The handwriting becomes markedly altered (especially when the eyes are closed), because all continuous curves are made with less ease than straight lines, if not too long.

The *pupils* frequently show the preternatural contraction which indicates that the *cilio-spinal centre* is implicated; and they also fail to *properly respond to light*, although they do move when vision is adjusted for a proper focus upon objects in proportion to the distance of the object from the eye, when within a space of less than twenty feet. This condition constitutes the so-called "Argyll Robertson's pupil." The tests for it have been already described. (P. 120.)

The various tests employed to determine the ability of any given subject to coördinate the muscles have been quite fully described in a preceding chapter. (P. 179.)

In rare cases, the loss of coördination affects the muscles which move the spine.

The *spinal reflexes* are greatly altered in this disease, and form an important factor in the diagnosis. The knee-jerk is usually abolished at an early stage. Tickling the soles of the feet generally fails to elicit the so-called "plantar reflex."

In the LAST STAGE, that of COMPLICATIONS, the bladder, rectum, sexual organs, eyes, bones and joints, skin and stomach, are liable to give more or less constant evidence of effects of the spinal disease upon them.

The *bladder*, which in the earliest stage was affected by the lightning pains, accompanied often by hasty urination and pain during the act, gradually exhibits the symptoms of paresis in slow micturition, dribbling, or incontinence. The latter symptom occurs as the result of retention of residual urine, which overflows at last when the distended bladder cannot longer control its escape. This condition should not be allowed to go unrelieved from a neglect to insert a catheter. Finally, cystitis is often produced; and, in rare instances, anæsthesia of the urethra is observed.

The *rectum*, which in the early stages is frequently affected with stabbing pains and a painful sense of excessive distension, suffers a

diminution in its reflex action later in the disease, which induces constipation. In the final stage, paralysis of the organ may be developed, and anæsthesia of its mucous lining is observed in exceptional instances.

The *sexual organs*, which in the early stages are often abnormally stimulated (the sexual desire being increased, but the act itself being more or less imperfect), are very much impaired in the advanced stages of the disease; the sexual appetite as well as the power of erection being often completely abolished.

The *organ of vision* suffers late changes in locomotor ataxia. The peculiar alterations of the pupil, first described by Dr. Argyll Robertson, have been already mentioned. In addition, ptosis, strabismus, diplopia, and loss of color perception are not infrequently recorded as complications of this disease. Atrophy of the optic nerve may also be one of the results of the first stage. Some authorities lay great stress upon the diagnostic value of the ophthalmoscope as a means of detecting this early symptom of locomotor ataxia.

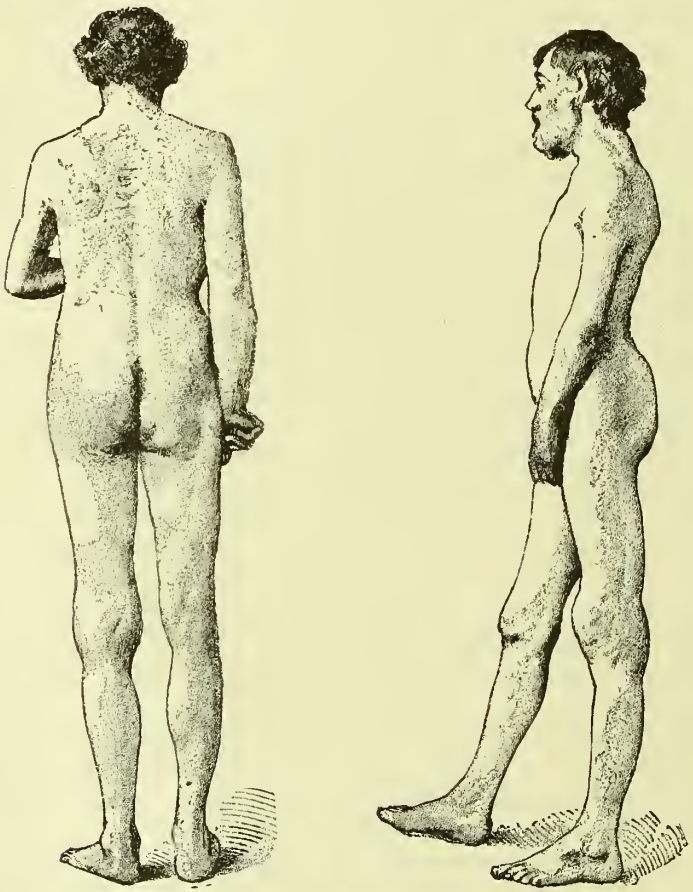
Charcot's Disease.—The *bones* and *joints* may become disintegrated, as a late result of this form of spinal disease, and deformities are thus produced. Charcot was the first to describe these conditions, and we owe to him much of our knowledge of the subject. The larger joints, such as the knee, shoulder, and elbow, are the most liable to be affected. Females are most frequently attacked.

The changes which occur are to be differentiated from those of chronic rheumatic arthritis by the following peculiarities: (1) that they are acute; (2) that they involve large joints and infrequently the hip, whereas the reverse is true of rheumatic arthritis; (3) that effusion into the affected joint is extensive, whereas the effusion is rare in rheumatic arthritis; (4) that dislocations are spontaneously produced, while they are extremely rare in rheumatic arthritis; (5) the changes are usually painless, probably because analgesia is associated with them; (6) sometimes spontaneous fractures occur. The most plausible explanation of these bone changes, to my mind, is that advanced by Buzzard, viz., that they are due to changes within the medulla oblongata.

Considerable discussion has taken place (chiefly of late in the London Clinical Society) relative to the pathological position and relations of "Charcot's disease." It was claimed by some eminent authorities at that time that the disease was but a type of chronic rheumatic arthritis. With this view I cannot agree. The points already mentioned seem to me to confute this deduction. I have personally seen three cases of a typical character, and I present a photograph of one. Each of these patients was afflicted with ataxia before the joint complications. Large joints (the knee in two cases and the elbow and both knees in one) were attacked. The small joints were normal. The disorganization of

the affected joints was rapid and so extensive as to result in a "flail-like" mobility. The morbid changes were *absolutely painless.* The *knee-jerk was absent* in every case.

Buzzard states that in Charcot's disease the Haversian canals are dilated; that the bones are thinned and eroded; that the inorganic



FIGS 118 AND 119.—CASE OF "CHARCOT'S DISEASE" OF LEFT KNEE-JOINT FOLLOWING LOCOMOTOR ATAXIA. (From photographs in the possession of the Author.)

constituents of the bones are diminished and replaced by fat; and that probably the specific gravity of the affected bones would show a decrease from that of those unaffected. In these respects, the morbid changes are totally at variance with those of rheumatic arthritis.

In connection with the changes in the bone and joints, *gastric complications* sometimes occur. They are characterized by attacks of

vomiting and severe gastralgia. They are more commonly observed in females than in males.

Finally, the *skin* may exhibit trophic disturbances in the form of herpetic eruptions and bed-sores.

Eichhorst has contrasted the percentages of the more important symptoms of locomotor ataxia, according to the deductions given by Bernhardt and Erb, in the following table:—

SYMPTOM.	AUTHORITY QUOTED.	
	BERNHARDT. (58 Cases.)	ERB. (56 Cases.)
1. ABSENCE OF PATELLAR TENDON REFLEX	100.0 per cent.	98.0 per cent.
2. LANCINATING PAINS	79.5 "	92.5 "
3. PARESIS OF BLADDER	74.1 "	81.0 "
4. PARALYSIS OF OCULAR MUSCLES	39.6 "	38.7 "
5. MYOSIS	27.2 "	54.0 "
6. RIGIDITY OF PUPILS	48.4 "	50.0 "
7. OPTIC ATROPHY	10.3 "	12.4 "
8. SENSORY DISTURBANCES	85.9 "	
9. ANALGESIA	31.6 "	69.0 "
10. DELAYED CONDUCTION OF PAIN	34.4 "	89.5 "
11. BRACH-ROMBERG SYMPTOM (swaying and tottering when eyes are closed.)	90.2 "	83.5 "
12. FEELING OF EXHAUSTION	92.0 "	97.9 "
13. ATAXIA	94.1 "	100.0 "
14. SEXUAL WEAKNESS	43.7 "	78.5 "
15. JOINT AFFECTION	9.6 "	
16. GASTRIC CRISES	5.2 "	

It will be observed that a wide difference exists respecting the relative frequency of symptoms 2, 5, 9, 10 and 14 of the table. Future investigation will probably tend to determine these percentages with greater accuracy.

The patellar reflex is being quite extensively investigated at the present time. It may be absent in apparently healthy subjects. It has been estimated that about *two per cent. of all adults fail to exhibit the knee-jerk.* Such subjects very frequently belong, however, to families which exhibit a strong neuropathic predisposition. The knee-jerk is liable to disappear in advanced age, and in subjects suffering from chronic alcoholism it may occasionally be totally absent. It has been suggested that an abnormal length of the ligamentum patellæ may exist in healthy subjects where the knee-jerk cannot be elicited. Such a condition would prevent the ligament being put upon the requisite tension.

The effects of locomotor ataxia upon the sexual functions are

usually manifested at first by an increase of desire, with hasty ejaculation of semen. Later on, frequent seminal emissions are apt to occur spontaneously, and impotency generally follows. I have observed exceptions to this rule, however, in several cases.

Respecting the diagnostic importance of *ataxia* as a symptom, the fact should not be lost sight of that lesions of the cerebellum and of the "lemniscus tract" within the pons or medulla may create incoördination of movement, and also that acute infectious diseases may occasionally tend to induce it.

Diagnosis.—Locomotor ataxia may be, and too frequently is, mistaken in its *initial stage* for rheumatism and neuralgia. Occasionally the abdominal crises of ataxia may cause the physician to suspect the existence of visceral derangements. The points by which the pains of ataxia may be distinguished from those of other affections of a painful character have been given already (p. 397).

In its *second stage*, locomotor ataxia must be distinguished from motor paralysis, spinal meningitis, cerebellar ataxia, hysterical manifestations, chronic myelitis, and multiple sclerosis of the cord.

The symptoms known as those of "*Charcot's disease*" must be diagnosed from those of chronic rheumatic arthritis.

Motor paralysis can readily be distinguished from ataxia by the gait (p. 164) and by testing the power of individual muscles. In ataxia there is no diminution of motor power, as may be demonstrated when the muscles are subjected to proper tests. There is no incoördination of movement when motor paralysis exists. The nutrition of paralyzed muscles is generally impaired. This is not the case in ataxic subjects.

Spinal meningitis can be recognized by the existence of pain on motion of the spine and when pressure is made over the spinous processes, both of which tests are negative in ataxia. Moreover, febrile disturbances and more or less motor paralysis are liable to accompany meningitis. The reflexes will be normal or exaggerated in meningitis; ocular symptoms will not be discovered; no incoördination of movement will be developed; nor will the clinical history of the two diseases be alike.

Hysterical ataxia may be recognized by the history of the case and the condition of the reflexes. If the patient be a male, hysteria can generally be excluded.

Cerebellar lesions produce a peculiar gait and attitude which closely resembles that of ataxia in some respects. It has been described on a previous page (p. 165). Vertigo is a symptom of cerebellar lesions, and gastric crises which are attended with vomiting are frequent. Rotary movements may be developed. The reader is referred to page 76 for further information respecting cerebellar lesions.

Chronic myelitis is to be distinguished from ataxia by peculiar combinations of sensory and motor phenomena, which are developed during the progress of the inflammatory affection; by the usual absence of incoördination of movement; and by the presence of spasms, contractures, bed-sores, cystitis, vesical paralysis, febrile disturbances, etc. The pupils are seldom affected, nor are typical ataxic pains ever encountered in myelitis.

Prognosis.—It is my belief that some cases of ataxia may be cured. The majority are usually capable of being greatly relieved by proper treatment, although some are not. If it proves fatal, it generally does so by the aid of an intercurrent affection. The duration of the disease can hardly be estimated. I have one patient now under my care who gives a clear history of ataxic pains for the past thirty years. Sometimes the second stage of the disease is never reached. In rare instances, the progress of the disease may be rapid. In one of my cases, general paresis set in after fifteen years of suffering. He is now in an insane asylum.

Treatment.—In the *early stages* of locomotor ataxia, it is my custom to place the patient upon the internal administration of hot water (p. 248), and to give ergot in large doses (one drachm of the fluid extract three times a day). I sometimes administer the bromides of potash, sodium, or calcium, in place of the ergot. Three times a week or more I usually employ heavy static sparks to the spine and muscles, and I employ also the actual cautery to the spine very actively at intervals. In this way, I have repeatedly caused a total arrest of the pains, which had been very frequent and severe up to the date when this treatment was begun. In a few instances a cure seems to have been accomplished. The knee-jerk has returned, and all symptoms of the disease have disappeared.

During the *paroxysms of intense pain*, hypodermic injections of morphine, or the internal administration of codeine are of service.

When a *distinct syphilitic history* can be obtained from the patient, I give the iodides and mercurial baths as an adjunct to some of the other agents mentioned.

When the *stage of incoördination has been reached*, the treatment must be somewhat modified. The bromides are of no service. The ergot may be continued, but the nitrate of silver in one-third grain doses should be administered in conjunction with it three times a day. Care should be exercised against continuing the administration of this agent long enough to cause staining of the skin. The electrical* and cautery applications should be regularly employed. Belladonna is of benefit when the bladder becomes affected. Hammond recommends the

* My experience with galvanism has not been as satisfactory as with the static spark.

hypodermic use of atropia in doses of one hundred and twentieth of a grain at first, the dose being gradually increased until one-thirtieth of a grain is administered.

The *employment of crutches* when walking, in order to prevent excessive use of the muscles, has produced beneficial results in the experience of some authors. I am inclined to think that the suggestion is a good one. They certainly aid the patient in walking.

The question of the utility of *stretching the sciatic nerves*, as first suggested by Langenbeck in 1879, for the relief of incoördination and the pains of ataxia is still undecided. Some remarkable results have been accomplished by this proeedure, and also some remarkable failures. The nerve is exposed by an incision made above the popliteal space. The finger should then be introduced beneath it, and the limb raised by means of the nerve, thus subjecting it to a tension sufficient to stretch it.

By means of the different methods of treatment suggested, incoördination of movement and the paroxysms of pain may be very markedly decreased in the majority of cases. One patient, for example, was lately placed under my care. He came to my office at first in a carriage, and was able to ascend the steps of my residence only by the aid of two canes and a body servant. In less than three months he walked alone to my house with only one cane, the servant having been dispensed with. Another had his pains (which were typical and of daily occurrence) arrested for nearly six weeks by six applications of the static spark.

Incontinence of urine is sometimes a serious complication of ataxia. As a rule, it is properly an overflow from a highly-distended bladder, rather than a true incontinence. It is always well to remember this fact, and to introduce a catheter into the bladder at once when this symptom is presented by an ataxic patient If the bladder be found to be distended with retained urine, catheterization should be employed several times each day at regular intervals, and the bladder washed out with care each day. This can be easily done by the patient himself by attaching a soft-rubber catheter to a fountain syringe. Raising the fountain fills the bladder, and lowering it siphons the fluid back into the rubber bag.

I recall a case, which was sent to me some three years ago, in which the patient (an ataxic) had worn a urinal in his trowsers-leg for many months, at the advice of a physician. When I introduced a catheter, over a quart of urine was taken from the over-distended bladder. The treatment mentioned above cured this symptom in less than a month, and the patient was relieved of a source of great annoyance and mortification.

CENTRAL MYELITIS.

In a previous table, I have seen fit to classify inflammation of the central gray matter of the spinal cord as a systematic lesion. I am at variance with some authors in so doing. The other varieties of myelitis, with the exception of poliomyelitis, should unquestionably be included under the head of focal spinal lesions, because they tend to spread transversely and to involve, as they progress, one column of the cord after the other. In this respect central myelitis seems to be an exception. It tends to spread chiefly both up and down the cord, and transversely to a limited extent only.

Morbid Anatomy.—This form of myelitis is a rare one. It has been known to extend throughout the entire length of the spinal cord. It may extend also to the anterior or posterior horns of the spinal gray matter. Its pathology does not differ from that of ordinary myelitis.

Etiology.—Little is positively known respecting the exciting causes of this peculiar form of myelitis, or the physical condition which particularly predispose to it. The remarks which I shall make later relative to the causes of myelitis are probably applicable to this condition.

Symptoms.—These will be modified (as might be inferred from the statements made in the early pages of this section) by the extent of the lesion. The combination of symptoms which the patient is liable to present may be an exceedingly complex one. All the disorders of sensibility mentioned on a preceding page (p. 354), as well as paralyses of motility of various types and degrees, with or without contracture, may be observed. Whenever the anterior horns are attacked, more or less atrophy of muscle may accompany or follow the clinical evidences of impaired motility.

The disease is essentially a chronic one; hence, sufficient time is afforded to carefully observe and study the development of the various symptoms. The inflammatory process may gradually extend to portions of the spinal cord which are diametrically opposed in their functions.

As in all inflammatory processes, a stage of irritation first exists. We observe, therefore, this train of symptoms early, but usually for only a short duration. The effects of irritation upon the motor and sensory apparatus have already been discussed.

Whenever destructive changes occur, a totally different set of abnormal nervous phenomena from those of the irritative stage are produced.

Finally, these destructive changes may become an exciting cause of a *secondary degeneration* of those bundles of nerve-fibres which are cut off by the destructive changes from association with certain cells in the cord which act as regulators of their nutrition (trophic centres).

The *symptoms of irritation* may be manifested by disturbances of sensibility, such as pain, numbness, paræsthesiæ, hyperæsthesia, etc.; and also by disturbances of motility, such as motor spasm, exaggerated spinal reflexes, contracture, and changes in the pupils.

Destruction of the spinal gray matter may result in a total loss of sensation, marked paresis or paralysis, marked atrophy of muscles, paralysis of the pelvic organs, the development of bed-sores or other atrophic disturbances of the skin, and Robertson's pupil (p. 120).

Diagnosis.—This disease cannot be confounded with any of the systematic spinal diseases previously described, because a *combination of motor and sensory phenomena* of an abnormal character is clinically observed.

Focal lesions of the cord closely resemble it in many respects. They can usually be recognized, however, by certain evidences which the patient presents during the progress of central myelitis of a progressive destruction of superimposed spinal segments. When the symptoms of focal lesion shall have been discussed, this distinction will be better understood.

Prognosis.—In the majority of instances, this disease is a fatal one. Active treatment, if commenced sufficiently early, may possibly arrest the morbid process in some cases.

Treatment.—This will be discussed under that of myelitis.

"NON-SYSTEMATIC" OR "FOCAL LESIONS" OF THE SPINAL CORD.*

By reference to a table on page 350, it will be seen that these lesions differ in their character from the systematic diseases which have been described.

The clinical features which they present differ in each individual case; because they are modified by the situation of the lesion, in respect to the different columns of the cord involved, as well as its height in the cord.

The *height of the lesion* is determined partly by the region to which the so-called "cincture" or "girdle sensation" is referred; partly by the extent of the motor paralysis or sensory phenomena; again, by the superficial spinal reflexes which are found to be unimpaired; and, finally, by the history of the case, when the seat of the exciting cause can be well defined.

Focal lesions differ from the systematic or non-focal lesions in that they tend to *spread laterally* from column to column. They often extend to the gray matter of the cord, and sometimes involve the entire structures of both lateral halves of the organ. At first such a lesion may be small and affect only a limited area; in such a case the symptoms may

* Several pages which relate to focal lesions of the cord are quoted from the Author's work, entitled "The Applied Anatomy of the Nervous System." D. Appleton & Co., N. Y.

be confined exclusively to either motor or sensory phenomena, depending upon the column which is attacked. As it spreads to adjacent columns, the symptoms are modified, new ones being added which indicate the direction of its growth. Physiological and anatomical knowledge can alone aid in deciding as to the height of the lesion in the cord, or the portions which are destroyed by it, provided that the cause of the symptoms is not of a traumatic character.

Before we pass to the consideration of lesions confined to special segments of the cord, it may be well to refer again to a few general statements which have been made on page 352.

Focal lesions commonly give rise: (1) to *paresis or paralysis of the extremities:* (2) to *anæsthesia or paræsthesiæ*,—such, for example, as *numbness, formication*, etc.; (3) to *modifications of the superficial and deep spinal reflexes:* (4) to *paresis or paralysis of the bladder and rectum;* and (5) to a *tendency to bed-sores.*

The spinal cord may be regarded from a physiological stand-point as composed of *numerous segments* which are superimposed: each of which is capable of an automatic action. In some of these are placed special centres which govern the action of the viscera, the sexual organ, and the calibre of blood-vessels.

The segments of the cord may be controlled, when necessary, by the ganglia of the brain which are of a higher order: but when this controlling power is interrupted from any cause, as in spinal lesions, for example, the spinal segments may still continue to act automatically. This is one of the many explanations that have been advanced to explain the exaggeration of the spinal reflexes (which often exists when focal lesions of the spinal cord are present), as well as the fact that the bladder, rectum, sexual apparatus, and the skin are sometimes affected by such lesions, and again are not.

It is often possible and of great practical importance to the diagnostician to tell in what region of the cord a lesion is situated, and to estimate the height to which it has progressed. Of course, this is much easier in focal lesions than in the systematic, because the different columns of the cord then simultaneously furnish symptoms which can be compared, and thus aid in the diagnosis. In the table, to which I some time ago directed attention, you will perceive that the focal lesions include traumatisms (of all forms): compression of the cord (chiefly by inflammatory exudation, bone, and tumors): transverse sclerosis of the cord: transverse softening of the cord: hemorrhage into the substance of the cord: and, finally, certain tumors which involve the cord itself. There are many other causes which might excite some local lesion, but these are the ones which will most frequently come under the notice of the practitioner.

A few *anatomical points* are suggested in this connection as of value in spinal diagnosis: (1) the *hypoglossal* and *pneumogastric nerves* arise from the medulla, which lies above the level of the axis; (2) the *phrenic* arises on a level with the spine of the axis; (3) the *brachial plexus* and the *ulnar nerve* are connected with the cord in the region of the neck (third and sixth cervical spines); (4) the *cilio-spinal centre* is situated between the fifth cervical and the second dorsal vertebræ; (5) the lumbar enlargement of the cord gives off the *crural* and *sciatic nerves* at different points, and the space between the eleventh dorsal and the second lumbar spines includes the point of origin of both; (6) the spinal cord ends at the *second lumbar spine*, although the nerves continue to escape from the spinal canal much below that point.

Before we discuss the various conditions enumerated in a previous table as "focal lesions" of the cord (page 350) as separate diseases, it may be advisable to consider in a general way the effects of focal lesions of the cord at different levels. These may be made use of in diagnosis.

We have already studied the effects of systematic lesions, both of the kinesodic and æsthesodic systems, and have noticed how perfectly the physiology of the spinal cord is confirmed by lesions affecting the anterior or posterior portions of the cord separately. We are now called upon to investigate those lesions which, by extending in a transverse direction, are liable to be accompanied by symptoms referable to both the sensory and motor portions of the cord.

Of course, the symptoms will be modified by the extent of the lesion in a transverse direction, so that they may be mostly sensory or motor; but the presence of both sensory and motor symptoms is *strongly diagnostic of focal lesions*, irrespective of a predominance of either, and is never produced by any systematic lesion of the cord, with the one exception of central myelitis.

We may start with a general statement in our study of focal lesions, as follows: Focal lesions usually give rise to *paralysis of motion;* to an *alteration in the reflex excitability* of the cord (usually an increase); and to more or less *anæsthesia*, *numbness*, and *pain.* The *bladder* and *rectum* are *often paralyzed*, and a *tendency to bed-sores* is frequently produced. The first two of these effects, and also the last, are due to alteration in the kinesodic system; the remaining ones are the result of some disturbance to the æsthesodic system.

In studying focal lesions situated in different regions of the spinal cord, we must adopt some system if we expect to grasp the fine distinctions which can be drawn between the results of lesions of the upper cervical region, the cervical enlargement, the mid-dorsal region, the region just above the lumbar enlargement, and, finally, the lumbar enlargement itself.

FOCAL LESION IN THE UPPER CERVICAL REGION.

Hemiplegia will be produced if one lateral half of the cord be alone affected; while paraplegia will be present if the lesion extends transversely to both lateral halves of the cord. The hemiplegia or paraplegia will be complete below the head, and the entire body may be rendered anæsthetic. Since the *phrenic nerve* arises at this point, the act of respiration will be interfered with, creating dyspnœa and hiccough; but respiration will not be arrested, since the pneumogastric nerves continue to excite it, and the auxiliary muscles of respiration can expand the chest without the action of the diaphragm. Should the lesion be a surgical one (as it usually is), the *respiratory centre* of the medulla may be affected, and death take place from asphyxia; but I do not think such a result can be explained as a simple effect of paralysis of the phrenic nerves alone. The involvement of the *cilio-spinal centre* in the lower cervical region may cause the pupils to show an irregularity, and the face and neck may manifest a marked increase of temperature. The pulse may be rendered variable, from irritation of or pressure upon the *accelcratory centre* of the heart.

Now, as I have before said, this type of lesion is almost always a surgical one, comprising pressure from fracture, dislocation, caries, tumors of the vertebræ, etc. These cases seldom live long enough for us to study the effects of such a lesion with much detail. In those rare instances where the lesion is non-traumatic and slowly developed, the effects of irritation have been shown in a hiccough (probably due to irritation of the phrenic nerve), acceleration of the pulse (from irritation of the acceleratory centre of the heart), and dyspnœa (from some interference with the phrenic nerve or the nucleus of the pneumogastric nerve in the medulla); while the paralysis has first appeared as a paretic condition of the arms, then of the chest, and, finally, of the lower limbs.

FOCAL LESIONS OF THE CERVICAL ENLARGEMENT.

This type of lesion differs in its effects if developed suddenly or gradually, and also when situated in the upper or the lower part of the enlargement. If the lesion be so situated as to create *only irritation* of the cilio-spinal centre, or the acceleratory centre for the heart (both of which are in that vicinity), the effects will differ from those due to actual pressure upon or destruction of those centres. In the first instance, the pupils will usually be dilated and the face pale, while the heart will be accelerated; in the latter, the pupils will generally be contracted, the face and neck flushed, and the pulse retarded. The effects will also differ if the lesion affects both lateral halves of the cord or only one.

Wherever the lesion be situated within the cervical enlargement, the arms and legs will gradually become paralyzed; the arms and hands usually becoming first numb and paretic, and the lower limbs exhibiting, for some time, only a sense of weakness and evidences of an increased reflex excitability. A sense of constriction around the chest (the so-called "cincture feeling") is generally present, the seat of which varies with that of the exciting lesion.

When the lesion is situated at the *upper part* of the enlargement, the motor and sensory symptoms will be manifested in the lower extremities, the trunk, and in nearly all the regions of the upper extremities. The constricting band around the thorax is referred to the *level of the clavicles*, and dyspnœa is often excessive.

The brachial plexus is associated with the upper part of the cervical enlargement, and the ulnar nerve with the lower part; hence, the paralysis of the arms in this case would naturally be manifested in almost all of the regions of the upper extremity, and also in those parts supplied by the brachial plexus above the clavicle.

If the lesion be situated in the *lower part* of the cervical enlargement, the symptoms exhibited will include a loss of faradaic reaction of those muscles which are supplied by the *ulnar nerve* (rather than those of the arm and the extensors of the forearm), and atrophy of these muscles will often be developed, chiefly in the flexors of the wrist and the small muscles of the hand. The sense of constriction (cincture feeling) experienced in most spinal lesions of a local character will exist, but it will be referred to the upper part of the chest. A paralytic condition of the muscles of the trunk (the intercostals, triangularis sterni, and the accessory muscles of respiration), as well as of the abdominal muscles, will be detected in severe cases, rendering both inspiration and expiration embarrassed, and thus adding to the danger to life. The lower limbs may exhibit evidences of numbness, anæsthesia, paresis, or complete paralysis, depending upon the extent of the lesion and the destruction done to the tissues of the cord. A condition of paralysis may also exist in the upper extremity.

In surgical injuries to the upper portion of the cord, a peculiarity is often noticed in the *temperature of the body*, which is sometimes greatly elevated. This clinical feature may be associated with a marked retardation of the action of the heart (apparently confirming the situation of an *acceleratory centre* for that organ in the spinal cord).

FOCAL LESIONS OF THE MID-DORSAL REGION OF THE SPINAL CORD.

In the early stages of these conditions the lower limbs become paretic, and a condition of increased reflex excitability is manifested by a rigidity and stiffness of the impaired muscles whenever the patient attempts to stand or walk. As the disease progresses, the muscles become paralyzed and contractured* (probably on account of changes of a secondary character in the lateral columns of the cord). In some cases the reflex movements assume the type of spasms, so as to exhibit both tonic and clonic contractions. It was this symptom which suggested to Brown-Séquard the name of "spinal epilepsy," since it occurs when the patient is exposed to the slightest peripheral irritation, and often when in the recumbent posture.† The sense of constriction around the body is referred to the region of the navel, or that of the lower ribs, or possibly as high as the axilla, since it may be taken as a relative guide to the highest limit of the lesion. A peculiarity exists in this condition as regards the bladder and the rectum; although they may be paralyzed, they are often enabled by the aid of reflex action to expel their contents, thus apparently having regained their function. In the early stages the urine and fæces may be too hastily expelled for the comfort of the patient, often compelling the performance of either act before a proper place can be reached; but, in the advanced stages the urine is retained to such an extent as to cause an "overflow," which is often mistaken for an actual incontinence, since a constant dribbling is present. This symptom is always an indication for the regular use of a catheter. The sexual function seems to be often unimpaired, as coition is frequently possible. It is seldom that the paralyzed muscles exhibit a tendency to atrophy, and the electrical reaction of the affected parts is either normal or exaggerated. The chief seat of weakness is usually first detected in the feet; subsequently the paralysis gradually involves the entire lower limbs.

FOCAL LESIONS ABOVE THE LUMBAR ENLARGEMENT OF THE SPINAL CORD.

In this situation, a focal lesion of the cord produces about the same sensory and motor symptoms as those described in connection with a lesion of the mid-dorsal region,

* A term used in contradistinction to the word "contracted," to designate a *permanent* shortening rather than a temporary response to a motor impulse.

† The presence of urine in the bladder or of fæces in the rectum may often create these spasms.

with the exception that the *reflex spasms*, present in the paralyzed muscles, are perhaps somewhat less violent than when the lesion is higher up the cord. These tonic and clonic spasms are, however, sufficiently well marked to constitute a prominent symptom,* and they indicate an increased reflex excitability of the gray matter of the cord below the seat of the lesion. An ingenious explanation of this increased reflex has been advanced by Professor Seguin, of this city, which seems to merit respectful consideration. I quote from a paper of his upon affections of the spinal cord, as follows:—

"The classic theory of the physiology of contracture in hemiplegia is that it is due to the secondary degeneration; *i.e.*, actively caused by the lesion of the postero-lateral column. Seven years ago (see *Archives of Scientific and Practical Medicine*, vol. i, p. 106, 1873,) I rejected this hypothesis, and suggested a different one, which I have since elaborated and taught in my clinical lectures. This hypothesis, which I intend shortly to publish in detail, is briefly that the spasm is due, not to direct irritation from the sclerosed (?) tissue in the postero-lateral column, but to the cutting off of the cerebral influence by the primary lesion, and the consequent preponderance of the proper or automatic spinal action—an action which is mainly reflex. This theory explains the phenomena observed in cases of primary spinal diseases with descending degeneration, and can be reconciled with results of experiments on animals (increased reflex power of spinal cord after a section high up, Brown-Séquard; inhibitory power of the encephalon on the spinal cord, Setchenow)."

The urinary and rectal organs are affected in about the same way as in lesions of the dorsal region. Coition is often possible, and erections are normally frequent. The rectum is paralyzed, as a rule, and constipation is usually present on that account. Micturition becomes slow and interrupted, as the bladder grows paretic, and retention and overflow are produced later on in the disease.

The paralysis of the extremities is first noticed in the feet, which have long before exhibited a sense of weakness and easy fatigue. Numbness and anæsthesia usually accompany the motor paralysis, and extend as high as the groin or the waist. The sense of a constricting band around the body is present here, as in lesion of other localities, and is referred to the waist, below the level of the umbilicus, or at the level of the hips.

FOCAL LESIONS OF THE LUMBAR ENLARGEMENT.

The lower portion of the lumbar enlargement gives origin to the *sciatic nerve;* hence, it is reasonable to expect that a lesion situated in the lower part of this enlargement would be manifested by symptoms of an incomplete paraplegia, in which the muscles supplied by the sciatic nerves would be the most affected. Now, this fact seems to be confirmed by clinical experience, since the feet, legs, posterior aspect of the thighs, and the region of the nates are chiefly paralyzed when the lesion is so situated. The bladder is unaffected, but the sphincter ani muscle is often rendered paretic, or it may be entirely paralyzed. The portions of the limbs which are to become the seat of paralysis usually exhibit a *sense of numbness* before the effects of the lesion are fully developed, and, in case the posterior columns of the cord be involved, complete anæsthesia may also exist in the parts supplied with motor power by the sciatic nerve. The condition of the paralyzed muscles, as to their electrical reactions, and the presence or absence of the evidences of increased reflex excitability will depend greatly upon how much damage has been done to the gray matter of the lumbar enlargement. If the gray matter be so destroyed as to impair its function, the reflex movements will be absent; and, if the trophic function of the cord be affected by changes in the ganglion cells of the gray matter, the paralyzed

* These reflex spasms have been called by Brown-Séquard "spinal epilepsy."

muscles will undergo atrophy. The sense of constriction, or "band feeling," will usually be referred, in this lesion, either to the ankle, leg, or thigh.

FOCAL LESIONS CONFINED TO THE LATERAL HALF OF THE SPINAL CORD.

In discussing the focal lesions of the cord, we have described the clinical points which are afforded by destruction, to a greater or less extent, of the substance of the cord in both of its lateral halves; hence the motor and sensory symptoms have been usually referred to both sides of the body. It was necessary to thus describe them, since focal lesions, unless traumatic, are seldom confined to one lateral half of the cord; but, in some cases which may be presented to your notice, where a tumor, a fractured vertebra, a hemorrhage, a severe contusion, or some other localized lesion exists, the injury done to the spinal cord may be confined exclusively to one lateral half, resulting in one of two named conditions, viz., "spinal hemiplegia" and "hemi-paraplegia."

Any lesion of a *lateral half* of the spinal cord must produce anæsthesia on the *opposite side of the body*, since all the sensory nerves *decussate* and enter the gray matter of the cord, which serves as a conducting medium for sensory impressions, while the *motor symptoms* produced by the same lesion must be confined to the *same side of the body as the lesion*, since no decussation probably occurs in the spinal cord (these fibres decussating only in the medulla oblongata).

Lateral lesions, as well as those which affect the entire cord, are modified, as regards their symptomatology, by the *height of the lesion* in the cord; since the motor nerves, and the special centres which are situated in the cord itself, will only be affected when they lie below the seat of the lesion, or are directly involved in the destructive process.

When the focal lesion is placed high up in the substance of the spinal cord, the motor paralysis affects *one side only* of the body (provided the lesion is confined to a lateral half), and the term "spinal hemiplegia" is applied to this form of paralysis in contra-distinction to a hemiplegia of cerebral origin. If the spinal lesion be situated in the dorsal region and be confined to the lateral half of the cord, a motor paralysis of *one half* of the same side of the body *below the seat of the lesion* is developed,—a condition to which the term "hemi-paraplegia" is commonly applied. In closing the clinical aspects of lesions of the spinal cord, it will be necessary, therefore, for us to consider the essential features of these two remaining conditions.

SPINAL HEMIPLEGIA.

In order to produce a typical case of this condition, it is necessary to have a lateral focal lesion of the cord in its uppermost part (in or above the cervical enlargement of the cord). If we suppose, then, that such a lesion be present, let us see what we might reasonably expect, on purely physiological grounds, would be the result. We can then examine the clinical records of such cases, and either confirm our deductions or gain some additional information. Such a lesion would, in the first place, shut off all motor impulses sent out from the brain to parts below the lesion, on the same side as the lesion, since the decussation of the motor fibres has already taken place in the medulla; hence, motor paralysis should, theoretically, occur in the arm and leg of the side of the body corresponding to the seat of the exciting lesion, and the trunk should also be paralyzed upon that side. This we find clinically to be true,* with the exception that the *intercostal nerves* often retain their motor power when the nerves of the arm and leg are no longer

* The researches of Brown-Séquard, as early as 1849, and his published memoirs (1863–65, and 1868, 1869) have probably done more to clear up this field and to place it upon a positive foundation than those of any other observer.

capable of carrying motor impulses. In the second place, we should expect to find that the *sensation* of the side of the body opposite to the seat of the lesion would be destroyed or greatly impaired, since the sensory nerves decussate throughout the entire length of the cord. This we also find confirmed by clinical facts; and so perfect is this anæsthesia that the line can often be traced to the mesial line of the body exactly, and upward to the limit of the exciting lesion. In the third place, the situation of the *cilio-spinal centre* in the cervical region of the cord would naturally suggest some effects upon the pupil, and the circulation and temperature of the face, neck, and ear of the same side. This is also confirmed, as the pupil does not respond to light, but it still acts in the accommodation of vision for near objects, and the skin of the regions named becomes red and raised in temperature. Finally, the presence of *vaso-motor centres* in the cord might occasion a rise in temperature in the paralyzed muscles; and, strangely confirmatory of this fact, we often find the temperature of the paralyzed side of the body hotter than that of the anæsthetic side.

In some exceptional cases, the face, arm, and trunk are alone paralyzed, the legs seeming to escape, and often giving evidence of reflex spasm (perhaps most commonly on the anæsthetic side). This must be explained as the result of incomplete destruction of the lateral half of the cord.

HEMI-PARAPLEGIA.

This condition is the result of some focal lesion of the spinal cord in the *dorsal region*, which involves only its lateral half. The results of such a lesion differ but little from those of one causing spinal hemiplegia, as regards the motor and sensory symptoms, excepting that the situation of the exciting cause is below the cervical enlargement, where the nerves to the upper extremity are given off, and where the cilio-spinal centre is situated. For that reason the muscles of the upper extremity are not paralyzed, nor are the effects upon the pupil and the skin of the face, ear, and neck (mentioned as present in spinal hemiplegia) produced. The muscles below the seat of the lesion are paralyzed on the side of the body corresponding to the exciting cause, and the skin is sometimes rendered hyperæsthesic upon that side;* while the integument of the side opposite to the lesion is deprived of sensibility. The bladder and rectum may be paralyzed in some instances. The sense of constriction, or "band feeling," will vary with the seat of disease in the spinal cord. The amount of *reflex irritability* and the presence or absence of *muscular atrophy* in the parts paralyzed will depend upon the depth of the lesion in the spinal cord and the changes which have been produced in the gray matter. The same increase of temperature in the paralyzed limb, which was mentioned as occurring in spinal hemiplegia, may also be present in this variety of paralysis.

Should the side affected with anæsthesia give any evidence of motor paralysis or muscular weakness, or symptoms of anæsthesia appear upon the side where the motor paralysis is present, you may regard either one as conclusive evidence that the exciting lesion is progressing, and that the opposite lateral half of the cord is being involved to a greater or less extent.

SPINAL MENINGITIS.

(*Pachymeningitis Spinalis—Leptomeningitis Spinalis.*)

The membranes of the spinal cord may become inflamed independently of, or in conjunction with, similar changes in the cerebral envelopes. I shall discuss the former variety here.

* This is probably due to some irritation of the gray matter of the cord.

Spinal meningitis may be of two forms: (1) *pachymeningitis* (internal and external varieties), and (2) *leptomeningitis* (acute and chronic varieties). I have chosen to include both of these conditions among the focal lesions of the cord, because their effects upon the functions of that organ are due chiefly to pressure. The pressure so produced is liable to spread in a lateral direction from column to column of the cord.

Although the acute variety of leptomeningitis (inflammation of the pia and arachnoid) is generally diffused over a much larger area than the chronic, the distinction drawn on a previous page between systematic and focal lesions of the cord (see table on p. 350) clearly justifies the classification adopted.

When the symptomatology of this disease is reached, many of the hints given in connection with the diagnosis of focal lesions (p. 411 to 415) will prove of great assistance to the reader.

Morbid Anatomy.—In the *acute variety* of leptomeningitis, the membranes (chiefly the pia) are rendered thicker than normal, more or less opaque and œdematous, sometimes ecchymotic, and often adherent to each other. The meshes of the pia are filled with a turbid exudation, which may be tinged with blood. It is sometimes clear, but generally more or less filled with flocculi of lymph. The consistency of this exudation varies. It may be fluid, or of the density of jelly. It is usually most abundant at the posterior part of the cord and around the posterior nerve-roots; but it may envelop the entire circumference of the cord.

In the arachnoid, hard cartilaginous plates are occasionally detected. The size of the plates may reach a quarter of an inch in diameter, but they are usually much smaller.

The *dura* may become involved in conjunction with the arachnoid and pia; or it may be separately affected, as is also the case with the dura lining the cavity of the cranium. We may thus have an *internal* and *external variety* of *spinal pachymeningitis*, as well as *leptomeningitis* (inflammation of the pia).

The *external form of spinal pachymeningitis* is primarily an affection of the dura. The other membranes may, however, occasionally become involved by an extension of the inflammatory process. Its most common seat is in the cervical region, and it seldom extends beyond the limits of two vertebræ. Several such foci of inflammation may coexist. The membranes become hypertrophied and encroach upon the spinal canal. This may result in a compression of the spinal cord. Myelitis may be thus induced. Again, the roots of the spinal nerves may be compressed by the changes in the membranes through which they pass, thus causing disturbances of sensibility and motility.

The external variety of pachymeningitis spinalis is confined to the loose connective tissue between the dura and the vertebral canal. This is more abundant in the posterior part of the spinal canal than elsewhere; a fact which helps to explain the circumscribed character of its morbid changes, as well as the tendency of these changes to become most marked posteriorly.

Swelling and redness are generally to be detected at the seat of the extra-dural cellulitis. Abscesses may develop in exceptional instances. In other cases the dura is simply thickened and adherent; or dry, cheesy masses of considerable size may be detected.

The pains, which are a prominent symptom of the first stage of this affection, are probably due to a slight compression of the posterior nerve-roots. Rigidity of muscles may also occur from a similar condition of the anterior nerve-roots. Later on, the compression of the cord may induce paralysis, contracture, and atrophy of muscles (chiefly those supplied by the median and ulnar nerves), anæsthesia of limited areas of the body, and possibly symptoms of incoördination of movement. We owe most of our knowledge of this affection to the publications of Charcot.

The *internal form of spinal pachymeningitis* (the hemorrhagic variety) was first fully described by Meyer, although it was partially recognized by Albers. A laminated sac enclosing a hemorrhagic condition of the spinal membranes exists in this disease, which is identical with that described in connection with the dura of the cranium. It seems to occur in connection with alcoholism, some cases of insanity, general paralysis, caries of the bones, and syphilis.

This form of spinal pachymeningitis usually runs a chronic course. It is not always of the hemorrhagic variety. The dura presents in rare cases upon its inner surface a series of concentric lamellæ of a fibrous character, while the arachnoid and pia have become similarly affected, constituting the so-called "*hypertrophic internal spinal pachymeningitis.*"

In this disease, the spinal cord is constricted by a ring of fibrous tissue (with numerous interstitial spaces) whose concentric lamellæ may exceed one-twentieth of an inch in thickness. This ring is generally situated in the lower half of the cervical enlargement of the cord. It tends to compress the spinal nerve-roots, and later the substance of the cord itself. When the latter occurs the spinal gray matter is apt to suffer. Frequently newly-formed canals are detected near to the gray commissure, which are lined with a membrane and contain fluid.

When spinal meningitis is developed in connection with syphilis, the lesion is usually of the type of gummata, and is more or less circumscribed. The antero-lateral portions of the cord are more often involved than the posterior; hence, we are more apt to encounter disorders of

motility than of sensation or coördination. When these gummata are not multiple, they are commonly found in the region of the lower dorsal and upper lumbar segments of the cord. If multiple, they may occasionally be detected higher up.

In the *chronic variety* of spinal leptomeningitis, the membranes are excessively thickened and puckered. The adhesions are also abundant and very firm. Finally, the opacity of the membranes is greater than in the acute form. In some cases pigmentation exists. Calcareous plates within the membranes are not uncommon. The amount of fluid within the subarachnoidean space is increased, and may contain flocculi of lymph, with pus, or blood. The cord is generally anæmic and frequently sclerosed. The nerve-roots are usually more or less degenerated and atrophied.

Etiology.—Among the *predisposing causes*, bad hygienic surroundings, an excessive use of alcohol and tobacco, indulgence in narcotics, exposure to cold or dampness, rheumatism, venereal excesses, scrofula, wasting diseases, tuberculosis, and general debility may be mentioned as prominent.

The *exciting causes* comprise injuries to the spine of all kinds, operations for spina bifida, syphilis, alcoholismus, some of the diseases of the cord, tumors of the cord or its meninges, caries of the vertebræ, tetanus, hydrophobia, cancer, spinal concussion, infectious febrile diseases, and rheumatism.

External pachymeningitis is almost always a secondary disease. Among the primary conditions which may induce it by extension through the intervertebral foramina, may be mentioned caries of the vertebræ, suppurations in the neck or pharynx, tuberculosis, pleurisy, empyema, peritonitis, pelvic suppuration, syphilitic ulceration, etc.

Youth and early adult life are more prone to this disease than old age. It is most frequent in males. The acute form is liable to be followed by the chronic. Spinal pachymeningitis is a very common complication of Pott's disease.

Leptomeningitis is most frequently met with during the winter months. Sun-stroke is said by some authors to induce it, but I think such instances must be very rare. The chronic variety generally coexists with locomotor ataxia, multiple spinal sclerosis, myelitis, and other organic spinal diseases.

Symptoms.—The most important and marked symptom of the onset of leptomeningitis is *pain*. This may be localized in the back, or it may shoot into those parts which are associated with the segments of the cord nearest to the seat of inflammation. The pain is generally constant, but it may often be intensified by movements of the spine, pressure over the spinous processes, or the application of an electric current to

the spine. Like some other symptoms which occur early, it may be regarded as a clinical evidence of irritation of the posterior nerve-roots.

A *chill* or severe rigor may usher in this disease in some cases. It is often followed by nausea, vomiting, a moderate rise in temperature, and a sense of weakness. The pulse is seldom greatly accelerated: it may even be below the normal standard.

Soon a *rigidity of the muscles* of the spine appears. Opisthotonos is developed in some cases, especially when the cervical region is involved. The patient generally maintains a fixed position, since movement intensifies the pain.

Convulsive twitchings of the muscles may be developed. They are exceedingly painful.

The spinal reflexes will be found, as a rule, to be exaggerated; this symptom indicates an irritation of the lateral columns of the cord.

Sensory and motor symptoms gradually develop. The skin may become intensely hyperæsthesic, and, as a rule, the motility of the hyperæsthesic parts is more or less impaired. It is common to encounter an incomplete form of paraplegia, or anæsthesia when the cord or nerve-roots are seriously affected.

Some of the other symptoms of this disease depend upon the *seat and extent of the lesion.* There may be frequent micturition or retention, whenever the vesical centre of the cord is involved. Dyspnœa may be produced when the lesion is high up. Sweating of a profuse type indicates an impairment of the vaso-motor nerves or centres. The pupils may be irregular from defective innervation of the cilio-spinal centre. Bed-sores may be produced on account of trophic disturbances. It is unnecessary to repeat here all that has been discussed when the effects of focal lesions of the cord were described. (Pages 411 to 415.)

Chronic spinal leptomeningitis generally follows an acute attack. There are exceptions to this rule, but they are infrequent. The extreme pain of the acute stage usually gives place to a sense of dull aching, soreness, or an itching and burning of the limbs. The "cincture feeling" is well marked, and is a guide to the height of the lesion. The intestinal, vesical, and sexual centres of the cord may be affected and cause marked disturbances of their functions. We are apt to have constipation or incontinence of fæces, incontinence of urine, impotence or priapism, bed-sores, etc. The lower limbs are particularly liable to become partially paralyzed. Hyperæsthesia is also a common symptom.

In the *external variety of pachymeningitis spinalis*, the symptoms closely resemble those of leptomeningitis. A stiffness in the back when the patient rotates the spine (or when sitting or standing for any length of time) is noticed early. The application of heat, cold, pressure, or the negative pole of a galvanic battery increases the pain at the seat of the

morbid changes. The "cincture feeling" is generally experienced around the body at a level approximating closely to the height of the lesion. The pain is apt to be more paroxysmal than in leptomeningitis. Sometimes the spine may present evidences of the primary disease which has induced the morbid changes in the extra-dural connective tissue. The neck is not usually rigid, because this condition is infrequent in the cervical region.

In the *internal variety of pachymeningitis spinalis*, the symptoms run a more chronic course, and more closely resemble those of spinal and meningeal tumors.

In the "*hypertrophic*" *form*, the stage of irritation is followed (after a lapse of a few months) by paralysis and atrophy of the muscles. These later symptoms are due to compression of the spinal cord or the spinal nerve-roots. The stage of irritation is characterized by paroxysms of severe neuralgic pain in the shoulders, the occiput, the cervical region of the spine, and the large joints of the upper extremity. It is not usually affected by pressure upon the spinous processes. Hyperæsthesia and paræsthesiæ are frequently observed early in these subjects. The skin of the upper limbs may show trophic disturbances, chiefly by desquamation, vesicular eruptions, or a roughness of the epidermis. The hand may assume the typical attitudes depicted by Charcot and Ross.

Diagnosis.—The different forms of spinal meningitis previously described are not always easy of diagnosis. The best clinicians have been misled in cases where the autopsy has revealed very marked lesions of the spinal meninges. As a rule, however, it is well to suspect the existence of spinal meningitis whenever we encounter a persistent and severe pain in the back which is aggravated by movement, and which occasionally shoots along the spinal nerves (those associated with the diseased area). Our suspicions are strengthened if we observe also a tendency toward muscular rigidity, distortions of the spine, or an exaggeration of the spinal reflexes, together with marked clinical evidences either of irritation or impairment of the motor or sensory functions.

The presence of marked febrile symptoms at the onset of any attack associated with spinal symptoms should lead us to suspect the development either of a meningitis or a myelitis. It may be justly said, however, that these two conditions usually go hand in hand, and that one or the other simply predominates. The existence of a "girdle pain" or a "cincture feeling" is rather diagnostic of myelitis; as is also the presence of exaggerated reflexes, with bladder and rectal complications, changes in the pupils, contractures, or trophic disturbances.

The difficulties in diagnosis are to be attributed chiefly to the fact

that it makes very little difference clinically whether the spinal cord is actually diseased or subjected to pressure, and that we cannot always discriminate between lesions of the cord and those which involve the spinal nerve-roots.

The table on the following page will possibly prove of value to the reader in making the diagnosis of the different types of spinal meningitis from each other, and in discriminating between them and other conditions which resemble them in many respects.

Prognosis.—In the external form of pachymeningitis spinalis, the prognosis is always grave. Recovery is very rare and seldom complete. In the internal variety recovery is possible.

In leptomeningitis spinalis, the acute variety is apt to coexist with a myelitis, and the prognosis is grave. It is especially so if paralysis and atrophy are developed, or if the sphincters are involved. A bad constitution, a high range of temperature, dysphagia, an extension to the medulla, and extreme youth or old age are all unfavorable to the patient. Relapses are very common, even if the case progresses favorably.

In the chronic variety, death is apt to occur from cystitis, bed-sores, an acute exacerbation of the disease, or an extension to the medulla. It is apt to last for years, and to lead to permanent paralysis and atrophy of muscles.

Treatment.—The cause of the morbid condition, as well as the symptoms which it creates, must necessarily modify the treatment of each case.

Irrespective of syphilitic origin, some authors advise calomel by the mouth. Personally, it seems to me a dangerous and unscientific way of controlling (?) inflammatory processes.

If syphilis exists, mercurial inunctions or fumigation may be employed with benefit, and the iodides may be given in conjunction with mercury. This subject is quite fully discussed on page 291.

Applications of ice-bags, wet-cups, leeches, or the actual cautery may be made to the spine over the painful regions.

Bed-sores may be prevented by extreme care respecting the patient and the bed, and by a change in posture (preferably upon the side or abdomen). Bathing the skin daily in alcohol, and applying diachylon plaster over any inflamed spots, may prove of service as preventative measures. Air-cushions are often employed to remove pressure from tender surfaces. If sores actually occur, they may be treated by the permanent water bath or by ordinary surgical dressings.

If the bladder becomes involved, and exhibits incontinence, overflow or retention, it is advisable to introduce a catheter at regular and short intervals, and to thoroughly cleanse the bladder daily by attaching the catheter to the pipe of a fountain syringe filled with water and

SPECIAL SYMPTOMS.	SPINAL LEPTOMENINGITIS.		SPINAL PACHYMENINGITIS.			AMYOTROPHIC LATERAL SCLEROSIS.	TETANUS.
	Acute variety.	*Chronic variety.*	*External variety.*	*Internal variety.*	*Hypertrophic variety.*		
PAIN	Is more or less *constant.* Increased by movement of spine. May shoot along special nerves.	Closely resembles that of the acute variety.	Is paroxysmal. *Intensified by heat, cold, or galvanism to spine.*	May be absent in some cases. May resemble that of rheumatism.	Is paroxysmal. Confined to *head, neck, shoulders,* and *upper limbs.* Not affected by pressure over spine.	*No stage of irritation exists. No pain.*	No pain exists except during paroxysms.
EVIDENCES OF SENSORY DISTURBANCE	Hyperæsthesia usually present. Marked. Tenderness of the muscles generally exists.	Hyperæsthesia (which may be very acute). Muscles less sensitive than in acute variety.	Hyperæsthesia. Hyperalgesia. Paræsthesiæ. Anæsthesia. Analgesia. Cincture-feeling.	Usually develop slowly. Similar to those of external variety of pachymeningitis. Cincture-feeling may exist.	Confined to regions specified above.	Absent.	Abnormal sensory phenomena are absent.
MOTOR SYMPTOMS CAUSED BY THE LESION	Twitchings of the muscles. Stiffness of neck. Distortions of the trunk. Retention of urine. Paresis of lower limbs may occur Exaggerated reflexes.	Spasmodic contractions. Distortions infrequent. Retention may occur. Gradual paresis. Reflexes normal or exaggerated.	Twitchings. Contractures. Atrophy. Paralysis.	May occur rapidly, if a sudden and extensive hemorrhage occurs.	Ulnar paralysis. Median " Radial " Atrophy (*from disuse*) not complete. No bulbar symptoms.	*Contracture. Marked atrophy Paralysis. Bulbar symptoms.*	Muscles of *mastication* are affected in common with those of limbs and trunk.
RIGIDITY	*A prominent symptom.* May affect *spine* or *extensors* of limbs.	Not so marked as in the acute variety.	*Not present in the neck* (as a rule).	May be absent.	Is generally due to *excessive action of the extensors,* as a result of paralysis of the flexors. Most marked in forearm.	Very marked *contractures* usually develop in legs or arms.	Present only during paroxysms.
CLINICAL HISTORY OF THE CASE	Marked febrile symptoms exist. Bladder and rectal complications may exist. Pupils may be irregular. Cheyne-Stokes respiration may develop, if the medulla be implicated.	No febrile symptoms. The symptoms are aggravated by the recumbent posture. Bladder may be paralyzed. Pupils seldom affected. Bed-sores commonly observed.	*Follows some previous disease* (accompanied by pus formation, as a rule). Febrile symptoms.	Frequent in *drunkards* and the *insane.* Cerebral symptoms are often present (since lesions are often in brain also).	Sense of constriction in chest. Peculiar attitudes of the hand are developed.	Affects both the *upper* and *lower limbs.* In some cases pupillary changes may be observed	No bladder complications. No pupillary changes.
TERMINATION	Recovery not probable, but possible.	Mostly of indefinite duration. Recovery often.	*Generally fatal.*	Recovery possible.	Recovery impossible.	*Always fatal.*	Recovery possible.

raising and lowering the bag. This alternately distends and empties the organ and affords great relief to many patients. The patient can regulate the pressure by having a string which is attached to the fountain run through a pulley in the ceiling directly over the side of the bed, so that he can raise or lower the bag without changing his posture in bed.

The pain is best controlled by opiates in full doses. I usually administer it by the hypodermic method. The stomach is less affected by so doing, and you can control the dose better than by trusting it to the patient or the attendants. If nausea is created, add one grain of atropia to one ounce of Magendie's solution of morphia in preparing the hypodermic solution. Acid should not be used in dissolving the morphia, as it increases the danger of abscesses.

Ergot and iodide of potash are commonly employed in full doses by most authors who have written upon this disease and myelitis. I have not much faith in the beneficial effects of the latter, and I seldom push ergot to extremes.

As the patient becomes able to walk it is best to insist on very gradual exercise, care being taken to avoid over-exertion. In this connection, sexual intercourse should be interdicted.

Paralyzed and atrophied muscles may be subjected to massage, faradization, galvanization, or static sparks of a mild character. I think that strong currents are generally detrimental in these patients.

Finally, strychnia, arsenic, iron, and a well-regulated diet are of service in building up the strength during convalescence, or in prolonging the life of the patient.

TUMORS OF THE SPINAL CORD AND ITS COVERINGS.

Within the substance of the cord, glioma is most often found among the tumors; and sarcoma comes next in frequency. Tubercle and gummata, as well as fibro-sarcoma and myxo-sarcoma, may likewise be detected at an autopsy. Tumors may also spring from the meninges and the vertebræ and affect the spinal cord indirectly.

Morbid Anatomy.—Glioma most often affects the cervical and lumbar enlargements. If extremely vascular (as it sometimes is) the tumor may be infiltrated with blood and contain blood-cysts. It probably starts from the neuroglia. Mixed varieties of glioma are sometimes found in the substance of the cord (chiefly glio-sarcomata).

In the *meninges of the cord*, we may encounter all the varieties of tumors mentioned excepting glioma; and, in addition, carcinoma, psammoma, parasitic growths, fibromata, and myxomata.

The *bones of the vertebral column* and their periosteum may be the starting point of intra-spinal growths. These may compress the cord.

Finally, *aneurisms* of the spinal arteries or of the thoracic or abdominal aorta may interfere with the functions of the cord. The latter can only do so by first causing absorption of the vertebræ.

Etiology.—Wounds, injuries, and the results of tubercular, cancerous, and syphilitic cachexiæ are the only definitely known causes of these morbid growths.

Symptoms—Tumors of the spinal canal cause symptoms either by irritation or compression of the cord or the spinal nerve-roots or by inducing changes in the bones. Even when of large size they may produce no symptoms. In some cases we may encounter all the clinical evidences of a myelitis, or of spinal meningitis. The general remarks made in reference to focal lesions of the cord (pages 411 to 415) are applicable to spinal tumors.

Diagnosis.—Although it is often impossible to recognize a spinal tumor and its seat during life with certainty, there are some symptoms which should lead to a suspicion of this condition. These comprise: (1) an excess of motor paralysis on one side of the body over that observed on the other, with an excess of anæsthesia on the side where motility is least affected; (2) a clinical history which would lead to the suspicion of tubercle, cancer, or syphilis in the patient; and (3) the long duration of the disease (usually from six months to several years) and the gradual development of the spinal symptoms. Moreover, the ability on the part of the patient to recognize with closed eyes the position of the limb during passive movements (muscular sense) is apt to be more affected on one side (that corresponding to the tumor) than on the other.

When more than one tumor exists, the diagnosis is even more uncertain than if the growth were single. It might then simulate multiple spinal sclerosis. I lately treated a case of this description. By a careful study of the symptoms I was enabled to recognize during life a multiple lesion of the cord and the seat of the morbid processes with some exactness, and I suspected either multiple sclerosis or multiple tumor. The autopsy confirmed the latter view, as sarcomata of the meninges were discovered.

Prognosis.—This depends upon the nature of the growth. If it is syphilitic, recovery under the treatment indicated on page 291 may be expected, provided the spinal cord has sustained no permanent injury from compression. Tubercle may, in exceptional cases, be recovered from. As a rule, however, spinal tumors are fatal.

Treatment.—Iodide of potash, arsenic, cod-liver oil, phosphatic salts, etc., may be employed as symptoms arise which seem to demand them, unless the case be clearly of syphilitic origin. In the latter form the most active specific treatment is indicated.

SPINAL HEMORRHAGE.

Blood is rarely extravasated into the substance of the spinal cord. It is generally poured out into the spinal meninges. We can therefore divide spinal hemorrhage into the *intra-medullary* variety or "spinal apoplexy," and the *extra-medullary* variety, or "meningeal hemorrhage."

SPINAL APOPLEXY.

(*Hæmatomyelia.*)

Etiology.—This condition is somewhat rare. It may occur from changes in the coats of the blood-vessels or from excessive blood-pressure. Probably the latter cause is not alone sufficient to induce it in health.

Morbid Anatomy.—We encounter intra-medullary hemorrhage in connection with gliomatous tumors, acute myelitis, and traumatisms. The clot is usually small in size, seldom exceeding that of an almond.

In exceptional cases, the blood may escape through the pia into the subarachnoidean cavity.

The age of the clot will modify its appearances at an autopsy. If recent it will be red or blackish-red. Older clots become brown or yellow in color. If sufficient time has elapsed to allow of still further changes, the clot may be found to be encapsulated by a fibrous deposit, or possibly an apoplectic cyst or a pigmented cicatrix may be all that remains of the original lesion.

Symptoms.—These depend upon the seat and extent of the lesion. As a rule, the patient is attacked with sudden paraplegia, accompanied by severe pain in the back. The pain usually subsides within twenty-four hours. Occasionally, the symptoms will indicate a unilateral lesion of the cord. (Fig. 67.) Again, the effects of disease confined to the posterior or anterior horns will be evidenced at the onset.

Prodromata are very infrequently observed. Consciousness is not lost except when the lesion is situated near the medulla. A slight fever is apt to follow the attack after a lapse of some hours. A high range of temperature only occurs as the result of complications, such as cystitis, bed-sores, etc.

The paralysis may be of the type of monoplegia, hemiplegia, or paraplegia.

The sensory functions may be disturbed, causing anæsthesia, analgesia, hyperæsthesia, paræsthesiæ, etc.

The bladder and rectum may be affected.

The reflexes may be abolished or exaggerated.

Vaso-motor and trophic disturbances have been observed, and also hæmaturia and albuminuria.

The effects of focal lesions of the cord at different levels may be studied in this connection with benefit. (Pages 411 to 415.)

Diagnosis.—Some of the more important symptoms of this affection can be contrasted with those of other spinal diseases (which closely simulate it) as follow:—

	SPINAL APOPLEXY. (*Hæmatomyelia.*)	SPINAL MENINGEAL HEMORRHAGE. (*Hæmatorrhachis*)	POLIOMYELITIS ANTERIOR ACUTA.	MYELITIS.
RAPIDITY OF ONSET	Rapid. May be instantaneous.	Rapid or instantaneous.	Never instantaneous.	Never instantaneous. Comparatively slow, as a rule.
FEVER	*Absent* at *onset*. Occurs on second or third day.	*Absent* at *onset*. Occurs on second or third day.	*Precedes paralysis.*	May be absent. Is generally marked.
PAIN	Severe in *back* at onset and gradually subsides. *Not marked in limbs.*	Marked in *back* and *limbs*, and are of considerable duration.	*No pain.*	Not prominent as a symptom. May be totally absent.
HYPERÆSTHESIA	May be wanting.	Generally present.	Absent	Not marked.
ANÆSTHESIA	May exist in parts below lesion. Appears at onset.	Usually imperfectly developed and appears late.	Absent.	An early sign, and is generally well marked.
CONTRACTURE AND SPASM	Occur at onset (if at all) as a rule.	Are strongly diagnostic.	Absent.	Not usually very prominent.
SPHINCTERS OF BLADDER AND RECTUM	Often paralyzed.	Unaffected until late in the disease.	Normal.	Peculiarly liable to be affected.
BED-SORES	Frequently developed.	Not developed.	Not developed.	Extremely common.
ATROPHY OF MUSCLES	Occurs rapidly only in those muscles connected with *affected spinal segments.*	May not occur.	Occurs rapidly in *all the paralyzed muscles.*	May not exist.
ELECTRICAL TESTS	Reaction of degeneration in atrophied muscles associated with affected segments of cord.	May be normal.	*Reaction of degeneration* in all paralyzed muscles.	May be normal, exaggerated, or abolished.
PROGNOSIS	Often fatal. Recovery always incomplete.	Complete recovery is possible.	Rarely fatal.	Generally unfavorable.

Prognosis.—The situation of the clot modifies the prognosis, as well as the amount of blood which is extravasated into the substance of the cord. Hemorrhages into the dorsal segments cause less serious symptoms than if present in the cervical segments (where the respiration may be disturbed by interference with the phrenic nerve), or in the lumbar segments (where the centres for the vesical and rectal sphincters are probably situated). The rapid development of bed-sores is an unfavor-

able sign. In any case, a complete recovery is impossible, and a long duration of life improbable.

Treatment.—To arrest danger to the patient from a further escape of blood, ice-bags should be applied to the spine as soon after an attack as possible, and large doses of ergotine should be given by the mouth or hypodermically. The patient should be placed in a horizontal posture, and absolute rest and quiet should be insisted upon. All undue excitement should be carefully guarded against. The subsequent treatment does not differ materially from that already recommended for myelitis.

SPINAL MENINGEAL HEMORRHAGE.

(*Hæmatorrhachis.*)

This morbid condition is not commonly encountered. It is more frequent among males than females. It may be secondary to a spinal apoplexy which has perforated the pia, or to an aneurism.

Etiology.—No cause can be discovered in some cases. In others, a history of traumatism, tubercular disease or cancer of the spine, violent excitement, suppressed menstruation, cardiac hypertrophy, attacks of spasmodic diseases, purpura, a hemorrhagic diathesis, some infectious disease, or the presence of an aneurism (which has at last burst into the spinal canal), may be detected. It may occur in the infant from the use of forceps. A cerebral hemorrhage has been known to be sufficiently severe to flow into the spinal canal.

Symptoms.—These are dependent upon the extent and seat of the hemorrhage. They are largely due to irritation or compression of the spinal nerve-roots at first, and possibly, later on, to spinal compression or myelitis.

The character of the onset depends to a great extent upon the rapidity of the effusion. A large clot will cause at once very severe shooting pains in the back and the limbs, with more or less muscular twitchings, cramps, spasms and rigidity.

The onset is unaccompanied by fever; and the paralysis and anæsthesia are not very pronounced at first. On the second or third day reactive fever sets in to a moderate extent.

There is apt to be a "cincture feeling" around the chest or abdomen.

If the bladder or rectum show any impairment, or when bed-sores occur, it indicates that the spinal cord is compressed.

Whenever the nerve-roots become seriously impaired by pressure of the clot, the functions of motion or sensation, as well as the spinal reflexes and the electrical tests of nerves and muscles begin to show a progressive deterioration.

Prognosis.—This disease lasts from two weeks to an indefinite period. It is possible for death to occur from shock, soon after the

onset, provided the clot be near the medulla. Spinal hemorrhage may induce a complicating meningitis; and the extension of this inflammation to the medulla may cause death. Permanent paralysis and atrophy of muscles may be induced by pressure upon the anterior nerve-roots, although the spinal cord may have entirely escaped injury.

The published records of these cases go to show that quite a large percentage tend to make a recovery without any very serious impairment of motion or sensation.

Treatment.—This is similar to that given for spinal apoplexy.

MYELITIS.

Inflammation of the substance of the spinal cord has been partially studied already under the heads of two systematic spinal diseases, viz.: poliomyelitis anterior, and central myelitis.

We now approach the consideration of structural changes in the cord of an inflammatory type which assumes the character of a focal or "non-systematic" spinal disease.

We shall discuss this morbid condition as of two varieties, the acute and chronic.

ACUTE MYELITIS.

This disease may be primary or secondary. It is comparatively a rare affection; more frequent in males than in females, and is generally observed during middle life.

Etiology.—The causes of this disease vary with its type; although a predisposition to it may be engendered by excesses in alcohol, bad hygiene, overwork, venereal excesses, and exposure to dampness.

The *primary variety* may be developed as the result of rheumatism, traumatism to the spine, severe emotional excitement and certain poisons. Among the latter causes, lead, arsenic, mercury, phosphorus, alcohol, and carbonic oxide have been known to induce it. Some of these poisons may be taken into the system while following certain manufacturing pursuits.

The *secondary variety* may be induced by diseases of the vertebræ and the spinal meninges; either by extension of inflammatory processes or as the result of compression of the cord. Again, it has been known to follow pneumonia, phthisis, syphilis, diphtheria, pyæmia, the eruptive fevers, and malarial poisoning.

Morbid Anatomy.—Myelitis of the acute form most commonly attacks the dorsal segments; occasionally the cervical and lumbar enlargements of the cord. The extent and seat of the inflammation varies. It may attack the central gray matter (*central myelitis*); again, it may traverse the entire spinal segment (*transverse myelitis*); finally,

it may be distinctly circumscribed and confined to one lateral half of the cord (*circumscribed myelitis*).

A very rare condition, known as "*perimyelitis*" or "*myelomeningitis* (in which only the periphery of the cord is inflamed) has been observed.

The appearance of spots of myelitis must not be confounded with post-mortem changes which are commonly detected during the warm months in subjects which have been kept some time.

When myelitis is present, we may encounter the characteristic appearances of red, white, or yellow softening. These have been described in connection with the brain (page 317).

The formation of a distinct abscess of the cord is sometimes observed in myelitis of the acute form. It is most often found in pyæmic and traumatic cases.

The meninges, especially the pia, are generally more or less reddened, softened, and infiltrated with pus. The nerve-roots may be markedly swollen and appear redder than normal. Finally, evidences of ascending or descending spinal degeneration (see Fig. 92) are generally to be detected.

Symptoms.—The onset of an acute myelitis may or may not be preceded by prodromata. If so, they are poorly defined and are those of slight febrile disturbance.

The *onset* may be accompanied by convulsions in children; and, in adults, by a chill and marked fever.

Within a short space of time the patient begins to notice abnormal sensory symptoms, such as pain in the back, a cincture feeling around the chest or abdomen, formication and shooting pains in the limbs, pains in the joints or cardialgia. The pain in the back is not increased by movement, unless a spinal meningitis coexists.

The muscles may exhibit twitchings or temporary contractures early. The bladder may be affected almost from the onset; causing either retention, overflow, or incontinence of urine.

Later in the disease, *paralysis* of the muscles of a complete or partial character develops. Complete anæsthesia may be observed in other parts.

The muscles *begin to waste rapidly* whenever the anterior horns of the spinal gray matter are attacked; and the "reaction of degeneration" is then developed in the nerves and muscles associated with the spinal segments thus affected.

If the muscles of the abdomen or chest are paralyzed, respiration becomes markedly interfered with, and slight pulmonary complications become a source of danger to the patient. Asphyxia develops suddenly when the phrenic nerve-roots are attacked.

In some cases *delayed sensation* (page 398) is observed. In rare instances, circumscribed stimulation of the skin, as in the case of a pin-thrust, is followed by a vibration of the limb. This is known as "*dysæsthesia.*"

The *vaso-motor nerves* generally give clinical evidence of their impairment quite early in the disease. The paralyzed limb may cease to perspire. The joints may swell from œdema, and become cold and peculiarly pale. Eruptions may develop, usually of the vesicular type. Over the bony points which sustain the weight of the patient, the skin is peculiarly liable to become reddened, and to undergo a rapid form of gangrenous destruction. We encounter bed-sores of this type chiefly in the region of the sacrum, and over the trochanters, malleoli, and the os calcis.

Cerebral symptoms are generally absent. In rare cases, atrophy of the optic nerve has been observed.

The *urine* may become ammoniacal, bloody, albuminous, and saccharine. Indications for regular catheterism are often clearly pronounced. Involuntary evacuations of the bowels may follow a paralysis of the sphincters.

The *condition* of the *reflexes* varies with the seat and extent of the lesion. If the lateral column is alone affected, they will be exaggerated. If the "reflex arc" (Fig. 34) is injured anywhere in its course, they will be decreased or abolished.

Diagnosis.—Acute myelitis is apt to be confounded with spinal meningitis, spinal apoplexy, spinal meningeal hemorrhage, hysterical paralysis, and multiple neuritis.

From the first three of these diseases, the distinguishing points are clearly shown in a diagnostic table on page 426.

From true *hysterical paralysis*, without organic spinal changes, acute myelitis is to be told by its rapid course, its febrile symptoms, its bladder and rectal complications, its trophic disturbances, and the sex affected.

From *multiple neuritis*, myelitis is to be differentiated in many cases by the absence of severe pains in the limbs, an imperfect history of excess in alcohol, the pain in the back, and the retention of normal electrical formulæ, both of nerves and muscles. In a few cases, the diagnosis is difficult. While it is not always easy to make the distinction, it must be remembered that bed-sores and a disturbance of the bladder and rectum are peculiarly characteristic of myelitis.

Prognosis.—If the myelitis is not of the hemorrhagic variety, the duration of the acute form is usually from one to several weeks. The patient may die of ammoniæmia, septicæmia, pyæmia, exhaustion, paralysis of the respiratory or cardiac centres, or some pulmonary

complication (chiefly pneumonia). Some patients pass into a subacute or chronic variety of myelitis. A few are said to have recovered completely.

Treatment.—The steps indicated in connection with the treatment of spinal meningitis are applicable to that of myelitis.

CHRONIC MYELITIS.

Under this head some authors place all inflammatory conditions of the substance of the spinal cord which are focal in type, run a protracted course, and are unattended with febrile symptoms. Some of the "systematic" diseases already described are but special forms of chronic myelitis; as, for example, primary lateral sclerosis, locomotor ataxia, amyotrophic lateral sclerosis, poliomyelitis anterior acuta, etc.

Etiology.—A congenital or acquired hereditary predisposition to this form of myelitis is more pronounced than in the acute variety. The exciting causes are similar to those of the acute form of the disease.

Morbid Anatomy.—The eye will usually detect a flattening or depression of the spinal cord, with an unnatural firmness of its substance, at the seat of the disease. The pia may be adherent over the diseased area; and it, as was the dura, may be markedly thickened. In exceptional cases softening of the cord is observed; possibly, also, the formation of cavities in its substance (syringomyelia) may be detected.

The microscope will usually show an increase of Deiter's cells, which are often enlarged and present numerous nuclei; a marked increase of the neuroglia; a thickening of the coats of the blood-vessels; a dilatation of the lymphatic sheaths of the blood-vessels; an enlargement of the axis-cylinders (chiefly at the periphery of the lesion); a disappearance of the medullary sheaths; and the presence of granulo-fatty cells.

Chronic myelitis may in exceptional cases be confined to one lateral half of the cord. It more often tends to spread transversely to both lateral halves. It may also assume a multiple form.

The abdominal and thoracic viscera may exhibit evidence of existing complications of myelitis.

Symptoms.—These are modified somewhat by the seat and extent of the lesion,—a statement which is true of all focal spinal lesions. In a general way, they resemble those of the acute variety of myelitis, save in the fact that their approach is more gradual and unattended with fever.

Hyperæsthesia, numbness, formication, and occasional severe pains in the limbs are commonly observed. These are followed or accompanied by a steadily increasing weakness of the lower limbs, usually associated with imperfect micturition and defecation.

Paraplegia is more frequent than unilateral paralysis in this disease. Whenever the lateral column of the cord is attacked, the gait of spastic paralysis may be induced (p. 163). We are particularly apt under these circumstances to encounter, in addition to the progressive paresis of the legs, contracture, muscular spasms, and exaggeration of the tendon reflexes.

The later symptoms of this affection are similar to those of the acute form already described. In some instances, "bulbar symptoms" (p. 384) may be added to those already mentioned.

Diagnosis.—All forms of paraplegia must be differentiated from each other. Whenever this symptom is clinically encountered, the differential tables given on pp. 422 and 426 will assist the reader in doing so; and the light thrown upon the symptomatology of diseases of the spinal cord in the first section, together with the general remarks on "focal" spinal lesions will aid in localizing the exact seat of the lesion which has induced paralysis.

It is very important in all focal spinal lesions (1) that the exact limits of both the motor and sensory paralyses be accurately mapped out in each individual case; and (2) that the vertical extent of the lesion be determined by testing each of the spinal reflexes. This can be done by the methods already described in Section II of this work. Those only are abolished which depend upon a reflex arc in the diseased segments. If a bed-sore develops it is clinical evidence, as a rule, that the nerves which supply that particular area of skin are involved directly in the spinal lesion.

Prognosis.—Syphilitic cases may make a complete recovery; provided treatment be begun before spastic symptoms become developed. The course of the other varieties of chronic myelitis is very protracted (often ten to twenty years), but is usually fatal.

Treatment.—The treatment suggested for spinal meningitis is applicable to this disease. Erb extols the effects of the "cold-water cure" in Ziemssen's Encyclopedia, and gives some directions for its use. I have never tried it personally.

If spastic symptoms are prominent, ergot and the nitrate of silver act better than strychnia, in my experience. When the paralyzed muscles are relaxed and flaccid, strychnia, iron, arsenic, phosphorus and quinine are of benefit.

I have obtained very beneficial results in several cases by the withdrawal of heavy static sparks from the spine and the paralyzed muscles. This treatment certainly surpasses any other form of electrical application. The machine must have large plates to generate sufficient quantity to yield good results.

SYRINGOMYELIA AND HYDROMYELIA.

Cavities in the substance of the spinal cord may exist either as a congenital or acquired condition. Their extent, situation, and contour vary in different cases. They usually contain a serous or hemorrhagic fluid, and occasionally a hyaline material.

These cavities may be single or multiple. They are most common in the posterior white columns of the cervical and dorsal segments of the cord. They may be totally independent of the central canal of the cord, but they usually communicate with it. The anterior horns are occasionally found to be the seat of such cavities.

Morbid Anatomy.—The wall of these cavities is generally composed of a firm fibrous tissue (which is apt to be friable and highly vascular), or of myxomatous tissue. Its inner surface may or may not be lined with epithelial cells of the cylindrical variety.

Acquired cavities may result from the softening and degeneration of clots or of spinal tumors (chiefly glioma), and from spinal compression. Chronic myelitis may possibly induce cavities within the cord. Meningeal adhesions have been considered by some authors as a possible factor in their development.

Symptoms.—Large cavities in the cord may exist without causing any symptoms during life. If they are created by the morbid conditions mentioned above, the symptoms will be those of the exciting cause.

The situation of these spinal cavities being, as a rule, confined to the anterior horns, the central gray matter and the posterior columns of the cord would naturally suggest the coexistence during life of interference with the sensory, vaso-motor, and trophic functions of the spinal segments involved, as well as more or less atrophy of the muscles, and possibly motor paralysis. The reported cases, where post-mortem observation has confirmed the diagnosis, seem to sustain such a conclusion.

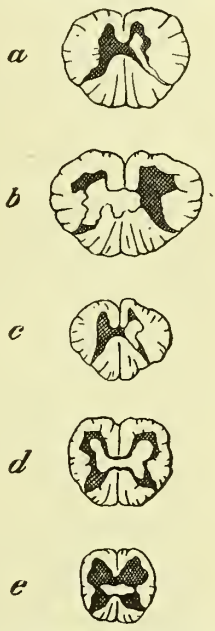


FIG. 120.—CAVITIES WITHIN THE SUBSTANCE OF THE SPINAL CORD, CONSTITUTING THE CONDITION KNOWN AS "SYRINGOMYELIA." *a*, *b*, Cervical cord; *c*, dorsal cord; *d*, *e*, lumbar cord.

Among the *abnormal sensory phenomena* noted by various observers, we find the following mentioned: Analgesia, an imperfect perception of varying degrees of temperature, and occasionally anæsthesia and imperfect localization of touch-impressions. The seat of these abnormal

sensory phenomena depends upon the spinal segments attacked. (See tables on pages 411 and 415.)

The *abnormal trophic or vaso-motor phenomena* may comprise any or all of the following conditions: Eruptions (chiefly of the bullous type), defective secretion of perspiration in some localized form, abscesses or intractable ulceration, fragility or atrophy of bones, lowering of the temperature of some parts, cyanosis, etc.

The *abnormal muscular phenomena* may comprise all the symptoms enumerated when describing the clinical history of poliomyelitis.

Diagnosis.—This disease may be confounded during life with multiple neuritis, amyotrophic lateral sclerosis, poliomyelitis, multiple sclerosis, and spinal tumors outside of the cord.

From *multiple neuritis*, it is told by the fact that impressions of touch, temperature, and pain are not equally and simultaneously impaired, as they are when a nerve-trunk is undergoing extensive degeneration. The history of the patient might also exclude the exciting causes of neuritis.

From *amyotrophic lateral sclerosis*, this affection is told by its being, as a rule, a unilateral spinal affection, and by the absence of the peculiar rigidity of the muscles and the characteristic deformity of amyotrophic spinal sclerosis. Moreover, the duration of life is much longer in syringomyelia.

From *poliomyelitis anterior*, it differs in that sensory symptoms generally coexist with paralysis and atrophy, and also in that the vaso-motor and trophic disturbances are quite marked.

From *multiple spinal sclerosis*, it may be told by the absence of tremor, and the limited number of spinal segments involved in syringomyelia.

From *spinal tumors*, pressing upon one lateral half of the cord, this disease is to be distinguished chiefly by the absence of all symptoms pointing to an implication of the vertebræ.

Prognosis.—Syringomyelia seems to follow a somewhat uncertain course. It may progress slowly, or become stationary for long periods of time. Occasionally it causes a sudden fatal termination.

FUNCTIONAL DISEASES OF THE SPINAL CORD.

In a preceding table (p. 350) we have included under this head the conditions of spinal irritation, functional paraplegia, spinal neurasthenia, writers' cramp or paralysis, and tetany.

Some of these will be discussed under the general head of functional nervous disease, to whose special consideration the sixth section of this work will be devoted. Others demand some passing notice in this section.

SPINAL IRRITATION.

(*Anæmia of the Posterior Columns.*)

Notwithstanding the opinions of many writers to the contrary, it is questionable to my mind whether it is proper to regard this condition as a special form of disease. The symptoms which are generally enumerated under this head are very often nothing more than manifestations of the hysterical or neurasthenic states. It is probably a purely functional derangement, and affects young adults (from fifteen to thirty-five years of age), chiefly of the female sex.

Etiology.—For information on this subject, I would refer the reader to my remarks relating to the causes of neurasthenia and hysteria.

Symptoms.—Pain in the region of the spine and marked tenderness over the spinous processes of the vertebræ and adjacent skin are the prominent symptoms of this affection.

The *pain* is of a most distressing kind, usually described by patients as a severe "ache." It is very commonly experienced between the shoulder-blades and in the lumbar region; but it may extend into the thighs and down the area of distribution of the sciatic nerves.

Nausea and *vomiting* may coexist with tenderness confined to the cervical spines.

The *hyperæsthesia* is often of an extreme kind. It exists over the vertebral spines. The slightest pressure along the spine may call forth evidences of acute suffering on the part of the patient.

The *general health* is usually below par. The urine may be loaded with phosphates, the digestive functions poor, the eyes asthenopic, and the mental condition sluggish and apathetic.

No evidences of impairment of motility are observed, nor is there any anæsthesia. The bladder or rectum is never paralyzed.

Treatment.—The reader is referred to my remarks on the treatment of spinal neurasthenia and hysteria.

FUNCTIONAL PARAPLEGIA.

The lower limbs may sometimes be paralyzed without an organic cause. We encounter this condition chiefly in women and children.

Etiology.—Among the causes of this condition may be mentioned hysteria, anæmia, blood poisons (malaria, lead, arsenic, phosphorus, ergot, alcohol, etc.), ovarian irritation, phimosis, and many other forms of reflex irritation. I believe that "eye-strain" is a factor in these cases too commonly overlooked.

Symptoms.—When *hysteria* exists, Drummond thinks that an insensibility to pain, but not to touch or temperature, is peculiarly characteristic. Duchenne, on the other hand, regards the loss of muscular

sensibility as of great diagnostic importance. Todd has described certain facial changes (p. 170) as of value in recognizing this condition.

Reynolds has described a type of functional paraplegia "*dependent purely upon idea*," in which the patient is strongly impressed with the fact that voluntary movement is impossible. He claims that this state is not necessarily hysterical.

In *malarial paraplegia*, the paralysis is said to be intermittent in type. I have never observed a case of this kind.

In *reflex paraplegia*, the bladder, external genitals, urethra, ovaries, and the eye may act as the exciting cause.

In *alcoholic paraplegia*, the symptoms of "multiple neuritis" are apt to be encountered; hence this condition is not purely functional in many cases. Pains in the limbs and the coexistence of the "reaction of degeneration" (p. 189) are diagnostic of the latter condition.

Treatment.—The removal of the cause and steps judiciously directed toward the improvement of the general health are indicated. Electricity, massage, tonics, etc., are beneficial. In my opinion, a correction of "eye-strain" will generally prove of immediate service to a large proportion of adult cases.

WRITERS' CRAMP.

(*Professional Cramp; Mogigraphia; Graphospasm; Cheirospasm.*)

Writers, pianists, violinists, telegraphers, the counters of paper bills, etc., often become unable to pursue their vocations from a peculiar form of weakness and pain, or a tendency toward sudden spasm of the muscles of the hand and forearm.

Etiology.—Any occupation which requires incessant use of a certain set of muscles of the forearm or hand may lead to this distressing condition. It is doubtful to my mind if injuries, sprains, or exposure to cold ever induced this morbid state. I regard causes of that character as mere coincidences.

Symptoms.—This condition develops gradually. The patient feels at first a peculiar sense of distress or fatigue in performing for any length of time the vocation which has induced it. This sense of distress may be in the fingers or forearm. It is accompanied sooner or later by a peculiar awkwardness in the finger-movements, a sense of stiffness in the fingers, or a tendency to uncontrollable spasm of the fingers when these acts are persisted in.

Gradually these symptoms increase in severity. The fingers become more and more uncontrollable when used by the patient in his vocation. For example, when writing, the pen may be flung from the hand or pressed violently upon the page. A pain becomes marked along the arm, often as high as the shoulder. After ceasing all attempts at writing,

the limb affected may feel relieved by rubbing it and kneading the muscles for some time. In many cases, the vocation which has occasioned the cramp has to be abandoned.

Strange as it may seem, these patients can use their affected hand for any other purpose with their accustomed facility. I have seen cases where the patient could draw for hours but could not write for one minute without distress. Some sufferers learn to use the left hand, so as to avoid using the afflicted member. If the left hand is then overtaxed, the condition tends to become bilateral.

This disease is very persistent, after it is well-developed. I personally suffered from it for many years; and am still unable to write continuously with a pen for any length of time without severe distress. I can use a type-writer, however, for hours without the slightest symptom of cramp.

Respecting the morbid anatomy of this disease, many theories have been advanced. Althaus regards it as an exhaustion and abnormal irritability of the coördinating centres in the upper part of the cord. Some authors consider it an affection of the muscular system only or of the terminal plates of the nerves. Ross claims that he can locate the disease by the electrical reactions of the affected muscles. He places it in the ganglionic spinal cells, when the reactions are diminished; and in the cortex, when the reactions are intensified.

Treatment.—Entire rest from the occupation that causes distress is the first step in the treatment. This must be ensured for many months, if possible.

Some patients who cannot do this are benefited by wearing a rubber band around the forearm; others by holding the pen in an unusual way; a few, by employing a cork pen-holder of an extreme size (often over an inch in diameter); while many have recourse to a type-writer for correspondence.

Showering the arm in hot and cold water alternately, and using friction, percussion of the affected muscles, and massage (Wolff's method) after the water application is often very beneficial.

Blisters and the actual cautery over the median nerve is of service in many cases. It must be kept up for some weeks

Static sparks to the cervical spinal segments and to the affected forearm and hand often give immediate relief.

TETANY.

This condition is characterized by paroxysms of tonic muscular spasm confined to groups of muscles. It is also known as "*intermittent tetanus*" and "*intermittent cramp.*"

The upper extremities are most often attacked. Generally the

spasms are bilateral in character. In exceptional cases they may be unilateral. Sometimes the spasms are confined to the legs, and occasionally the muscles of the back, thorax, and abdomen may be involved. Cases where the attacks have been general in character, affecting the limbs, trunk, and even the face, have been reported.

Etiology.—This disease is most frequently encountered in children at the time of dentition, and at the age of puberty. It is rare in advanced life.

Heredity seems to be apparent in some cases. Several of one family have been so afflicted, according to Murdoch, and the researches of Bouchut seem to show a history of neurotic affections in the ancestral line of many so afflicted.

A *state of low vitality* is generally present in these subjects. Rickets, acute infectious diseases, impaired digestive functions, etc., are among the predisposing causes.

Among the *exciting causes* may be mentioned a marked exposure to cold or dampness, rheumatism, peripheral irritation of all kinds, and violent mental excitement.

Morbid Anatomy.—Little is positively known respecting the morbid changes which probably exist in the nerves or the nerve-centres. Weiss believes that diseases of the sympathetic system exists and induces circulatory changes in the spinal cord.

Symptoms.—These may be grouped into two classes, the prodromal and the actual.

The prodromal symptoms may include pains in the limbs, formication, coldness of the extremities, vertigo, a sense of confusion in the head, and tinnitus aurium. They may exist for days or weeks prior to the attack.

The symptoms of an attack may occur after mental excitement or excessive muscular effort. They may occur at night or during the day.

When the *upper limbs* are attacked, the flexors of the fingers (usually of each hand) and also the flexors of the wrist cause the attitude of the hand to assume a position which Trousseau very aptly compares to that of an obstetrician when about to pass the hand into the vagina. Occasionally the forearms are flexed, and the arms are drawn to the chest to an extent sufficient to cause a crossing of the distorted hands over the epigastrium. In very exceptional instances the spasm is unilateral and the extensors may be attacked. During the paroxysm the muscles are very prominent and firm, and are sensitive to pressure.

When the *lower limbs* are attacked the foot is distorted at the ankle by spasm of the calf-muscles, the leg is extended upon the thigh, the big toe is drawn beneath the adjacent toe, and the thighs are strongly adducted.

When the *trunk* is attacked the back muscles may cause opisthotonos or pleurosthotonos. Again, the spine may be bent anteriorly. The chest-muscles may cause disturbances of respiration of an alarming kind. The muscles of the neck may create cyanosis, prominence of the jugulars, and protrusion of the eyeballs.

During the paroxysm the contractures may be partially overcome by a voluntary effort, but the deformity returns at once when the effort is suspended. The contractures may even persist during sleep. Fibrillary contractions are often observed during the paroxysm.

The *duration* of the attacks varies from a minute to several days. They may return with great frequency or at long intervals.

The *attacks are not excessively painful*, as a rule. They are generally accompanied by a sense of tingling, formication, coldness, or slight neuralgic pains of a shooting character. Fever and sweating may be observed in some cases.

Trousseau lays much stress upon the diagnostic importance of a test to be employed during the intervals between the paroxysms, which consists in the ability to *induce these attacks at will by pressing upon the arteries or nerve-trunks* of the arm. After such pressure of two or more minutes the spasm occurs. It rapidly disappears when the pressure is removed. The same test can be applied to the crural artery and the sciatic nerve, but with more uncertainty.

The *electrical irritability of the affected motor nerves* is markedly increased. The nerve responds to abnormally weak faradaic currents. Applications of galvanic currents to the nerve-trunk by the polar method show the following conditions: C.C.C. and A.O.C. occur very early; cathodal-closure-tetanus and anodal-closure-tetanus are rapidly developed; finally, anodal-opening-tetanus is produced in almost every case with ease, and cathodal-opening-tetanus in some cases.

Diagnosis.—This disease may be confounded with tetanus, hysterical contractures, and ergotism.

In *tetanus*, there is an inability to use the muscles of mastication, more pain, a traumatic history, and a general rigidity and abnormal posture of the limbs and trunk.

In *hysterical contractures*, the test of Trousseau is inoperative, there is no increase of the mechanical and electrical irritability of motor nerves, children and males are seldom attacked, and the history of the case is suggestive of hysteria.

In *ergot poisoning*, the history of the case would point clearly to the exciting cause of the attacks.

Prognosis.—These sufferers usually recover perfectly after a lapse of time. The disappearance of Trousseau's phenomena, and the abnormal irritability of the motor nerves, is indicative of a favorable change in

the patient. Recurring paroxysms are to be anticipated for some months after the first attack.

Treatment.—If a history of rheumatism or a rheumatic tendency can be elicited, it is well to give iodide of potassium, salicylic acid, or the oil of wintergreen. Ice-bags, wet-cupping, and blisters to the spine; the application of the actual cautery, and galvanism to the spine have been recommended by different authors.

The general health of the patient should be restored by all judicious means. Tonics, massage, good hygiene, nutritious food, stimulants in moderation, and moderate exercise will conduce toward that end.

Among the electrical applications, static sparks to the limbs and spine, general faradization, the polar action of the anode to tender points applied by the stabile method, and labile applications of the anode to the peripheral nerves (stroked slowly from the distal extremity of the nerve toward the proximal end) have proven of service in many cases.

THOMSEN'S DISEASE.

(Myotonia Congenita—Congenital Muscular Spasm.)

In this disease, a *tendency of the muscles to tonic spasm* during attempts at voluntary movement is the characteristic feature.

By such spasms, the execution of intended movements of the limbs is always more or less delayed, and sometimes entirely prevented.

This disease is also known as "Myotonia Congenita," because it is seldom, if ever, observed except in patients who are not predisposed to it by heredity. Dr. Thomsen, who first described this affection, noted its occurrence in five generations of his own family. He suffered from it himself, as did also one of his sons. A very complete monograph on this subject has been published by Erb, who has collected and analyzed all cases reported to that date. Jacoby and Dana have lately added to the literature of this affection.

Etiology.—As has already been stated, heredity plays a very important part in this disease. In one reported case, fright seems to have acted as an exciting cause. It is questionable, however, if this disease ever occurs without some congenital defect either in the spinal cord or in the muscles themselves. A late monograph upon this subject by Dr. G. W. Jacoby seems to show conclusively that muscular anomalies were present in the case reported by him.

Morbid Anatomy.—Although this disease has been classed by me as a functional disease of the spinal cord (because no spinal changes have ever been shown to exist in connection with it), it must be said that the muscles appear to show characteristic conditions which are probably congenital. The individual muscular fibres are greatly augmented in

point of size, and the number of their nuclei is in excess of that observed in healthy muscle.

The muscles are generally unnaturally large in this disease. This gives to the patient an appearance of strength, which is in marked contrast to the actual power of contraction which the patient possesses. The anomalies of muscular construction which have been referred to necessarily add to the size of each individual muscle. But, on the other hand, such a muscle appears to be more liable to become tetanic when called into play by the act of will.

Symptoms.—Typical cases of this disease exhibit in very early youth, to a moderate degree, the disorder of movements, which becomes more pronounced later in life. A history of a similar affection can be found upon inquiry to have existed in some of the patient's ancestry. After a period of rest the patient experiences a peculiar tension and stiffness of the muscles when any voluntary movement of the limbs is attempted. This stiffness may be so marked in some cases as to completely arrest the intended movement for a time. It gradually disappears, however, and, by the aid of continued movements, the patient after a time regains complete control over his muscles.

In addition to this peculiar muscular state, the patient is also rendered unable to voluntarily relax the muscles quickly.

The muscles of the lower limbs are more frequently affected than those of the upper. In some cases, the muscles of the tongue, face, eyes, and also those of mastication, are affected. Involvement of the tongue by spasms of this character gives rise to a peculiar hesitancy in speech. Awkwardness in the mastication of food is observed whenever the muscles which move the lower jaw are attacked.

When the muscles of the lower limbs are affected with this disease the patient is very apt to experience great difficulty in attempting to rise and walk, after a prolonged recumbent or sitting posture. Such subjects have been known to fall as soon as efforts to walk were attempted. Fibrillary contractions of the muscles may occasionally be detected. Continued movement and the application of heat tend to diminish the spasm, while mental excitement and cold usually aggravate it.

Again, the muscles in these patients show an abnormal excitability to mechanical and electrical stimuli. Artificially produced contractions are apt to be very much prolonged. Erb describes peculiar "wave-like contractions" in the muscles of the limbs, whenever galvanic currents of sufficient intensity are employed upon the patient by the stabile polar method. These contractions, according to this author, always tend to pass toward the anode. After a time they subside "like waves of water produced by a falling stone." Any increase of the strength of the current, however, tends, as a rule, to reproduce them.

To test this reaction in the upper extremities, one pole may be placed at the nape of the neck and the other in the palm of the hand or at the annular ligament of the wrist-joint on its palmar aspect. To test it in the lower extremity, one pole should be at the neck and the other may be placed adjacent to the patella or upon the tendo-Achilles. The strength of the current employed varies from six to twenty milliampères. Jacoby has observed an absence of any fixed relationship of Ca.C.C. and An.C.C. to each other, as exists in healthy muscle (page 190).

The duration of this disease is limited by the life of the patient; although remission and exacerbations have been described by different observers.

Diagnosis.—This disease is to be distinguished from muscular hypertrophy by the presence of the spasms, and the peculiar electrical phenomena already described. The reflexes give evidence, also, of an unusually prolonged muscular response.

Treatment.—Gymnastic exercises, warm baths, and judicious electrical treatment may possibly afford some relief.

ACUTE ASCENDING SPINAL PARALYSIS.

(*Kussmaul-Landry's Paralysis.*)

This disease, as far as we at present know, is not associated with anatomical changes in the nervous system. It consists of a tendency toward progressive paralysis, which slowly creeps from below upward in a more or less irregular way. There is an absence of atrophy; and no sensory or trophic disturbances are observed. There is no paralysis of the bladder or rectum. The irritability of the paralyzed muscles is retained.

Etiology.—This disease is a rare one. It is more common among males than females; and, as a rule, it affects middle life.

Its exciting causes are very obscure. It has been observed to follow mental excitement, exposure to cold, suppressed menstruation, acute infectious diseases, coitus in the standing posture. The syphilitic history may be detected in a certain proportion of persons so afflicted.

Morbid Anatomy. — Little if anything is known respecting the changes which occasion this disease. Westphal concludes from his investigations that it is the result of some unknown infection; because he detected changes in the intestinal follicles and the mesenteric glands in a number of cases.

Symptoms.—The paralysis may develop suddenly; or it may be preceded by slight fever, pain in the back and limbs, tingling and other forms of abnormal sensation. The paralytic symptoms do not always follow a strictly ascending course. They may begin in one or both feet and then skip to the upper extremities, the neck, chest, or abdomen.

This, however, is not always the case. In rare instances, the impairment of motion has apparently pursued a descending course; and, in one case reported by Westphal, the nuclei of the medulla were alone implicated and "bulbar" symptoms appeared at the onset.

In most cases, a parcsis first appears; this subsequently deepens into complete paralysis. A sense of fatigue in the limbs is first noticed by the patient, and walking soon becomes extremely difficult. For this reason these patients usually take to bed early.

When the back muscles become paralyzed, it is impossible for the patient even to sit up. Paralysis of the muscles of the abdomen renders coughing, sneezing, expiration, defecation and micturition difficult. When the intercostal muscles are paralyzed, inspiration is seriously disturbed, and the most marked difficulty in breathing may occur whenever the phrenic nerve becomes affected. Sooner or later the movements of the upper extremities are rendered difficult or are totally lost. Whenever the medulla is implicated, speech becomes very much impaired, and the act of swallowing may be attended with great difficulty. It is very rare to observe any paralysis in the nerves of cerebral origin.

No atrophy is detected in the paralyzed muscles, and they retain their normal irritability to electrical stimulation.

In very exceptional instances only do the sensory functions give any evidence of serious impairment. Cases have been reported, however, where the sensations of pain and temperature have been imperfectly conducted, and where the muscular sense has been somewhat diminished Anæsthesia and hyperæsthesia have also been observed. There seems to be a tendency to diminution or abolition of the skin and tendon reflexes late in the disease.

In some cases a marked enlargement of the spleen and clinical evidences of albuminuria have been detected.

Diagnosis.—This disease may be confounded with an ascending myelitis, poliomyelitis anterior acuta, and acute multiple neuritis.

From *myelitis* of the *ascending* type, it may be recognized by the absence of fever and sensory disturbances, by the fact that bed-sores do not occur, and by the non-occurrence of vesical and rectal complications.

From *poliomyelitis*, it may be told by its progressive character, and the absence of rapid atrophy in the paralyzed muscles. The "reaction of degeneration" is present in poliomyelitis; while it is generally absent in ascending paralysis.

From *acute multiple neuritis*, it differs in that marked pain and sensory disturbances are usually absent, and in the fact that the affected nerves and muscles do not rapidly lose their irritability to electrical currents.

Prognosis.—This disease usually runs an acute and progressive course; hence the prognosis is naturally grave, although recovery has been observed. The development of "bulbar" symptoms generally indicates the approach of a fatal termination. The more rapid the development of paralysis of a complete kind, the more serious is the outlook for the patient. The duration of the disease is generally a short one. It may prove fatal in from four days to as many weeks.

Treatment.—If the disease can be shown to be connected with any of the clinical manifestations of syphilitic infection, the remedies suggested on page 291 should be administered. It is well to make use of the actual cautery, dry cups, or ice-bags to the spine. The internal remedies suggested by authors comprise the iodide of potash, full doses of ergot, belladonna, and strychnia. The galvanic current may be applied to the spine, preference being given to the polar action of the cathode.

ABNORMAL VASCULAR CONDITIONS OF THE SPINAL CORD AND ITS COVERINGS.

Under this head I have included, in a previous table, spinal congestion, spinal anæmia, spinal embolism, atheroma of the spinal vessels, fatty degeneration of the vascular coats, and aneurismal dilatations.

Of these, only the first two can be described as conditions which are clinically recognized. The other four are pathological states which tend when present to induce structural changes within the substance of the spinal cord. They are more directly concerned, therefore, with the etiology of organic spinal diseases than with their symptomatology. One form of spinal anæmia has been already considered under the head of "spinal irritation."

SPINAL CONGESTION OR HYPERÆMIA.

The distinction between congestion and hyperæmia is one of degree rather than of kind. In both conditions we encounter dilatation of the vessels with an excess of blood. In hyperæmia, the current is unusually rapid; in congestion, it is unnaturally slow.

Clinically, the line of distinction between hyperæmia and inflammation is very difficult, if not impossible, to draw. One may be simply a precursor of the other. As the vessels of the pia are the chief sources of supply to the spinal cord, hyperæmia of the cord and meninges usually go hand in hand. Its symptoms must, therefore, be of necessity closely allied to those of spinal meningitis and myelitis. When the pia is diseased, the spinal cord is almost invariably affected simultaneously to a greater or less degree.

Etiology.—A sudden checking of the perspiration by draught of cold air, bathing, etc., is generally regarded as tending to excite this condition.

Excessive fatigue, violent excitement, unnatural indulgencies in venery, suppression of the menstrual discharges, the effects of compressed air, prolonged physical or mental exertion, blows and falls, etc., have also been mentioned by some authors as apparent causes of spinal hyperæmia.

Personally, I am inclined to believe that most of the symptoms usually attributed by authors to this morbid state are dependent upon a neuropathic tendency whose exciting causes will be discussed in full in the section which relates to functional nervous diseases.

Symptoms.—These are to be attributed in a general way either to irritation or a state of depression of the spinal functions. They may, therefore, vary with each case, and closely simulate the first symptoms observed in spinal meningitis, spinal tumors, and myelitis.

Hammond, Browne-Séquard, Radcliffe, Ollivier, and others, who have written upon this condition, describe among the symptoms many clinical features which, in my opinion, are not always distinguishable from those occasioned by the organic diseases mentioned. Thus, for example, pain, disturbances of motility and sensation, the cincture-feeling, a lowering of the temperature in parts below the lesion, interference with breathing and the action of the heart, a loss of control of the bladder and rectum, a diminution of the electro-muscular contractility, the development of bed-sores, etc., are what we are apt to observe whenever the spinal cord is subjected to irritation or when its functions are in any way interfered with. The clinical history of each case, combined with prolonged observation of the patient, can alone enable us to exclude organic spinal changes.

Respecting the *pain* of spinal congestion, it is claimed that the recumbent posture increases it; and also that the standing posture adds to the distress when the congestion is localized in the lower spinal segments. This is attributed to the effects of gravity. It is also stated that a sudden blow or shock, as a false step, for example, adds to the pain in the spine.

Anæsthesia, or a *sense of tingling and formication*, may exist in the feet (chiefly in the plantar surface of the toes) whenever the dorsal or lumbar segments are locally congested.

Paresis of the legs, or *actual paraplegia*, may be developed. The patient can usually move the limbs when sitting or in bed, although they may be incapable of supporting the body.

According to Hammond, the symptoms of spinal congestion are always more marked on rising than as the day advances.

Diagnosis.—This condition may be confounded with spinal anæmia, myelitis, spinal meningitis and spinal tumors.

In *spinal anæmia*, the bladder, when affected, is impaired before the

development of motor weakness in the legs, while the reverse order is observed in spinal congestion. Hyperæsthesia is developed in place of anæsthesia and formication. The effects of a recumbent posture tend to cause an improvement in the symptoms.

In *myelitis*, the urine is apt to become alkaline, irrespective of decomposition from retention within the bladder. Moreover, the paralysis is more decided, the development of bed-sores more frequent, the cincture feeling is more decidedly marked, and the pain in the cord is more severe.

In *spinal meningitis*, the tendency to muscular spasm, the pain on movement of the spine and of the paralyzed limbs, the febrile symptoms, the muscular twitchings, and the tendency toward muscular rigidity are all in contrast to the symptoms of simple congestion.

In *spinal tumors*, the loss of motility is most marked upon one side, and sensory disturbances (anæsthesia) upon the other. There is also a history of tubercle, cancer, or syphilis. The spinal symptoms develop very gradually, as a rule.

Prognosis.—There is a tendency in all cases of spinal congestion for the disease to progress along the cord. Moreover, the development of structural disease of the cord is liable to be a result of excessive vascularity. The prognosis is not unfavorable, if the case be one of a localized type and unaccompanied by organic or inflammatory disease of the cord or its membranes.

Treatment.—In cases of an acute character, where the symptoms develop rapidly, *leeching the anus* will indirectly deplete the cord, and *dry-cups* over the spine may also tend to relieve the congestion. Hammond also suggests the daily use of three drachms of the sulphate of magnesia in divided doses to cause watery stools, which require a determination of blood to the intestinal canal.

Ergot should be administered in large doses. I have given it in doses of a drachm of the fluid extract after each meal for many weeks at a time to patients without any symptoms of ergot poisoning. Belladonna, in doses of fifteen drops of the tincture, may be given with benefit three times a day.

The employment of the *hot douche* to the spine—the water being poured from a height of two feet upon the bare back for five minutes daily—is highly recommended by Hammond.

Electricity is of service in the treatment of this disease. I prefer the withdrawal of static sparks from the spine to galvanism or faradism. I have also employed the same treatment to the paralyzed muscles with good results.

Strychnia and phosphorus are *strongly contra-indicated*, according to Hammond.

SPINAL ANÆMIA.

One form of this condition has already been discussed under the head of "spinal irritation." This disease is believed by some observers to depend upon an anæmia of the posterior columns of the spinal cord.

Another variety is thought to affect the *antero-lateral columns* of the spinal cord (Fig. 91). If this condition be recognized as a distinct disease, the symptoms will be of necessity connected with motility; and possibly with exaggerated reflexes, contracture, and atrophy. It will also cover all of the so-called "functional paralyses" whose pathology is now unknown.

I cannot express my full concurrence with these views; but, with deference to those advanced by others, I shall here give the main features of the disease as generally taught.

Etiology.—Extreme cold, sleeping on damp ground, exhausting diseases, spinal embolism, thrombosis or atheroma, and interference with the circulation through the abdominal aorta, from compression, thrombosis or aneurism of that vessel, may cause spinal anæmia. Moreover, the spinal vessels may be influenced to contract through the agency of the vaso-motor nerves, as an indirect result of peripheral irritation from any cause, such as the ovaries, intestine, genitals, eye-strain, injuries to nerves, etc.

Symptoms.—The affected segments of the cord give evidence of deficient blood-supply early by *paresis* of certain muscles. It is claimed that the anterior tibial muscles and the peronei seldom escape. The paresis rarely prevents walking, although the gait is generally feeble and the patient's endurance slight. The upper limbs are seldom paretic.

The *sphincters* of the bladder and rectum are seldom affected; and the paresis of the limbs is not usually progressive in type.

Sensory disturbances are infrequent. The cincture feeling is not developed.

The *reflexes* may be normal or exaggerated slightly. They are never abolished.

Prognosis.—If the exciting cause can be removed, the chances for a complete recovery are good; if not, the spinal cord may undergo softening.

Diagnosis.—The chief points, which relate to the discrimination between this disease and spinal congestion, have already been given (page 445).

Treatment.—The utmost care should be exercised in ascertaining the cause. My remarks concerning the effect of "eye-strain" in a preceding section should be carefully considered, and all necessary tests should be made early to determine the condition of this organ and its

museles. In the light of late researches made in this direction, I am inclined to discredit the value generally placed by the profession upon many of the other reflex causes enumerated, although more than one cause may exist in any individual case.

The general treatment should be directed toward improving the vitality of the patient.

SECTION V.

FUNCTIONAL NERVOUS DISEASES.

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Under this heading I propose to discuss certain abnormal conditions of body, in consequence of which some special form of disturbance or derangement of the nervous functions may be exhibited, which has not, as yet, been shown to depend upon any positively recognized pathological state.

Among this class of conditions may, in my opinion, be included a certain percentage of *epilepsy*, *chorea*, *hysteria*, *and hystero-epilepsy*. In this percentage, the existence of organic lesions can be excluded. Again, *neurasthenia* (with its endless variety of manifestations), typical attacks of *migraine* or "*sick headache*," certain obstinate types of *neuralgia*, and, in some cases, evidences of *imperfect performance of some of the functions of the abdominal and thoracic viscera*, are unquestionably to be regarded as functional neuroses.

I am aware that I am at variance with the majority of authors in thus grouping so many diseased conditions that are apparently discordant under one head. I may be severely criticised possibly by some for so doing. I may even be taken to task for the selection of the term "functional nervous disease," which is rejected by many enthusiasts in pathological research.

To show, however, that I am not alone in the position taken, I take the liberty of quoting the following paragraphs from the preface of a late work* upon this special field :—

"Pathological anatomy has exercised such an enormous influence upon the advances made in practical medicine within the last twenty-five years, that many pathologists sneer at the term 'functional' disease, and deny its very existence.

"While we fully agree that there can be no morbid manifestations without a change in the material structure of the organs involved, we are nevertheless fully convinced, in view of the fruitless search of pathological anatomists, that the diseases which we have considered in this work present no primary anatomical changes which are visible to the naked eye or the microscope; in other words, that the changes are of a molecular nature."

While the truth of this statement appears to me self-evident, I have, moreover, other reasons than those urged by this author for including

* Putzel—"Functional Nervous Diseases," 1880.

under the term "functional" nervous diseases, the abnormal states specified by me, as will appear later. These will be more apparent when I call attention to what I regard as of vital importance in some of these cases.

THE RELATIONSHIP BETWEEN FUNCTIONAL NEUROSES AND ANOMALIES OF THE VISUAL APPARATUS.

The study of defects in the adjustment of the eye-muscles and the relationship which exists between such defects and nervous diseases, has not been generally regarded as of very great practical importance until of late. Many of our best text-books upon the eye do not deal with any such muscular defects, except in relation to strabismus. Some give directions for testing the ocular muscles, that are in direct opposition to the views which are here advanced. A few are positively misleading; chiefly on account of errors of statement concerning points where physiological optics come into play.

I may be pardoned, therefore, if I review, in a general way, a few points which have a practical bearing upon a method of examination and treatment of the visual apparatus, which is to-day exciting considerable attention among scientific medical men, especially among those whose interest centres in the study of nervous diseases and in ophthalmology.

What I have to say here includes the discussion of the following points of inquiry:—

(1) What steps may be deemed as essential to success in the diagnosis and treatment of certain anomalies of the visual apparatus.

(2) Why it is that observations in this direction, when too hastily or imperfectly made, are peculiarly apt to be untrustworthy.

The limits of a few pages will hardly suffice for me to cover more than a few of the more important points comprised under these headings. What I have to say will, therefore, be as condensed as seems to me permissible. A personal experience, derived from several years of continuous research in this field upon a class of patients afflicted exclusively with nervous derangements, and from more than five hundred graduated tenotomies upon the recti muscles of the orbit, justifies me, I think, in expressing positive convictions.

The views which I shall discuss here constitute the basis of a systematic method of examination for and treatment of certain ocular defects, whose relationship to functional nervous diseases seems to me to be now established beyond dispute.

Since these views were first advanced by Dr. George T. Stevens, they have attracted no small amount of professional attention. In spite of the fact that his contributions in relation to this subject are remarkably clear and succinct, considerable misapprehension still appears

to exist in the minds of the profession at large relative to the views advanced by him.

I may be pardoned, therefore, if, as an exponent of these views, I repeat in substance much that has already appeared in print. By so doing, I hope to concentrate attention upon certain steps employed in the examination of the visual apparatus, whose order is deemed by no means unimportant, and in some of which the observer should exercise no small amount of care.

The following statements are, therefore, deemed by me as worthy of your attention :—

(1) The view is held that *errors of refraction* (by which I mean near-sightedness, far-sightedness, or astigmatism) *often modify apparent muscular anomalies to such an extent as to render the early detection and correction of refractive errors imperative.*

This point is of vital importance in the treatment of many patients. Clinical observation has conclusively shown that one of the most important steps in correcting what is commonly known as a "squint," or "cross-eye," is first to properly detect any existing error in refraction and to properly correct it. Such defects should always be sought for early, and the effect of a proper glass upon the deviation of the axes of vision from their normal position which demands relief should first be carefully noted. Many cases are observed by oculists where spherical glasses alone have corrected a marked "squint." The neglect of this important step may prove to be a serious omission, as it may lead to an error in diagnosis or treatment. Let me impress upon you the fact that each eye of every patient must be separately examined for refractive errors, and rendered as nearly emmetropic as possible, before any test relating to the ocular muscular conditions can be considered as reliable. It is not enough, therefore, for a neurologist to provide himself simply with a set of prisms with which to examine his patients' eyes for suspected muscular errors. Any tests so crudely made are certainly unscientific, and probably inaccurate.

(2) The view is held that *errors of refraction can only be positively determined after the full effects of atropine; hence the step of dilating the pupil is deemed of importance in most cases.*

There are two sources of error which are possible in all ophthalmoscopic examinations as a step toward the determination of refraction.

The first of these is that the observer may not be able to perfectly relax his own "accommodation" while using the instrument. Most oculists of large experience believe that they can do this with certainty,—a belief which, in my opinion, is perhaps not always well founded. The second source of error lies in the "accommodation" of

the patient. This cannot always be relaxed by instructing the patient to look at an object twenty or more feet distant from the eye.

I am satisfied that mistakes in the determination of refractive errors by the ophthalmoscope are far more frequent than are generally supposed.

For the past four years I have examined the eyes of every patient intrusted to my care by the aid of test-type both before and after the pupils have been fully dilated by atropine. I am not aware that I have ever lost a patient by the use of this drug. In my experience, intelligent persons are always willing to submit to a temporary inconvenience for the purpose of obtaining positive information respecting any point that is deemed of scientific value in relation to themselves. I have personally come to regard the ophthalmoscope as an unreliable instrument for the determination of refraction. Its use is rendered compulsory, however, in very young children, and in those who, from ignorance or feeble-mindedness, are unreliable in their reading of test-type.

It is generally accepted, furthermore, among our best oculists that astigmatism (a recognized source of nervous perplexity) is always estimated more accurately with the pupil widely dilated by atropine than with the normal pupil.

The reasons which I have already given must suffice to explain why the use of atropine constitutes a most important preliminary step to the detection and estimation of any error in the eye-muscles, although many other arguments might be brought forward to prove its advisability in some subjects.

(3) The view is held that *no examination for suspected muscular error in the orbit should be regarded as conclusive for diagnosis, or as a basis for any surgical procedure, until the eye has been proven to be free from refractive error, or rendered as nearly emmetropic as deemed advisable* by properly selected glasses.

It is, of course, advisable during the first interview with each patient to note and record any "manifest" defect in sight. If such exists, each eye should be provided with the glass which gives the best vision for each eye (the two eyes being always tested independently of each other). After such correction, the different tests employed to detect muscular anomalies should then be made, and the results of each test should be recorded as the "manifest muscular error."

At the second interview, with the pupils fully dilated by atropine, the same steps should be repeated. We thus learn, in many cases, the existence of refractive conditions which the first interview did not reveal. We record such as "latent" refractive conditions. By the aid of suitable glasses, any latent refractive error found is then to be corrected; subsequently, at this interview, the muscular movements are to be tested with each eye temporarily adjusted to distant vision by suitable glasses.

(4) The view is held that *all tests employed to detect muscular anomalies must be made with the test-object* (preferably a candle flame) *at a distance of at least twenty feet from the eye.* In this respect, the method of conducting examinations advocated here is somewhat at variance with that commonly described in most text-books.

In the practical office work of many oculists the so-called "line and dot" test is generally employed (at a distance of fourteen inches from the eye).

It is usually advisable to employ this test in addition to the "candle flame" test at twenty feet; but, when it is employed, the results obtained by each test should be separately recorded. The words "in accommodation" have been suggested by Dr. Stevens as a suffix to designate the results obtained when the test-object is placed at fourteen inches from the eye.

While it is deemed desirable in most instances to record the results of both tests described above, all operative procedures are invariably based upon the results obtained by placing the test-object at a distance of twenty feet from the eye.

To a lack of uniformity in the tests made by oculists to detect muscular anomalies in the orbit many of the discrepancies frequently met with between observations made by different men upon the same patient are unquestionably due. For example, a patient may exhibit an insufficiency of the externi at twenty feet, and of the interni at fourteen inches, in spite of the fact that all precautions have been taken to previously rectify existing refractive errors. This field is too large to discuss here, but it is a very important one.*

(5) The view is held that *observations made for muscular anomalies in the orbit, when the test-object is within the limits of accommodation, are not usually reliable as a basis for operative procedure undertaken for the relief of such anomalies.*

Experience goes to show that deviations of the visual axes observed when the test-object is placed at twenty feet from the eye more correctly represent the muscular error which needs correction in any given case than when made at a nearer point.

I have encountered several interesting cases where extremely satisfactory results upon functional nervous phenomena of a distressing type have followed an operative procedure upon the eye-muscles, which would have been strongly contra-indicated if I had attached as much importance to the results of tests made with the test-object at fourteen inches from the eye as the statements found in most of the text-books

* See articles by G. T. Stevens, in *New York Medical Journal*, December, 1886, and in *Archives of Ophthalmology*, June, 1887; also a paper read by the same author before the International Medical Congress at Washington, D. C., September, 1887.

would justify. These cases impressed me very strongly at the time. They bear the strongest testimony in favor of the view that convergence of the eyes is a factor which should be eliminated as far as possible in searching for muscular anomalies of the orbit.

(6) The view is held that *muscular anomalies in the orbit may be partially or totally "latent."*

The amount of muscular error detected in any given case does not necessarily indicate the full amount of error that actually exists.

The results of ordinary tests simply tell us how much eye-tension exists which the patient cannot overcome by any effort of which he is capable.

Upon this one point too great stress cannot be laid, as it sheds much light upon the clinical history of many patients who suffer from eye-strain.

All authorities recognize the fact to-day that a patient may have a very marked congenital shallowness of the eye, and apparently have normal vision, or possibly appear to be even near-sighted, prior to the use of atropine. Subsequently to its use, the same patient will, however, show a high degree of far-sightedness (hypermetropia), because the ciliary muscle (temporarily paralyzed by the atropine) cannot overcome, or (to speak more technically) compensate for the abnormal shallowness of the eye.

Unfortunately for science, we have as yet no drug which aids us in determining the existence of a "latent" muscular error in the orbit.

Yet, are we justified in concluding that latent muscular anomalies do not exist? Most assuredly not. There is the strongest clinical evidence to the contrary.

Only a few weeks ago, I examined the eyes of a prominent physician on three consecutive days, and I was unable to detect (either before or after prismatic exercise of his eye-muscles) any change in his ocular condition from the one noted at the first examination. His symptoms, however, led me to believe that a greater muscular error existed than he showed, although the anomaly detected was a very marked and important one.

I therefore instructed him to wear a prism, which nearly corrected the error then detected, until the next examination. Less than two hours later, I accidentally had the opportunity of again examining his eyes. His muscular error was then exactly double what it originally appeared to be. He was again given almost a full prismatic correction for the defect detected. Twenty-four hours later he was examined for the fifth time, and he still showed an excess of two degrees over the record of the day previous. He was again given a further prismatic correction; but from that time he failed to exhibit any further alteration in his ocular

tests. The relief afforded by prisms was so instantaneous and permanent (while they were worn) as to prove conclusively that the prisms were wisely selected, and that the "latent" insufficiency, which was developed after and by means of their use, more accurately represented his true condition than did the original observations made at the first interview.

I mention this case, not because it is at all unique (for many such instances have been observed) but because it illustrates admirably the existence of latent insufficiency, which happened in this case to be developed rapidly by the temporary use of correcting prisms.

In the second place, it is not at all uncommon to observe the development of latent muscular anomalies in the orbit after a graduated tenotomy has been satisfactorily and scientifically performed for the correction of a "manifest" muscular error. Sometimes, quite a long interval elapses before latent insufficiency shows itself. Again, it shows itself almost immediately.

An epileptic, upon whom I operated for eye-defect, and who has now been free from attacks for over one year and a half, in spite of the cessation of all drugs, showed me originally only one degree of esophoria. This defect would, I think, have been heretofore regarded by most oculists as hardly worthy of correction—even by a prism. The subsequent treatment of this case demanded repeated partial tenotomies upon both of the interni; and proved not only that I had a high degree of "latent" trouble to correct (which a one-degree prism would not have helped), but also that the attacks have thus far been totally arrested by the relief of abnormal eye-tension.

In the third place, it has been proven that systematic daily exercises of the various eye-muscles (accomplished by teaching the patient to fuse images which have been rendered momentarily double by a prism held before the eyes) will in some cases develop latent muscular anomalies of the orbit.

In other words, a patient, after a week's muscular drill, will often show a greater flexibility of the eye-muscles and the existence of a lack of equilibrium in the eye-movements, which they did not exhibit at the earlier examinations. I am aware that an injudicious use of such prismatic tests in the hands of a novice might cause "asthenopia," and seriously affect muscular conditions; but this fact can hardly be used, I think, by fair-minded critics, to explain the phenomena alluded to here.

Finally, it may be stated, in this connection, that one examination of the various eye-movements is not, as a rule, sufficient for a positive diagnosis respecting muscular anomalies. Repeated tests have often to be made before a complicated problem may be satisfactorily solved, even by an expert in this line of examination.

(7) The view is held that *prismatic glasses are not only inadequate as satisfactory remedial agents in most cases, but that they may be positively injurious to certain classes of patients.*

Few, if any, of our prominent oculists have perhaps ordered as many prismatic glasses as has the chief advocate of the method now under discussion. Yet, in spite of this fact, strict limitations upon their field of usefulness (not generally taught) seem to be rendered probable by late investigations.

A careful study of the different movements of the eyeball, and of the combination of muscles required to produce some of them, must impress even the most casual reader with the idea that an agent (such, for example, as a strong prism) which tends to restrict the movements of any one muscle, may do harm if persistently worn.

Some patients are peculiarly susceptible to such influences. I have encountered a large number of patients whose eyes refused to tolerate a prismatic glass. Their symptoms were at once made worse whenever they attempted to correct an existing muscular anomaly by wearing a prismatic glass.

On the other hand, many patients are benefited at once by the use of prisms, and suffer no inconvenience of any kind from them.

What are we to infer from this statement? Are we to surmise that the prisms were either injudiciously selected or improperly placed, simply because the patient could not tolerate them? I think not. Such might possibly be the case in the hands of a novice, but presumably it is not the case in the experience of one skilled in eye-examinations.

My own experience in several such instances has shown me that a properly graduated tenotomy of the muscle exhibiting the greatest tension has been followed by a complete cessation of the nervous symptoms for which the patient sought relief, in spite of the fact that prisms prescribed to correct the same error have proved intolerable to the patient, and have markedly aggravated the symptoms.

There is, however, a practical and important field for prismatic glasses. It is well to keep, as a part of a physician's office equipment, a large number of prisms of different angles. These can be slipped into a frame with the base inward, outward, upward or downward, as the exigencies of any case seem to demand. They may be loaned from time to time to patients, for the purpose either of verifying a diagnosis or, by giving relief to a "manifest" ocular tension, of developing a latent muscular error which the physician may be led (by repeated examinations of the patient) to suspect. When they are well-tolerated, the physician may often learn a great deal by their protracted influence. When they are not well borne, it is advisable, as a rule, to discontinue their use at once.

It is often wise to prescribe prismatic glass, also, for a class of patients who are unable (for one reason or another) to submit at the time to a graduated tenotomy.

Sooner or later, I find that such patients usually return. As a rule, they do so for one of the following reasons: (1) because they have developed an additional "latent" muscular error, which the prisms naturally failed to correct; (2) because they do not tolerate them well, and are made decidedly worse by their use; (3) because they prefer a tenotomy to the inconvenience of a glass which has to be constantly worn; and (4) because they suffer from eye-fatigue, on account of the disturbance to coördinate movements of the eyeball.

There is no doubt that very many cases of nervous diseases are materially helped (if not radically cured) by the aid of prismatic glasses; but the question naturally arises to my mind in this connection, "Would they not have been more rapidly benefited and permanently relieved with far less inconvenience to the patient by tenotomy?"

(8) The view is held *that a graduated tenotomy is the only way of satisfactorily and permanently relieving abnormal tension of a muscle in the orbit.*

There are only two ways of overcoming an abnormal tendency of the visual axes to deviate from parallelism whenever the eyes are directed upon an object more than twenty feet off. One of these is by the aid of a prism; the other is by a graduated tenotomy of the muscle, which directly aids in producing and perpetuating the deviating tendency.

Whenever prisms are prescribed, they afford relief practically in the same way as a "rubber muscle" does in orthopædic surgery; in other words, they compel the muscle which is opposed to the base of the prism worn by the patient not only to overcome the antagonistic muscle, but also to so adjust the eye as to compensate for the refractive effect of the prism. They practically act, therefore, as a "pulley-weight"—a mechanical device seen in all gymnasiums.

Now, if the wearing of prisms had no deleterious action upon those particular muscles, which, in each case, are not at all at fault, and if they invariably exerted only beneficial effects, this principle of treatment could be more generally applied with benefit. Even then, the existence of latent insufficiency might, unfortunately, remain unrecognized for a greater or less period of time, possibly to the serious detriment of the patient. On the other hand, if it be satisfactorily demonstrated that the operation termed "graduated tenotomy" has been rendered a safe and accurate method of correcting muscular anomalies in the orbit, a fact has certainly been noted that opens a new and shorter route to relief. Such a step enables us, moreover, to decide the question of "latent" muscular defects in any given case.

(9) The view is held that the *difficulties previously experienced in attempting to correct so-called "muscular insufficiencies" in the orbit by a surgical procedure upon the stronger muscles have now been satisfactorily overcome.*

Space will not permit of a discussion here of the demerits of operations previously devised for this purpose. Suffice it to say that the operation first suggested and performed by my friend, Dr. G. T. Stevens, preserves the normal line of traction of the muscle.

This is a point of vital importance to the patient, and one which cannot be claimed, in my opinion, for any other operation previously devised for this purpose with which I am familiar.

Any disturbance in the proper adjustment of the eye-muscles, which must ensue from an alteration in the line of traction of any one or more of the six muscles which move the eye, cannot help but be a serious matter.

The full details of the operation alluded to have already been published.* I quote from an article lately read before the Neurological Society of New York by the chief advocate of this method:—

"In the main it consists of making a small opening through the conjunctiva, exactly over the insertion of the tendon, when the tendon is seized by extremely fine forceps, and divided in each direction, preserving the extreme outer fibres, or, at least, the reflection of the capsule of Tenon, which serves as an auxiliary attachment."

It may be stated in this connection that this operation is absolutely painless when done under the influence of cocaine; that stitches are never employed; that no subsequent dressings are rendered necessary; and that patients frequently go from the operating chair directly to their business. A slight amount of redness and irritation about the wound, and occasionally some sub-conjunctival hemorrhage (both of which tend to rapidly disappear) are all the inconveniences which this operation commonly entails. I have personally performed this operation about six hundred times up to the present date, and I have never known suppuration to occur, or any complications to be induced which caused me serious perplexity.

(10) The view is held that "*eye-strain*" *from any cause* (be it refractive or muscular) *is a serious matter, and that its tendency is to predispose to nervous derangements and to perpetuate them when once developed so long as this factor exists.*

This is one of the most important, if not the chief claim made. It is substantiated by many carefully made and collected observations.

It is chiefly in those cases where, in spite of a muscular error, the images of the two eyes can be blended by a great effort that the patient

* *Archives of Ophthalmology*, June, 1887.

begins to experience the deleterious physical influences of abnormal muscular tension in the orbit. Placing a plain red glass before one eye of a patient suspected of having a slight degree of strabismus will often reveal to a patient a diplopia of which he or she may have been unconscious. Such cases do not belong to the class discussed here as those of "insufficiency of the ocular muscles."

It is not hard to understand why it is that an animal too heavily laden is unable to rise after a fall has occurred until the load is taken from it.

So it is with many nervous patients. The incessant efforts made to fuse the images perceived by the two eyes into a single image (when a muscular defect renders such an act possible, yet one of extreme difficulty) are liable sooner or later to exhaust the nervous force of the patient and to excite some form of functional nervous disturbance.

This is the line of argument, which apparently seems difficult to understand. It is a train of reasoning which many enthusiastic pathologists naturally prefer to discard, because it puts an end to a search for a pathognomonic lesion which no human eye (even with the aid of a microscope) has ever yet been able to detect in many hopeless and chronic cases of chorea, epilepsy, insanity, neuralgia, headache, hysteria, and neurasthenia. It is a view which will probably be opposed by some, because it comes into direct antagonism with the prolonged administration of the various bromide salts; in spite of the fact that the injurious effects of such administration are too frequently encountered to be ignored. It is a principle relating to functional neuroses which is naturally combated on general grounds, because it is new, and opposed to preëxisting views.

Respecting the views here advanced, I take the liberty of quoting a few selected paragraphs from a singularly lucid paper lately read by the pioneer in this field before the Neurological Society of New York.* I do so because they appear even yet to be misunderstood by some who listened to the paper quoted from. The author of that paper says:—

"A doctrine so much at variance with ordinary beliefs must of necessity excite suspicion that the proposition has been based upon insufficient data, or that observations have been imperfectly made. That neither of these suspicions is correct it is hoped may be shown to the satisfaction of reasonable inquirers. If the proposition appears extreme, and tending at best to the recognition of a single class of causes to the exclusion of others, let me recall the fact that the proposition fully recognizes any and all causes of nervous irritation, and that the influences indicated are held to be preëminent, but not exclusive permanent causes. If greater importance is conceded to the influences mentioned in the proposition than to others, it is from no unmindfulness of the

* *New York Medical Journal*, April 16, 1887.

possibility of other conditions acting as irritating influences, or that certain known or unknown influences may give character to the results of irritation arising from the causes mentioned. Let it be remembered that it has been universally conceded that the nature of the neuropathic tendency is unknown. If one preëminently important element is demonstrated, it is not to be rejected because it may not include the whole.

"In the explanation of the etiology and treatment of disease, neither settled theories nor novel doctrines are to be accepted only as they are confirmed by undoubted facts. Nor can isolated facts, nor facts divested of their natural environments, be accepted as valid evidence in support of theories, old or new. The facts must be uniform, occurring so regularly as sequences as to demonstrate that they are consequences. Unless the skilled observer is able to predict, with a reasonable degree of accuracy, the result of certain combinations of circumstances, such result, when occurring, must be considered accidental."

"The principle of ocular irritation is of wide application, and is not to be compared with the occasional irritation set up by such accidental and usually secondary causes as phimosis is, the presence of calculus, the existence of a stricture of a passage, the effects of decayed teeth, and of many other peripheral irritations which might be mentioned. All these are of importance, and are not to be overlooked.

"The conditions to which I have especially called attention are, however, in general, commensurate with the life of the patient, and exist in a vastly greater number of instances than either or all of the conditions belonging to the other class just mentioned. Not only are those painful or irregular conditions, usually described as neuroses, in great proportion responsive to the relief from ocular tensions; but a great variety of conditions, commonly regarded as local affections, yield as readily, and prove that they are in fact reflex phenomena.

"If it be said that the origin and prevention of nervous diseases is to be found in a great variety of circumstances, I reply, let us find them all, and adapt our measures to them all, but let us not neglect this because there may be others.

"For myself, I do not think that another as important class of causes of nervous disturbance will be found as that which attends the anomalies of the parts engaged in the performance of the visual function. In any case, our aim is to prevent the evils of nervous derangement by the early removal of any known mischievous tendency, and our duty is, when such nervous derangement actually occurs, to remove every perplexing cause. In the observance of such a principle, we may leave to superstition and to ignorance the practice of expelling nervous diseases by means either fashionable or obsolete."

ARE FUNCTIONAL NERVOUS DISEASES FREQUENTLY REFLEX MANIFESTATIONS?

The view that a direct relationship exists in many subjects between epileptic seizures (that are apparently not associated with organic lesions of the brain) and abnormal muscular tension within the orbit seems to have received most valuable indirect confirmation in the startling experiments published by Drs. F. X. Dercum and A. J. Parker, of the University of Pennsylvania.* According to these observers, convulsive seizures were artificially induced in apparently healthy subjects by prolonged muscular tension of a single muscle or groups of muscles in the limbs.

I regard these experiments as perhaps the most important ones that have yet been brought forward in support of the general view that epileptic seizures are, in the large proportion of cases, simply one of the many types of manifestation that a reflex cortical disturbance is capable of exhibiting.

It is unquestionably true also that such reflex causes are too often not sought for by the profession with sufficient care or in the proper way.

The methods of examination that have been generally regarded until of late as conclusive, when defects in adjustment of the eye-muscles have casually been sought for, were certainly most crude and unscientific; and a modification of them appears to be most timely.

I take the liberty of quoting from the published experiments of Drs. Dercum and Parker the following paragraphs:—

"The subject being seated, the tips of the fingers of one or both hands were so placed upon the surface of a table as to give merely a delicate sense of contact, *i.e.*, the fingers were not allowed to rest upon the table, *but were maintained, by a constant muscular effort, barely in contact with it.*† Any other position involving a like effort of constant muscular adjustment was found to be equally efficient. Any one object in the room was now selected, and the mind fixed upon it, or some subject of thought was taken up and unswervingly followed.

"After the lapse of a variable period of time, extending from a few minutes to an hour, and depending upon individual peculiarities to be noted, ... the subject was frequently thrown violently to the ground in a general convulsion, preceded by tremors which rapidly became more violent.

"Seizures equalling in violence a general convulsion were by no means induced in all subjects, and were generally the result of experiments repeated many times during the same evening. In the experimenters the convulsions became so easily induced that it was thought advisable to desist for a long period."

* *Jour. of Nervous and Mental Diseases*, 1884, pp. 579 and 636.
† Italics my own.

Dr. Chas. H. Thomas, of Philadelphia, when speaking of these experiments in a late contribution to this subject,* says :—

"The effort of constant muscular adjustment here spoken of appears not unlike the condition found in the eyes in cases of insufficiency of the ocular muscles; and it seems not unreasonable to infer that if such strain of the muscles of the forearm would produce results of the kind reported by the authors just named, that the strain upon ill-balanced ocular muscles (which *must be continuous during the whole of the time that the eyes are opened*) should be productive of even more serious, and, indeed, permanent results."

Within the past year, an extremely valuable paper respecting one of the much neglected and perhaps not infrequent causes of epilepsy has been also published by Dr. A. P. Brubaker,† of Philadelphia, entitled "Dental Irritation as a Factor in the Causation of Epilepsy." The following extracts from this paper have an important clinical bearing, and possibly shed some light upon the proper treatment of convulsive diseases and other forms of reflex nervous conditions :—

"In all the wide divergence of view as regards the nature of epilepsy there is a general consensus of opinion that its essential feature is of the character of an explosive discharge from the higher nerve-centres, the nerve-force thus liberated bearing down upon the centrifugal distributions of the motor nerve-tracks with such an excess of energy that incoördination of movement reaches the stage of convulsion and spasm. Owing to the periodicity of the convulsive seizures, it has been assumed that in individuals predisposed to epileptic attacks the higher nerve-centres are in a state of high tension, of unstable equilibrium, and that it only requires a stimulus of a definite quantity or intensity to excite the explosive discharge.

"The object of this paper is to direct the attention of physicians to a cause of epilepsy which has not hitherto been estimated at its full value, inasmuch as in none of the standard works upon neurology is the subject even alluded to,—viz., pathological states of the dental structures. That dental inflammations and disorders are more often provocative of epileptic seizures than is commonly supposed appears quite certain from the following cases, and also from the character of the cause and its effect. Many reasons might be given why dental disorders are peculiarly adapted to call forth this periodical discharge, and why these disorders are habitually overlooked by the physician, but they need not be detailed here. As exemplifying these phenomena, some interesting and instructive cases are adduced.

"The interest aroused by the result of the preceding case led to an examination of medical literature for reports of similar cases. I find that no less than sixteen cases, entirely and immediately cured by the removal of an irritating tooth, have been recorded by different observers, and which are here arranged in chronological order. It is not supposed that this collection embraces all the recorded cases, but it is hoped that it will elicit references to many others, and, what is more important, the reporting of many new cases."

* *Trans. Phila. Co. Med. Soc.*, Mar. 14, 1888.

† *Jour. of Nervous and Mental Diseases*, 1888, p. 117.

In the light shed upon this subject chiefly by recent contributions to medical literature,* the view is gradually being accepted by many in the profession that certain nervous diseases (whose pathology, to say the least, is still in doubt) are possibly not dependent in every case upon an unrecognized organic lesion; and they are being led to coincide with the statement that the term "functional" nervous disease may be properly applied, in some instances at least, to the graver nervous conditions,—such, for example, as epilepsy, chorea, hysteria, or other manifestations of nervous exhaustion, and insanity. In other words, the professional mind seems more willing now than in the past to discard an apparently fruitless search for a pathognomonic lesion for each intractable nervous condition, and to look more calmly upon tangible clinical facts, even if they are radically opposed to preëxisting views.

If the view that eye-strain, dental irritation, or other causes of reflex disturbance may be a frequent cause of functional nervous derangements proves to be the correct one, beyond the possibility of doubt or cavil, it is not difficult to see that a hope of marked relief or of ultimate recovery is practically extended to many hopeless sufferers upon whom drugs have exerted little or no benefit.

In order that those of my readers who have possibly not given much attention to the views which most of my incorporated cases are particularly selected to illustrate may properly understand the train of reasoning that offered a solution to my mind of the symptoms here recorded, I take the liberty of quoting a few paragraphs from a paper which I lately read before the International Medical Congress at Washington, entitled "Does a Relationship Exist between Anomalies of the Visual Apparatus and the So-called 'Neuropathic' Predisposition?"† This paper was based upon a carefully tabulated analysis of the records

*The reader is referred to the articles by Dr George T. Stevens on "Chorea" (*Medical Record*, 1876); on "Anomalies of the Ocular Muscles" (*Arch. of Ophthalmology*, June, 1877); and on "Ocular Irritations and Nervous Diseases" (*New York Medical Journal*, April, 1877); also to his work on "Functional Nervous Diseases" (D. Appleton & Co., N. Y., 1887); also to a contribution by Dr. H. D. Noyes, on "Tests for Muscular Asthenopia and Insufficiency of the External Recti," read by him before the International Medical Congress, Copenhagen, 1884; also to papers by the author on "The Eye as a Factor in the Causation of Some Common Nervous Symptoms (*New York Medical Journal*, February 27 and March 15, 1886); on "Eye-strain in Neurology" (*New York Medical Journal*, April 16, 1887); on "Eye-strain in its Relations to Functional Nervous Diseases" (*Medical Bulletin*, September, 1887); and an abstract of an essay read before the International Medical Congress at Washington, entitled "Does a Relationship Exist Between Anomalies of the Visual Apparatus and the So-called 'Neuropathic Predisposition?'" (*Medical Register*, November 19, 1887). The articles by Drs. Dercum and Parker, Dr. C. H. Thomas, and Dr. A. P. Brubaker, of Philadelphia (already quoted), are worthy of special notice in this connection.

†An abstract of this paper was published in the *Medical Register*, November 19, 1887.

of one hundred consecutive cases of typical neuroses taken from my private case-book.

In this paper I say :—

Until there is a uniformity in the methods employed for testing the eye-muscles,* and of terms for the recording of anomalies so detected, the profession must unfortunately continue to be more or less embarrassed in this line of research. I do not feel justified in personally discussing this subject here, as it has only an indirect relationship with this paper; but I can not refrain from saying, in this connection, that to defective methods of examination, made venerable chiefly by their antiquity, we owe to-day, in my opinion, much of our ignorance of anomalies of the ocular muscles.

Some time ago I was struck, on looking over a children's magazine, with an illustration designed to teach the reader the dependence of the various organs of the body upon the brain. It represented the brain as the head of a manufacturing establishment sitting at his desk, and around him were the various departments,—as, for example, the liver-department, the stomach-department, the eye-department, etc. These departments were connected with the head of the establishment (the brain) by telegraph-wires, through which each could make its wants known and receive information regarding them.

Probably the designer of this sketch (made for the purpose of illustrating to the child the dependence of the organs upon the brain for their successful operation, as well as their actual support) built "better than he knew." He embodied in his drawing a graphic representation of certain fundamental principles of physiology which are not clearly understood even by many adult minds in their bearings upon the general health.

The lungs do not make us breathe; except in an indirect way, by asking the brain to start the necessary muscles into action. The stomach does not perform its functions until after the brain has been requested by it to turn on the blood-supply in sufficient quantities to produce the requisite quantity of gastric juice. The intestine performs its incessant worm-like movements by no inherent power of its own. The heart keeps up its rhythmical beating only when permitted to do so by the great centre of nerve-force.

Now, is it at all inconsistent with physiological principles to advance the view that any excess of nervous expenditure to one organ over the normal amount which should be furnished is done at the expense of the others sooner or later?

No one can draw incessantly upon his reserve capital of nerve-force without incurring a risk of ultimately exhausting it. A *bankruptcy in the reserve capital of nerve-force* entails untold ills to the individual.

The day of reckoning is postponed in any given case in direct proportion to the drafts made upon the reserve and the amount of the reserve. This may help us to explain why some escape it indefinitely, while others are precipitated into indescribable distress when life is hardly begun.

In case the bearing of eye-strain upon the problem of nervous expenditure is not very clear to some of my readers, I deem it wise to quote here some extracts from a late brochure of mine upon this subject (*N. Y. Medical Journal*, Feb. 27 and March 13, 1886).

* See article by Dr. G. T. Stevens in the *Archives of Ophthalmology*, 1887 and 1888.

Speaking of hyperopia, I say:—

Fortunately for our nervous system, the normal eye takes pictures of surrounding objects *without any muscular effort* when the object is more than twenty feet away; hence, during the larger part of each day the *normal eye is passive*, and is practically at rest, although performing its functions. How different is the condition of the far-sighted or "hyperopic" eye, however, from the normal! For this eye (since it is *too short* in its antero-posterior axis) all objects *have to be focused by muscular effort*, irrespective of their distance from the eye. Such an eye is never passive. It has no rest while the body is awake. It is always straining more or less intensely to bring properly upon the retina the images of objects seen.

The "hyperopic" condition of the eye, or "far-sightedness," as it is called, is a very common defect. It is especially frequent in persons of tubercular parentage. It is well, therefore, to suspect the existence of this defect in children or adults whose ancestors have died of "consumption."

Again, speaking of muscular anomalies, I use the following illustration:—

A high-couraged horse feels the will, as well as the support, of his driver through the reins by means of the bit. Although his course and rate of speed are changed from time to time at the will of the driver, the reins are never slackened. The horse becomes acquainted with the desires of his master by a sense of increased or diminished tension upon the reins. He is guided to either side by a difference in the tension of the two, although the driver does not entirely relax his hold upon the opposing rein while he uses the guiding one, and the difference in tension may be very slight.

So it is with the normal eye. It is both controlled and supported while performing its movements within the orbit by the eye-muscles (which are its reins). The brain is the driver. At its command the eye revolves, or remains stationary at any desired point. The tension of muscles, opposed to any movement of the eye required, is so modified by the brain as to insure the requisite support to the eyeball, and to steady it as it moves. Thus, a perfect equipoise is constantly established between opposing forces, adjusted with the nicest care to meet the full requirements of the organ under all possible circumstances. The normal eye does not tremble or wabble when it moves or the attempt is made to hold it in any fixed attitude. It is a piece of machinery, perfect in all its parts, reliable in its movements, perfectly controlled by its master.

The eye with "muscular insufficiency" is like a horse with an inexperienced and incompetent driver: the proper tension upon the reins is not maintained at all times, as it should be; there is no equilibrium between antagonistic muscles; fixed attitudes are maintained with difficulty for any length of time; the brain becomes more or less disturbed by its inability to properly control the eye-movements, and exhausted by the continual strain imposed upon it by the efforts required to do so even imperfectly.

A point may now be raised concerning which some misapprehension seems to exist among medical men (judging from remarks which I occasionally hear expressed). I refer to the relationship of actual squint to nervous disturbances.

No one can deny that people frequently live for long periods of time in houses impregnated with sewer-gas and in the most malarious regions without apparently suffering in consequence. Yet no intelligent

man would attempt to prove to-day that sewer-gas poisoning and malarial infection were delusions simply because some people had escaped their influence.

The argument has been advanced that, because some cross-eyed people have escaped epilepsy, chorea, insanity, and functional neuroses of the milder types, it is erroneous to maintain that eye-strain has anything to do with these conditions. *This is absurd upon its face.* The hint might, perhaps, be pertinently dropped in this connection that cross-eyed people practically suffer but little from their muscular error, simply because they have *habitual double vision, which no effort on their part can correct.* These subjects learn very quickly to practically discard one image (the one seen by the crossed-eye) and to use one eye only for ordinary vision. In other words, they never try to blend the images of the two eyes, except in certain attitudes of the head, which result in a single visual image without an effort on the part of the patient.

It is only in those cases where (in spite of a muscular error) the images of the two eyes can be blended by a great effort that the patient begins to experience the delcterious physical influences of abnormal muscular tension in the orbit.

If we admit the proposition that eye-defects, or anomalies of the ocular muscles, are liable to become causes of impaired nervous energy (because they demand an excess of nervous expenditure), we are forced to the conclusion that the earlier this source of physical depression is removed the better are the prospects of the person so relieved of escaping diseases which impaired nervous energy necessarily tends to hasten or develop. We are naturally led to question if the so-called "neuropathic predisposition" is not dependent (in a certain proportion of cases, to say the least) upon "eye-strain." We might possibly also be led to think that the so-called "tubercular tendency" (which is present, as far as my observation goes, in nearly 50 per cent. of all cases of marked functional nervous disease) might, in some cases, be modified, controlled, or perhaps arrested before its physical results become apparent by taking from the life of such subjects a load which their small reserve capital of nervous energy particularly unfits them to endure.

It is hard to give up the view, so universally conceded, that a predisposition to disease means a "constitutional taint." Yet, in many cases, we are absolutely unable to demonstrate that any evidence of physical weakness or disease has appeared until sufficient time had elapsed from the date of birth for the development of a serious impairment of nervous energy. What has caused it? Has it been deficient nourishment, a lack of maternal care or solicitude during childhood,

gross violations of the rules of hygiene, or a lack of prudence on the part of the individual when of matured experience? The history of case after case answers "no" to such surmises. These, then, are not the all-important factors in every case. Phthisis, epilepsy, chorea, headaches, neuralgias, hysteria, dyspepsia, obstinate constipation, nervous prostration, inebriety, and many other evidences of the neurasthenic state are markedly hereditary. What is the load (if any) which many sufferers of this type are carrying through life? *Have they a congenital burden*—which is, perhaps, too often unrecognized? I leave these questions for future research to solve.

In this section I will call attention to a few cases selected from my own case-book where the relief of ocular defects produced remarkable and unexpected benefit after all hope of recovery had practically been abandoned by the patient.

I bring these cases prominently forward in the interest of science only; because the improvement made by these patients is attributable not to drugs, but solely to Nature, when a burden of which she could not rid herself was taken away and recuperation became possible.

Did you ever see a tired horse fall prostrate under an excessive burden? *How long would he remain so, were the burden not removed?*

Now, it should constantly be borne in mind that no two cases exhibit identical manifestations of nervous depression or irritation. Some patients who are suffering from such conditions manifest the effects in physical, others in mental disturbances. The heart's action may be alone disturbed in some cases, the stomach may give out in others, some may complain alone of spasmodic muscular troubles, some may notice its effects in the eyes, some are rendered sleepless, many suffer from more or less persistent pains, a few complain alone of skin disturbances, and so on throughout the different parts of the entire human organism.

We can understand how these apparently discordant facts may be reconciled when we recall the fact that by means of the brain and spinal marrow, and the nerves which unite these centres to the different parts of the body, we are enabled to see, hear, taste, smell, appreciate touch, swallow, breathe, and perform voluntary muscular acts. It is by means of our nerves alone that the heart beats; the digestive processes go on without our knowledge or control through the same agencies; the blood-vessels contract and dilate in accordance with the demands for blood telegraphed to the nerve-centre by different organs and tissues; and every process pertaining to life is thus automatically regulated.

It requires no medical knowledge to see at once how a disturbance of so complicated an electric mechanism as the nerve-fibres and the nerve-cells of a living animal are can upset all or any one of the

individual functions enumerated. Many of our houses are furnished to-day with electric bells by means of wires distributed in the walls. In some houses we light the gas-jets, and even the rooms themselves, by means of the same subtle fluid. When the battery becomes weak, or when the wires are disarranged or broken, what may be the results? Some of the bells may cease to ring when the button is touched, while others work properly. Perhaps the electric light may fail in some rooms and burn with its accustomed brilliancy in others. The gas-jets may not be properly ignited. So it is with the nervous apparatus of man. From the same cause one patient may have nervous dyspepsia, another sleeplessness, a third headache or neuralgia, a fourth weakness of the muscles, a fifth disturbances of sensation, a sixth hysteria, chorea or epilepsy. It is needless to multiply illustrations.

The nervous system of man has been very aptly compared to a mountainous region where any atmospheric disturbance calls forth a "series of echoes" at distant points. So it is with many of the so-called "functional diseases." They may be simply the manifestations of a disturbance of the nervous system, entailed by causes which have been overlooked or imperfectly relieved.

Before I leave this subject it is but proper to say that a few cases reported by me in this chapter (while not a large number in the aggregate) were, without exception, well-marked cases of typical and intractable neuroses. The improvement noted in each case after well-directed treatment of the eyes or the eye-muscles tends to cast a doubt upon the existence of any organic disease. No other causes of reflex nervous disturbances outside of the eyes were detected after a careful search in any of these cases; otherwise it would have been my manifest duty to *relieve all that were found* in my efforts to benefit the symptoms manifested by each patient.

It is not to be expected, nor do I anticipate, that views so radically opposed to the ordinary methods of treatment by medication, now generally advocated for functional nervous diseases, will be accepted at once by the profession at large, even if correct and satisfactorily demonstrated. No great advance in science has ever been made until time has tempered prejudice and modified the prevailing tendencies of thought.

Of late years we, as a profession, have had our attention drawn, however, more seriously than ever before to the clinical importance and the necessity of detection of remote sources of irritation to the nervous centres. We have already learned that the ovaries, the womb, the prepuce, the urethra, the rectum, the alimentary canal, etc., can, in some instances, induce serious nervous conditions which closely simulate the evidences of organic disease. Complete paralysis of both legs has been known to be cured in a child by circumcision. The operation devised

by Battey for the removal of the ovaries in subjects attacked with hystero-epilepsy is to-day sustained by the profession, and often performed with the view of removing a merely suppositious source of reflex disturbance. This supposition, in many cases, is based, unfortunately, upon tests much less scientific and therefore less reliable than the tests employed to detect anomalies of the visual apparatus. It is safe to question, therefore, if the source of reflex irritation in many patients of this class has been carefully sought for, and if it does not lie more in the eyes than in healthy ovaries,* which are not infrequently sacrificed. Personally, I should not feel justified in taking so serious a step with any patient until every other possible cause of reflex disturbance had been carefully sought for in vain.

There is no doubt that many physicians of prominence are devoting more attention to-day in their practical office work to the determination of latent refractive errors in the eye and disturbances of equilibrium in the eye-muscles than was their habit in years past.

This long-neglected but important element in the "neuropathic tendency" (and perhaps also in the "tubercular predisposition") is now receiving from many sides the most thoughtful consideration. Sooner or later, in my opinion, our views of the causes of functional nervous disease will no longer be those now advanced in most of the works devoted to that field. We shall in time more clearly recognize the fact that drugs do more harm in functional neuroses than good whenever any exciting cause of such a morbid condition persists and can be removed; just as we to-day rely, in case of a joint-disease, more upon mechanical separation of the surfaces of the inflamed joint than upon anodynes to relieve the pain. We shall learn to search more carefully and intelligently for obscure causes of reflex disturbances, and to try the effect of their removal before we resort to drugs. Medication must eventually, in my opinion, become the *dernier ressort* of the physician, in this particular class of nervous diseases, rather than the haven of refuge.

We are now prepared to discuss some of the various forms of functional nervous disturbances commonly encountered in medical practice.

The term "*disease*" can hardly be applied to a condition whose morbid anatomy is unknown (as is the case with epilepsy, chorea, hysteria, hystero-epilepsy, migraine, and certain forms of peripheral paralysis, and neuralgia). These abnormal states are, properly speaking, but *symptoms*, the exciting cause of which may often be involved in obscurity, and the removal of which must, of necessity, form a very important factor in the treatment.

*Cysts in the ovary are seldom, if ever, wanting; hence, they can scarcely be pronounced (when small) an evidence of disease.

EPILEPSY.

Of all the so-called "functional" nervous diseases, this condition merits attention first, because it is the most grave. It consists of periodical convulsive attacks, associated, in typical cases, with a loss of consciousness.

The paroxysms may vary in regard both to their frequency and severity.

The extent of the coma, the duration of the fit, the parts convulsed, the mental aberration, and the constitutional effects which follow the attack vary also in different subjects. Among the ancients, this condition was regarded with peculiar horror, and was attributed to the "possession of a devil."

Varieties.—Custom has established a classification of this condition into types, as follows:—

(1) The "grand mal" or *typical attack*, where consciousness is totally absent during the convulsive stage.

(2) The "petit mal" or *mild attack*, in which consciousness may be wholly or partially retained, and the convulsive movements may be slight or absent.

(3) To these, may be added a condition known as "irregular epilepsy."

These distinctions are not clinically accurate. Cases do occasionally occur where consciousness may be wholly lost, and yet the convulsive movements may be partial rather than general. Again, general convulsions of a severe type have been observed in rare cases where consciousness has been perfectly retained. I have encountered, moreover, a few cases of *petit mal* where certain peculiar attitudes of the extremities and face have taken the place of convulsive movements and consciousness has been only imperfectly lost.

I have frequently seen patients have an epileptic attack in my office while sitting upon a chair without falling from it. One patient now under my charge has had numerous attacks while walking in the street, which, as his attendant assures me, did not demand any aid on his part toward supporting the patient.

Etiology.—Heredity plays an important part in the history of many cases. A record of epilepsy, hysteria, insanity, chorea, migraine, or phthisis is commonly found to exist in some branches of the ancestral line. Inebriety may also have been frequently observed on the father's or mother's side.

Some adults are apt to trace their first fit to masturbation; or to excessive venery, some great mental excitement or strain, or some infectious disease (prominently syphilitic infection).

Most epileptics also give a history of *some severe injuries* received, to which they attribute (too often erroneously) their convulsive attacks. Investigation will usually show that these injuries are apt, however, to be the result of an attack rather than cause, or to be a mere coincidence. Traumatism may, unquestionably, in rare cases, prove an exciting cause of epilepsy, but I believe the frequency of such cases to be greatly overestimated. The same remark would apply to some organic brain-lesion.

In the third place, any condition which creates *marked reflex irritation*—such, for example, as eye-defect, dental irritation, phimosis, gastric or intestinal disturbances, cicatrices involving nerve-filaments, foreign bodies or wax in the ear, ovarian irritation, uterine or rectal diseases, the first menstruation or coitus, etc.—may, in some subjects, induce epileptic seizures. A large proportion of the epileptic attacks of infancy are unquestionably brought about by trivial reflex causes.* In adults, however, the reflex causes enumerated (with the exception of "eye-strain" and bad teeth) are, in my opinion, less apt to induce true epilepsy than the profession generally suppose.

It is very uncommon, in my experience, to meet with male or female adults (in whom attacks of epilepsy have commenced after the seventh year) which fail to show some abnormality of refraction, or in whom the condition known as "ocular insufficiency" is not found to exist to a marked degree.

I have one patient under my care at present who, for years, had always been seized with an attack on suddenly going from the light into darkness. Another (who, happily, has apparently about recovered after partial tenotomies performed upon the eye-muscles) had the first two attacks when exposed to the dazzling reflection of the bright sun upon a rough sheet of water, and was rescued from drowning twice in consequence. A third patient of mine, who had several attacks daily when on land, would, strange as it seems, skip many weeks without a fit when on shipboard, where his efforts to accommodate vision for near objects were infrequent. He had very serious refractive and muscular anomalies.

Finally, it must be conceded that *organic changes within the brain-substance* or *irritative lesions of the cortex* may, in a certain proportion of cases, induce epileptic attacks. A depressed fracture of the cranium or an exostosis, for example; a meningeal thickening; an abscess within the skull; parasitic deposits; gummata, etc., have been shown to have induced attacks of this character.

We may assume in cases of epilepsy that, as a rule, the condition becomes more grave after the seventh year than in early childhood, —especially if no well-ascertained cause of reflex irritation can be

* Worms, indigestion, phimosis, teething, etc., are often accompanied by epileptic attacks in infancy or early childhood.

discovered. It is impossible as yet to express any definite knowledge regarding the relative frequency of such causes to the total number of epileptics encountered,—because the published records of such cases are singularly imperfect in reference to the examination of the eye. The following statistics (taken from Dr. Stevens' prize essay) will, however, prove of interest in this connection :—

Total number of cases reported, ... 140 } These include all cases up to
Total taken from the author's private practice, . 85 } 1882.

An analysis of 100 consecutive cases of typical epilepsy, from number above mentioned, shows the following refractive errors to have existed :—

Hyperopia or Hyperopic Astigmatism, . . .	present in	59	cases, or	59 per cent.
Myopia or Myopic Astigmatism, ...	"	23	"	23 "
Refractive errors of less than 1 dioptre, . . .	"	18	"	18 "
	Total,	100		

The *condition of the eye-muscles* was not sufficiently well reported in many of these cases to allow of percentages being given in reference to this important factor.

My own examinations up to 1887, of a much smaller number, show that my private cases possess a much larger percentage of hyperopic abnormalities (in excess of 1 dioptre), and a smaller proportion of myopic defects. In fourteen cases carefully observed, prior to 1887, nine were markedly hyperopic, four were myopic in excess of one dioptre, and only one was emmetropic. In every case but one, a marked condition of *esophoria* was found. In five, *hyperphoria* existed in addition to esophoria. In the remaining case, a hyperphoria of about $\frac{1}{2}$ degree was all that was detected. The following summary of these cases may shed possibly some additional light upon this new field of inquiry :—

Total cases, 14. (All of the severe type.)

FREQUENCY OF THE ATTACKS (IN SPITE OF ACTION OF BROMIDES) WHEN FIRST SEEN.	EXAMINATION OF THE EYES.		MENTAL STATE OF PATIENT.	REMARKS.
	Refractive Error.†	*Muscular Error.*		
Average of over five fits each day, 1 case.	H., 7 cases.	*Esophoria*, 13 cases.	Markedly affected in 5 cases.	One of these cases had 52 convulsions in 8 hours, after stopping the use of the bromides,—each fit lasting 3 minutes.
Average of three per day, 4 cases	Ha., 4 cases.	*Hyperphoria*, 5 cases.	Slightly affected in 3 cases.	
Average of two per day, 2 cases.	M., 4 cases.	*Exophoria*, 0 case.	Unaffected in 6 cases.	
Average of one per week. 2 cases.	Ma., 1 case.	*Heterophoria*, 14 cases.		In all of these cases the frequency of the fits was greatly increased when I discontinued the use of the bromides.
Average of one per month, 3 cases.	Em., 1 case.	*Orthophoria*, 0 case.		
Occasional attacks, 2 cases.				
Total cases=14	Total=14 *	*	Total=14	

*Some of these patients showed a more than one eye-defect; hence, these two columns cannot be tallied.

†The symbols used in this table are explained on p. 130.

Morbid Anatomy.—In spite of the fact that much has been written upon this subject, no primary changes of an anatomical character can be asserted to be pathognomonic of epilepsy.

In this view, I think that most of the later authorities stand agreed.

The view of Schroeder Van der Kolk (1859) that the vessels of the posterior half of the medulla (those affecting chiefly the roots of the vagus and hypoglossal nerves and the fourth ventricle) were unnaturally dilated is not now accepted. That of Kroon (asymmetry of the olivary bodies); of Solbrig (constriction of the spinal canal, with secondary atrophy of the medulla); of Lélut (sclerosis of Ammon's horn); of Cooper (compression of the carotid arteries); of Landois (venous hyperæmia of the brain and spinal cord); of Nothnagel (irritation of the "convulsive centre" in the region of the medulla and pons); and many other equally fanciful hypotheses have been proven to be more or less fallacious.

The exhaustive paper of Hughlings-Jackson (1873), in which he advocates the theory of "explosive discharges" of the cells of the brain in epileptic attacks, is rather physiological than pathological in its scope. If, as he believes, a mal-nutrition of the brain-cells exists as a primary state (which predisposes to these paroxysmal explosions) the importance of the detection and removal of its cause becomes the more apparent. The paper referred to aids us more in localizing an intra-cranial lesion of an irritative type than in explaining the occurrence of genuine epilepsy as we commonly encounter it.

In the light of later researches, it is, in the opinion of the author, not necessary to discuss at length the view of Lasegue (1877) that epilepsy proceeds from cranial asymmetry or malformation.

Brown-Séquard has shown that animals can be made epileptic by injuries to the spinal cord and peripheral nerves; but, as some weeks are required to induce this result, these experiments seem to show that some secondary changes in the nerve-tissues had probably developed as a result of the injury inflicted.

Eccheverria claims to have discovered serious changes in the cervical sympathetic ganglia and the sympathetic fibres in connection with epilepsy. Similar changes have been observed, however, by Mayer and others in connection with diseases of the brain associated with vascular disturbances.

Symptoms.—The general character of a severe epileptic attack has been partially described already in a previous section (page 155). It will simplify description to consider certain features of the attack separately.

GRAND MAL.

PREMONITORY SYMPTOMS.—Most epileptics affected with "grand mal" have some peculiar sensations (the *auræ*) which indicate the approach of an attack. Some subjects experience a marked change in temperament for a day or two prior to the fit. They become either gloomy, sullen, inclined to anger, or, in exceptional instances, more cheerful than usual. Again, the skin of the face and neck may assume a a dusky hue some hours before the attack. In others, headache, cardiac palpitation, pain in the præcordium, a sense of distension of the abdomen, a diarrhœa, attacks of vomiting, sensitive spots on the limbs, attacks of agraphia, giddiness, unnatural loquacity, etc., have been reported as forerunners of an epileptic fit.

Auræ connected with the special senses are not uncommon. Some patients perceive an unnatural color (usually red or green or blue) in the field of their vision. Some patients of this class habitually see objects enlarged or diminished in size,—an evidence of eye-defect. Others see a rising mist or various unnatural objects (as in a dream). To smell bad odors, to hear strange or unnatural noises, to taste unexpectedly obnoxious things, or to feel numbness, etc., are not infrequently a note of warning to epileptics to seek a recumbent posture.

The *muscular auræ* observed comprise an unexpected and uncontrollable spasmodic movement in some part of the limbs or body, a sudden activity in the facial muscles, a sudden tendency to rotate the body, or to break into a run while walking, and a sudden stiffness or immobility of a part.

Vaso-motor auræ are not uncommonly observed. They comprise spots of pallor or flushing upon different portions of the skin, an unnatural heat or coldness in some part, a tendency to sudden local perspiration, and flashes of heat or of cold shooting over the whole body.

Psychical auræ are occasionally exhibited in the form of delusions, hallucinations, and illusions.

The Epileptic Cry.—In a certain proportion of cases, a peculiar cry precedes the fit. It may be a shriek, or again it may be scarcely audible. I have known it to awaken all the patients in a hospital ward from deep slumber. The patients are usually unconscious of having uttered it, and it is probably due to a very forcible expiration through a partially closed and rigid glottis.

Indications of Alarm.—Some epileptics have a facial expression of great alarm just preceding the fit. This is more common in children than in adults. In rare instances, the attitude of the hands and arms express the same mental state.

Actual Symptoms.—At the approach of the fit, it is common to observe a marked *facial pallor*. Even in "petit mal" this is generally present. In exceptional cases the face may appear congested.

The Stage of Rigidity.—If the fit is a severe one, the body becomes at first as rigid as a board. The limbs are extended, the feet inverted, the fingers and toes are commonly flexed, and the head is thrown back and usually turned to one side. The whole body may be twisted backward or laterally. The eyes are fixed during the tonic stage, and the pupils are, as a rule, widely dilated. The breathing is partially or totally arrested. Sensibility is abolished.

The Stage of Clonic Spasms.—After the tonic stage has lasted for a short time (about two minutes or less), convulsive movements begin, the whole body being alternately thrown into violent contraction and relaxation. The face is distorted by terrible grimaces and assumes a purplish hue. The breathing gradually becomes loudly stertorous; the saliva escapes from the mouth as froth, and is often bloody from wounds inflicted by the teeth upon the tongue. The urine, semen, and fæces are often passed involuntarily. The clonic spasms generally subside by degrees, growing less and less violent until they entirely cease. As the convulsions subside the face becomes less dusky. During this stage the eyes may stand open and seem to protrude from the socket; but they are generally turned upward, so that the white of the eye only shows beneath the blinking eyelids. They sometimes become intensely congested, so as to give the eye a resemblance to raw beef.*

The pulse cannot be satisfactorily observed in this stage on account of the muscular movements.

Stage of Recovery.—When the convulsive movements have entirely ceased, and the breathing has resumed its normal character, the patient lies limp and helpless for a short time and gives evidence of a desire to sleep. If aroused, he acts as if dazed and stupid; answers questions with reluctance or imperfectly; looks about him in a semi-conscious or frightened way; mutters to himself some unintelligible sentences; and relapses into a lethargic sleep. Cases where complete epileptic coma has lasted forty-eight hours have been reported. As a rule, however, a sleep of a few minutes suffices to enable the patient to walk with slight assistance.

The fit usually leaves a sensation of dull headache for several hours and great muscular fatigue.

*Serious difficulties may occasionally arise from this intense congestion after a partial tenotomy of the eye-muscles has been performed. I have had two patients intensify the effect desired from a graduated tenotomy by a fit occurring soon after the operation.

In very severe fits, the teeth, and even the jaw itself, have been broken by the violent muscular movements, the tongue completely divided, the clavicle fractured, and muscles torn across. One of my patients worked his way through the ash-door of a furnace (12x16 inches) during an epileptic fit, and the brick-work had to be removed to extricate him. Fortunately no fire was in the furnace.

Many epileptics fall very violently when the fit comes on, because of insufficient warning. It is very common, therefore, to encounter scars on different portions of the body in these subjects. The edges of the tongue usually give more or less evidence of previous attacks of "grand mal." They are badly scarred, or ragged, from imperfect union of old wounds.

PETIT MAL.

The milder forms of epilepsy may assume a variety of types. Personally, I do not regard any attack as one of true epilepsy unless consciousness is more or less completely lost; hence, I do not usually include among this particular class those subjects who suddenly have some of the premonitory symptoms of *grand mal*, already described, and still retain a perfect knowledge of their surroundings.

Subjects afflicted with *petit mal* often assume a fixed attitude without any premonition of an attack, and stare unconsciously for a few seconds. I once had a patient who would frequently do this at a card-table without dropping a card or losing the run of the game. He played for very heavy stakes and was usually a winner.

Occasionally, these subjects will stop in the middle of a sentence, grow pale or red, and remain motionless for half a minute or more with the eyes staring into vacancy. They will then finish the sentence and be unaware of the intermission which had possibly alarmed the rest of the company.

Facial grimaces or slight twitchings of the muscles may occur in more severe attacks of this character, and the urine may be voided unconsciously.

Some attacks of this type are exhibited by the patient walking about in an aimless way, with inarticulate mutterings, as if in search of something.

Petit mal is usually associated with some ill-defined aura, which leads the patient to suspect that he has had an attack accompanied by a temporary loss of consciousness. Some subjects compare these attacks to "a dream;" others feel dizzy or nauseated; a few suffer an indescribable physical distress. I have had several patients of this class who have told me that they "became blind" for a moment. An uncontrollable dancing of the eyes (nystagmus) occurs during these attacks in one of my patients, of which he is conscious.

Momentary strabismus is not infrequently observed in epileptics. It may occur independently of a fit or in conjunction with one.*

IRREGULAR EPILEPSY.

These attacks are of a peculiar kind, which are indicative of a condition designated by Hughlings-Jackson as "mental automatism." Hammond classes them as aborted paroxysms.

Such attacks are characterized by acts on the part of the patient of an impulsive and unnatural character, of which he is unconscious. They simulate in some cases attacks of momentary insanity. There are no muscular twitchings, as a rule. These attacks may occur in subjects who have never had either *grand mal* or *petit mal*. They are usually of short duration (a few minutes only, as a rule). Acts of violence are not uncommon in these attacks. Patients often wander without proper preparation through crowded streets, commit acts of immodesty or indecency, utter lewd expressions, etc., during these attacks, without a knowledge of doing so. Some suddenly find themselves standing or sitting in unexpected places (as in a closet), or committing some act which they had no will and often no motive to perform. These attacks may occur at any time of the day or night, and cannot usually be traced to any special cause. Kleptomania and other unconscious acts of crime may be attributed (in some cases) to this particular form of epilepsy.

Diagnosis.—The various types of epilepsy may be confounded with comatose states and with other convulsive attacks; such, for example, as those of cerebral congestion; alcohol- or opium-poisoning; the convulsions of uræmia, hysteria, apoplexy, cerebral organic lesions, and feigned epilepsy. The table on the following page will aid the reader in the diagnosis of some of the more important conditions mentioned.

COMPLICATIONS OF EPILEPSY.

Various forms of *mental disturbance* may develop in connection with epilepsy. I have observed several cases where such disturbances have assumed the condition of permanent insanity; but they are, as a rule, of short duration. The condition described as "irregular epilepsy" is particularly liable to manifest itself in this way. It is stated by Reynolds that one-tenth of all cases of epilepsy devclop epileptic mania. This proportion seems to me to be somewhat excessive.

* In this connection I may remark that the constant efforts which epileptics commonly and unconsciously make to avoid being cross-eyed is, in my opinion, one of the causes of their attacks. If they were actually cross-eyed, they would learn soon to disregard the visual image of the distorted eye, and their diplopia would be clinically of no account. As it is, they suffer in many cases from a very high degree of "latent" insufficiency, which they instinctively endeavor to overcome in order to prevent diplopia. This subject has been discussed on a preceding page.

	EPILEPSY.	CEREBRAL CONGESTION.	APOPLEXY.	URÆMIA.	HYSTERIA.	MALINGERERS.	SYNCOPE.	ORGANIC LESIONS OF BRAIN.
SENSIBILITY.	Abolished during fit.	Usually unaffected.	*Anæsthesia*, as a rule, in one side.	May be unimpaired.	May be diminished.	Normal (in conjunctiva).	Not abolished.	Affected, if sensory area is involved.
PUPILS.....	Dilated during fit.	Contracted.	Often *irregular*.	Variable.	Normal.	Normal.	Frequently dilated.	May be irregular.
TONGUE.....	Scarred or ragged.	Not bitten.	Not bitten, as a rule.	Not bitten, as a rule.	Not scarred or bitten.	Not scarred or bitten severely.	Not bitten.	Not bitten.
CONSCIOUSNESS.....	Lost during fit.	Retained, as a rule.	*Prolonged coma.*	*Coma.* Very prolonged and deep.	Rarely lost entirely.	Can be made to exhibit it.	Incomplete coma.	May be unaffected.
URINE.....	Normal.	Excess of phosphates. Oxalate of lime	Normal.	*Albumen and casts.*	Excessive in quantity; pale in color.	Normal.	Normal.	Normal, as a rule.
HISTORY.....	*Inherited predisposition.* Previous attacks of a similar kind.	Insomnia. Cranial pain. Vertigo.	Usually negative.	*Ascites. Nausea. Vomiting. Waxy pallor. Urinous odor. Headache.*	Hysterical symptoms precede or accompany the attack.	Suspicious.	Heart-lesion. Anæmia. Chlorosis, etc.	Pain. Tremor. Paralysis. Imbecility. Affections of speech. Affections of special senses.
STUPOR.....	Follows fit, and is short.	May occur.	Is prolonged. Stertorous breathing.	Liable to be prolonged.	Does not follow fit. No stertor.	Assumed.	Doesnot follow attack.	May be developed.
PARALYSIS...	Absent.	Absent.	Present; usually of motion and sensation on one side.	Absent.	May exist.	Absent.	Absent.	May occur.
RECOVERY ...	Moderately rapid.	Modified bythe type which exists.	Slow, if not fatal.	Slow, if not fatal.	May be slow or rapid.	Rapid.	Rapid.	Slow, if not fatal.
PULSE.....	Small or absent during tonic stage.	Irritable. Easily excited.	Slow and full.	Variable.	Small, feeble, rapid.	Normal.	Rapid.	Modified by seat of lesion.
TEMPERATURE OF BODY.....	High during attack (103°).	Usuallynormal	Lowered.	Low; may reach 92°.	Normal.	Normal.	Normal or lowered.	May be raised or lowered.
ASPHYXIA....	Very marked in clonic stage.	Absent.	May exist.	May ex st.	Only partial or absent.	Absent.	Absent.	May exist.

Epileptic mania may lead to motiveless and atrocious crimes in some cases, and to acts of indecency and vulgarity in others. Again, illusions of the special senses, melancholy, or preternatural buoyancy of spirits may be evidences of this complication. Epileptic delirium is therefore not always of a dangerous character; although it is liable to become so, and deserves careful watching.

When idiocy is associated with epilepsy, the former is generally congenital or the result of a prolonged use of bromides.

Prognosis.—Authorities differ respecting the indications for an extremly unfavorable prognosis in epilepsy. Some assert that extreme frequency of the paroxysms have a serious significance; while others claim that the most intractable cases are those who have attacks at prolonged intervals.

It is generally conceded that cases of long standing are more rebellious to any form of treatment than in those where the "epileptic habit" has not been developed to the same extent.

The criticisms which I would make, in the light of my preliminary remarks to this chapter, upon the two former paragraphs would be this: First, that it is difficult (in cases where the attacks are very infrequent) to tell positively when all causes of reflex irritation are removed without waiting a long time; secondly, that most cases of long-standing (irrespective of the frequency of the attacks) are usually thoroughly bromidized before reflex causes are searched for in a scientific way, and are suffering as much from the deleterious effects of drugs as from the epileptic state.

Although mental failure is generally regarded as a sign of evil import in epileptics, I have seen several cases in private practice where a tenotomy of the eye-muscles has been followed by marked recovery of the intellectual faculties, after all drugs have been discontinued. Photographs of several patients operated upon by my friend, Dr. Stevens, in the Willard Asylum, during the summer of 1886, seem to prove quite conclusively that some cases of even chronic insanity as well as epilepsy are capable of rapid improvement when the delcterious effects of eye-strain and drugs are no longer borne by the patient.

One case of insanity recovered his full mental faculties in a few weeks after I performed a tenotomy of his internal recti muscles, and he has shown no tendency to relapses. An epileptic, in whom all convulsive attacks ceased after I performed the same operation upon her, was very deficient in intellect before that step and is now rather above the average in mental power. One of the most remarkable cases that ever came under my observation was that of a combination of chorea, epilepsy, and idiocy in a girl about eleven years of age, who completely recovered her health, strength, and mental faculties when a refractive error in the eyes

was corrected by glasses, and a serious combination of muscular defects in the orbit was adjusted by tenotomy. This case was one that I saw some three years ago, in connection with the practice of Dr. Stevens. At the first examination, the child could not walk without being supported on both sides, drooled constantly, talked unintelligibly, answered questions with apparently little conception of their import, could hardly sit unsupported in a chair on account of chorea, had epileptic seizures repeatedly during the day and night, and presented a most pitiable and apparently hopeless aspect. I saw her again, about a year after the operations were performed, at the request of Dr. Stevens. I found her free from chorea and epilepsy, able to run and skip a rope unaided, rosy-cheeked, and in full possession of her mental faculties. Photographs of this case have been already published by Dr. Stevens.

That a persistence of epileptic attacks for years does not necessarily render recovery impossible is proven by the fact that I have personally had three cases where convulsive seizures have been thus far arrested by tenotomies which I performed upon the eye-muscles. All of these cases had been kept constantly under bromides for several years without apparent benefit before they were placed under my care. Over a year has now elapsed since two have taken any drugs or have had an epileptic fit, and the third has passed several months without an attack. Dr. Stevens reports several such cases; which tend to prove that permanent organic changes in the cells of the cortex do not necessarily occur as a result of frequent and long-continued epilepsies.

Finally, it may be said that little danger to life is to be apprehended in epileptic seizures. I have never known of a case where death occurred directly from the fit; although many cases may have met their death indirectly from that cause, through suffocation, drowning, etc.

Treatment.—From what I have already said in the preliminary remarks concerning functional nervous diseases, it must be evident that medication in these affections constitutes with me rather a *dernier ressort* than a harbor of refuge. In this view, I feel that I am, as yet, quite alone among neurologists; but I am too sure of my position to doubt that in time the views which I here advocate will not be without other enthusiastic followers.

The first step in the treatment of epilepsy (and the same remarks hold good for all functional nervous diseases) is, in my opinion, to ascertain (by all scientific means yet noted and by careful observation and record) if any *cause for reflex irritation* exists. While I believe that such a cause will be found, in a very large proportion of such cases, in the eye, I would not be construed as stating that the examination of this organ should constitute the beginning and the end of such a search. The womb and the ovaries in females, the genitals in the

male, and the teeth and rectum in both sexes may require a careful examination.

I should be very strongly led to suspect an eye-defect or an "insufficiency" of some of the ocular muscles in case (1) the patient was a young adult; (2) if he had a tubercnlous ancestry; or (3) if he gave a history of epilepsy, hysteria, chorea, or insanity, in some branches of the ancestral line.

The conditions of the visual apparatus which seem to predispose to these conditions are peculiarly liable "to run in families," and to be transmitted (as features and mental traits are) from parents to their children. It will generally be found that "sick-headache" is another "family ailment" among this type of subjects; in case a heredity to the severer forms of functional nervous disturbance is not clearly brought out on a careful line of examination. The close relationship which exists between this common and excruciating form of pain and eye-strain is now too well recognized to need further comment.

The next question which I should feel myself called upon to decide in a case of epilepsy (provided the previous lines of inquiry respecting reflex causes had been prosecuted with due regard to the details given for such examinations and had yielded negative results) would be to determine if *traumatic conditions* did not exist, which might account for the epileptic seizures.

The steps indicated for the relief of such conditions would depend entirely upon the nature and seat of the injury received. Cerebral localization would then come into play, and surgical interference would be justified or contra-indicated by the conclusions drawn from that source.

In the third place, it is always best to examine with care in any case for the symptoms of *organic changes* of a local character either in the brain or its coverings. The hints furnished in Section II of this work can be reviewed with advantage in this connection.

Finally, if all of these lines of investigation give us negative results, medication must, of necessity, come to our aid,—as an empirical and most unsatisfactory way to an inquiring mind of controlling or modifying symptoms which we are unable to explain, and upon the cause of which pathologists have shed but little if any light.

A few illustrative cases may be cited in this connection with possible advantage to the reader:—

Case I. *Chronic Epilepsy.*—Male, aged forty-three, merchant. Began to have severe epileptic fits when seventeen years of age. Had masturbated when a boy, and had been addicted in later years to excessive venery.

Family History.—One brother is a confirmed dipsomaniac; the father died of paralysis; one sister is a victim to sick-headaches; no phthisis has existed in the family, so far as could be ascertained.

The epileptic seizures of this patient varied in frequency from two or three a week to one in three months. He came under my care in 1871 (when twenty-eight years old), and was treated by me for many years with enormous doses of the bromides of potassium and sodium. These salts reduced the attacks to about four a year. Stopping the bromides invariably increased the frequency of the attacks.

*Eye-defects.**—In January, 1886, his eyes were examined after his return from an extended residence in the South. He showed under atropine a *latent hyperopia* of 2.50 D., and also a manifest *esophoria* of 4°. Subsequently several degrees of "latent" esophoria also manifested itself.

Partial tenotomies were performed upon both interni, and hyperopic glasses (+ 1.50 D.) were given him. Since the first operation (January, 1886) he has taken no bromides and has not had a convulsion. He has twice been "at death's door" with fevers, but he has shown at no time any epileptic tendencies.

Case II. *Chronic Epilepsy.*—Female, aged twenty, unmarried.

Family History.—The father died of apoplexy. No hereditary tendency to nervous disease or phthisis could be discovered

The epileptic seizures had existed for five years and developed after an excessive use of the eyes in sewing upon a black material. Menstruation was regular. The epileptic fits were, however, more frequent during the week prior to and following the menstrual epoch. Under large doses of bromides and ergot she had once in her history passed six weeks without an attack; but she averaged, when I first saw her, about six attacks each month. An epileptic attack could usually be induced by *fixing the eyes for a few minutes intently upon some near object.* She had at one time as high as thirty severe fits in twenty-four hours. When the bromides were withdrawn from this patient, the fits increased to several each day (often as high as ten severe seizures). She had for years suffered from obstinate constipation and pain during her menses.

Eye-defects.—This patient was found to be *absolutely emmetropic* both before and after the use of atropine. She showed, however, an esophoria of 5° and a very low power of abduction. Subsequently, a large amount of latent esophoria developed.

Several partial tenotomies were performed upon the interni of this patient during an interval of some four months until all latent esophoria was apparently overcome. After the first operation, the lacrymal secretion, which was singularly defective, became normal, her mental despondency disappeared, and her attacks rapidly diminished in frequency. Since June 10, 1886, she has had no fits to my knowledge, nor to that of her physician so far as I can ascertain. The case was lost sight of by both of us some months ago, much to my regret. When I last tested her eyes, she showed no defect and was apparently in perfect health. She had passed several menstrual epochs without any epileptic seizures.

In this case I would call attention (1) to the fact that emmetropia existed; (2) to the fact that the eye was the apparent exciting cause of her attacks; (3) to the fact that epileptic seizures could be induced by excessive use of the internal muscles of the eye; and (4) that the esophoria was "latent" to a very marked degree.

In addition to these points of interest, another fact is worthy of passing remark, viz., that the relief of the "eye-strain" was followed in this case by a total disappearance of the habitual constipation that

* For the meaning of terms employed in connection with muscular anomalies of the orbit, the reader is referred to page 143.

previously had existed, and that menstruation ceased to be accompanied by pain up to my last notes on the case. This experience, although apparently a coincidence, is not by any means infrequent with female patients, in my practice, after a partial tenotomy of an eye-muscle for the relief of an abnormal tension within the orbit. It is probably to be attributed to the fact that the nerve-power of the patient improves rapidly after the excessive expenditure of nerve-force demanded by abnormal eye-tension is arrested, thus allowing of an improvement in the functions of the other viscera. The introductory remarks of this chapter will, I trust, make this explanation clear to my readers and bring this statement more into apparent harmony with physiological laws.

Case III. *Chronic Epilepsy.*—Male, unmarried, aged twenty years.

Family History.—No consumption among the ancestors or immediate family. Several members of the family suffer from headaches.

Eye-defects.—Hyperopic astigmatism (0.75 Dc. and 0.50 Dc.), esophoria (manifest) of 6°, and latent hyperphoria of a very high degree. The latter has proved very persistent, and has only lately been satisfactorily corrected. The progress of the case has also demonstrated that a high degree of latent esophoria had to be corrected in excess of what he at first manifested.

This patient was a victim to the severest type of chronic epilepsy. His attacks were extremely frequent and severe. Going from light into darkness would invariably cause an attack and a total loss of consciousness. I personally attended him, in connection with Dr. G. W. Leonard, of New York, when he had fifty-two epileptic seizures in eight hours, each fit lasting exactly three minutes. All medicinal treatment had proved inoperative. His attacks began while he was at school as a child, and were preceded by a difficulty for months of keeping his place on a page while reading. He used to hold his finger on the line to aid him in reading.

His improvement, after repeated tenotomies upon both interni and the left superior rectus, has been most remarkable. His attacks have been decreased over 75 per cent. He is still under observation, with a prospect of still greater improvement, if not of a permanent cure. He has taken no bromides or medicine of any kind save an occasional diuretic (tincture of iron) for sluggish kidneys.

The photographs of this patient, which I now possess, hardly show the happy change which has occurred since the last was taken, in spite of the complete cessation of the bromides for about eighteen months.

In place of the dull, apathetic, and sluggish features which indicate the impaired mental state of the patient from the bromides, in the second photograph an animated expression and a shortening of the face can be seen, which are much more apparent to-day even than when it was taken. The change in his general health and physical strength has been even more marked than his facial changes.

Case IV. *Chronic Epilepsy.*—Female, unmarried, aged nineteen years.

Family History.—Paternal grandmother died of phthisis. The mother has migraine. The brother has migraine. A paternal aunt was insane.

Eye-defects.—A high degree of myopia (3.75 D.) and myopic astigmatism (1.50 D.).

Esophoria (manifest) of 9°. A high degree of latent hyperphoria was also discovered later.

Prior to my first examination this young lady was considered a hopeless epileptic. She had been for years under the care of several of our most noted neurologists and oculists. After a partial relief of her abnormal eye-tension by tenotomies, she went over seven months without an attack, and regained her mental faculties, which had been somewhat impaired by bromides. Within the past four months she has had six attacks of epilepsy, two of which followed fright, one an imprudence in eating salads very late and immediately before going to bed, and the fourth after the excitement attending a departure for a pleasure excursion. She has lately manifested a latent muscular defect which I have yet to overcome. From a letter addressed me by her father not long since I quote the following paragraphs:—

"Nothing but an inability on my part to pay for your services would persuade me to remove my daughter from your care, and if I could not pay I would ask you for charity to keep her.

"Her whole being has been altered, and her physical condition is better than for eight years."

The photographs of this patient show a very marked alteration in her physical and mental states, as a result of the relief of the muscular errors detected in the orbits. She is now allowed to enjoy privileges which were considered impracticable prior to this treatment, such as an unrestricted diet, horseback exercise, visits to the city unaccompanied, attendance at social gatherings, etc.

As this case is well known to several prominent medical men of New York City, it may be proper for me to state that since October 10, 1886 (some nineteen months), this patient has had but eight epileptic seizures. Prior to that date my records go to show that, even when under bromides, from one to six fits a day often occurred, and that the nocturnal attacks (which were very frequent) were not always noted. It is safe, therefore, to say that, had she received no medicinal treatment or correction of her eye-defects during the past nineteen months, the total number of seizures would probably have been more than a hundred times this number. She had been known to have as high as seventeen epileptic seizures during one night. Her attendant has assured me that since my treatment was commenced all nocturnal attacks have ceased.

The point may be raised that a report of any epileptic case, until the full limit of three years has been passed without an attack and without the employment of any medicine which would control the epileptic tendency, must be considered as somewhat premature. In reply, I would say that the cases I have brought forward are not represented as cases of radical cure. They are adduced simply as a clinical evidence that the frequency of such attacks has been greatly modified, and in two instances completely controlled for long periods of time, without the aid of drugs. On the other hand, there are now to my knowledge several cases of chronic epilepsy similarly treated that might be brought forward (did I

deem it necessary to quote from the experience of another) which have fulfilled all the requirements which would justify the belief in a radical cure of chronic epilepsy. One of these patients has passed over seven years without a fit, and several have exceeded the three-year limit.

We all admit, I think, that epilepsy is certainly the gravest of all the functional nervous maladies, and that it is, as a rule, incurable by drugs; hence, as I have remarked in a previous discussion concerning this subject, "one radical cure of epilepsy without the aid of drugs offsets a thousand failures as a scientific proof of a discovery."

It is impossible for any one not familiar with the difficulties encountered in the treatment of these subjects (already discussed) to appreciate the fact that, in some cases of epilepsy, eye-defects may exist which can not be thoroughly rectified; and that, even in favorable cases, time and patience are important factors in the treatment.

Epileptics usually present, in my experience, in addition to errors of refraction, anomalies in both the lateral and vertical movements of the eyes; and in some cases the oblique movements are probably at fault. Moreover, experience with these subjects demonstrates clearly to my mind that the muscular anomalies which exist are generally "latent" to a marked degree.

It should be remembered also that a victim to chronic epilepsy who is rendered by any treatment as free from attacks *without the bromides* as he was when under their deleterious influence has been *very markedly benefited;* again, that if a marked diminution of the attacks has been effected, the patient has double cause for gratitude; finally, that if the attacks are arrested *in toto* without drugs, it is to-day one of the most remarkable facts recorded in medical literature.

There is a point where opposition to carefully made clinical statistics respecting new views ceases to be conservatism. True conservatism is the brake upon the engine of progress. It is to be used in checking its speed when going too fast, but not in preventing its advance, even if the country is an unexplored field. Too often in medicine the term conservatism has become a favorite synonym for bigotry and intolerance.

My own personal observations of epileptic subjects, since my attention was first drawn to the importance of a systematic examination of the eyes and the eye-muscles in these cases, are not sufficiently extensive perhaps to warrant any general deductions respecting the percentage of such errors which would be accepted as conclusive; and those of Dr. Stevens upon a larger number of cases might also be regarded as somewhat insufficient. As yet, no scientific records of the examinations of epileptics in respect to abnormalities of the ocular muscles have been systematically made, to my knowledge, in other quarters. Ophthalmoscopic tests have been published and errors of refraction have been

repeatedly noted, but the tests to determine the existence of esophoria, exophoria, and hyperphoria at twenty feet (and also at fourteen inches if modified by accommodative efforts on the part of the patient) have yet to be made on a large number of cases by competent experts before this matter can be spoken of in an authoritative manner. I can only say, in this connection, that I have yet to meet a case of epilepsy in my private practice (which was not clearly the result of traumatism or syphilis) that did not exhibit more or less muscular error connected with the eye after the refractive error (if such existed) was corrected with the proper glass. It is but natural, therefore, to expect that further records of epileptic cases which will doubtless appear hereafter from time to time shall give satisfactory evidence of care and skill in these important examinations.

The opposite page shows the results of an eye-examination upon sixteen consecutive cases of epilepsy which came under my care during the early part of the past year in my private practice. It also has a bearing upon some other points relating to epilepsy which have been discussed in preceding pages.

It will be perceived that most of these cases are of several years' duration,—enough to be considered chronic. In only two of these cases was the eye emmetropic. All but one (a syphilitic case) showed a departure from the physiological state of equilibrium of the eye-muscles. Several had impairment of intellect. In most of the cases, the epileptic seizures were very severe and of frequent occurrence. All but two had been kept under the bromides for long periods of time.

After deducting from this list those cases which either refused to have tenotomy performed, or who failed from other reasons to put themselves under treatment, eight remain that have been operated upon with the object of correcting some existing muscular error. Of these eight, three have been thus far very markedly relieved of epileptic seizures by partial tenotomies of eye-muscles; and five are still under observation. No medicines have been administered to any of these eight cases to aid in controlling the epileptic attacks since they have been under my care. Of the five that are still under observation, one has granular kidneys as a complication; and the remaining four have still some muscular errors in the orbit that have not been perfectly corrected up to the time of writing. One of the four has had epilepsy over twenty years, during most of which time she has constantly taken large doses of bromides of potash, soda, and ammonium.

The three cases which are to-day apparently relieved of attacks were all of the chronic type. All had taken enormous doses of the bromides, and had reluctantly been forced to abandon them, either at my request or when they ceased to be efficacious in controlling the attacks.

CASE.	AGE.	COMMENCEMENT OF ATTACKS.	FREQUENCY OF ATTACKS.	SEX.	EYE EXAMINATION.		MENTAL STATE AT DATE OF EXAMINATION.	REMARKS.
					Refractive Error.	*Muscular Error.*		
1	46	At age of 26.	Has had 15 in a day.	M.	H.+Ah.	Esophoria, 10°. Hyperphoria, 2°	Has had attacks of epileptic mania.	Refused operation. Addicted to chloral habit.
2	18	At age of 14.	Every 6 months.	F.	H.	Esophoria, 6°.	Normal.	Refused operation. Addicted to chloral habit.
3	43	At age of 14.	One in 4 months.	M.	H.	Esophoria, 4°.	Normal.	Apparently relieved by operation. No fit for the past year.
4	13	Since a baby.	Several daily.	M.	H.+Ah.	Esophoria, 5°. Hyperphoria, 1°.	Fairly intelligent.	Still under treatment.
5	19	At age of 13.	Has had 17 in a day.	F.	M.+Am.	Esophoria, 6°. Hyperphoria, 1°+.	Markedly impaired.	Apparently relieved by operation. No fits for 7 consecutive months.
6	21	At age of 12.	Has had 52 attacks in one day.	M.	M.+Am.	Esophoria, 10°. Hyperphoria, 1°.	Markedly impaired.	Has granular kidneys. Attacks greatly lessened since operation.
7	20	At age of 15.	Has had 10 attaeks in a day.	F.	Emmetropie	Esophoria, 5°.	Normal.	Apparently relieved by operation. No fit for past year.
8	26	At age of 22.	Average of 1 a week.	M.	H.	Esophoria, 3°.	Became insane and dangerous.	Sent to an asylum.
9	19	At age of 11.	Average of 2 a week.	F.	Am.	Hyperphoria, 1°.	Impaired.	Refused operation.
10	30	At age of 28.	Has 2 in a month.	M.	H.	Normal.	Normal.	Due to syphilis. Cured by specific treatment.
11	19	At age of 12.	Average of 1 a day under bromides.	M.	Emmetropie	Esophoria, 13°.	Markedly impaired.	Still under treatment. Attacks very infrequent since operation.
12	17	At age of 13.	Average of 1 a month.	M.	M.+Am.	Esophoria, 9°. Hyperphoria, 1°.	Normal.	No attacks since last operation; performed some 9 weeks ago.
13	21	At age of 8.	Average of 2 a week.	M.	H.	Esophoria, 3°. Hyperphoria, 1°.	Markedly impaired.	Operated on. Has gone three months without a fit.
14	37	At age of 16.	Average of 8 a week under bromides.	F.	M.+Am.	Esophoria, 2°. Hyperphoria, 1°.	Markedly impaired.	Not operated upon as yet. Has been 20 years under bromides.
15	3	Since a baby.	Average at first 2 a day —now infrequent.	F.	H.	Esophoria, 5°.	Normal.	Operation postponed on account of uncertainty of tests made.
16	12	At age of 8.	Average 2 a week.	M.	H.	Esophoria, 5°.	Semi-idiotic and at times insane.	Refused operation.

The terms used in recording the eye examinations are fully explained in Section II of this work (pages 147 and 148). Photographs of all cases operated upon were taken before surgical treatment was commenced.

One had had epileptic seizures for nearly thirty years, one for six years, and one for five years prior to the operation.

Finally, the cases reported by Dr. Brubaker of recoveries from epilepsy after the removal of all sources of *dental irritation*, and of many others which show that relief has followed the cessation of *ovarian irritation*, should not be allowed to pass without notice.

Such cases as these have a very important bearing upon the question which is now being most earnestly investigated by some of the leading minds in the profession, viz., whether the present methods of medicinal treatment of epilepsy and its allied disorders of the nervous system are not destined to be superseded by more rational and scientific methods of research for the underlying causes of their imperfectly solved problems.

In the preceding table, it will be perceived that the degrees of insufficiency which existed in each patient is noted. It may be well to state, in this connection, that the highest degree of error detected during my observations of each case is noted (rather than the lowest), because this probably indicates the closest approximation to the actual state of the patient which we can scientifically record. Probably the "latent" insufficiency vastly exceeded in each case the amount actually noted (see p. 456). In one of my cases (now apparently cured) one degree of esophoria was all that was detected on the first examination; yet, in spite of this fact, a very free but incomplete division of both interni was required to establish the physiological equilibrium. The attacks of epilepsy then ceased, and have not returned, to my knowledge, for over thirteen months.

Finally, it may be added, in justice to the views here advanced, that it is not impossible that in some of these apparently successful cases a latent insufficiency may still be lurking, which the patient may be able to overcome for a longer or shorter period of time without producing any abnormal nervous manifestations. Should such a recurrence of ocular tension ever manifest itself, it ought to be corrected by a repetition of tenotomy, even if the epileptic seizures should not reappear or the patient apparently suffer from its presence. One such case occurred under the observation of Dr. Stevens, where epileptic seizures were arrested for several months by partial tenotomies in an apparently hopeless subject. A return of the attacks led the patient to again seek his advice, and it was found that a high degree of latent insufficiency had manifested itself. This patient refused, under the advice of friends, to continue the treatment (which had produced such marvellous results but a few months before), and he returned to bromides. This case has been used by an opponent of these views as an evidence of the lack of permanency of the results obtained. The injustice of such a conclusion must be obvious to any intelligent reader.

Clinical facts go to prove conclusively that in almost all epileptic and choreic subjects the manifest insufficiency is much less than that which actually exists. It is only after patient waiting, in some cases, that we find we have more to deal with than the patient at first disclosed. Whenever we find it, it should be rectified by proper methods and at the proper time. It should be anticipated and persistently looked for, over a period of many months. If it fails to develop within a year, we may be hopeful that we have overcome all that originally existed. If it develops, we have simply noted a condition that it was reasonable to expect, and the necessity for further operative procedures is clearly indicated.

In one of my successful cases no latent insufficiency has thus far shown itself, although over two years have elapsed since the last operation. In another I have not had an opportunity of examining the patient for some months. In the third, a slight hyperphoria remains, which the patient now tolerates, but which may require correction at a later date.

Diet Treatment.—Many cases of epilepsy are materially benefited by a restricted diet. Perhaps the one which has given the best results consists in depriving the patient of meat-foods of every kind and description. Even soups are prohibited. Eggs and cheese are strictly forbidden.

The principle involved in this diet is the *withdrawal of nitrogen*, as far as it is practicable to do so, from the patient.

Milk is allowed, as are also its various preparations, such as buttermilk, skim-milk, koumiss, etc. Vegetables of every kind, bread, oatmeal, cracked wheat, ripe fruit of all kinds in moderation, and other non-nitrogenous articles of food constitute (with milk) the best means of nourishing these patients.

Medicinal Treatment of Epilepsy.—No work would be complete without some reference to the methods of treatment now generally adopted for the purpose of holding these attacks in check, in spite of the fact that all are more or less detrimental to health, and generally unsatisfactory both to the doctor and patient. Personally, I have never traced up a reported case of cure of genuine epilepsy with drugs alone, without finding that the cessation of the drug has been followed by bad results. The best authorities speak with extreme caution respecting the permanent benefits which may be expected of medication.

In *infants* and *children*, *epileptic seizures very frequently get well if let alone*—a fact that is to be explained by the susceptibility of the young to reflex nervous disturbances, and the multiplicity of causes of such derangements. This fact is too often overlooked when the efficacy of drugs in such subjects (affected with epilepsy) is called into question.

If, in spite of careful scrutiny into all the possible factors which have been mentioned as liable to cause epileptic seizures, nothing can be

found; or, if (when all that may have been discovered have been either satisfactorily removed or justly pronounced incurable) the epileptic attacks persist, nothing, unfortunately, remains for such a patient but the hope which drugs may afford of lessening or preventing subsequent attacks. In such a dilemma we are left, as physicians, to choose from the following list of drugs, such as seem best adapted to meet the indications: (1), the bromides of potassium, sodium, ammonium, iron, arsenic, calcium, etc; (2), preparations of zinc, preferably the oxide; (3), the various preparations of arsenic; (4), the nitrate of silver; (5), belladonna, in some of its various forms; (6), hyoscyamus; (7), osmic acid; (8), digitalis; (9), curare; (10), the nitrite of amyl; (11), strychnia; (12), the hydrate of chloral; (13), the iodides of potassium, calcium, and iron (especially if syphilis is suspected to be a factor in the case); and (14) preparations of nitro-glycerine.

The *bromides* of *sodium*, *potassium*, *lithium*, *ammonium*, and *calcium* probably stand first in professional estimation to-day as a remedy in epilepsy. These salts are commonly given in doses which vary from fifteen grains three times a day at the commencement to one hundred grains, as the patient becomes tolerant of them. They are preferably given in solution, and Seguin's suggestion that Vichy water be used as a solvent is a good one. Gower's method is to give very large doses at the beginning of treatment after breakfast in a goblet of water. He advises that two, three, four, five, and six drachms of bromides be given on successive mornings. He omits treatment for a week or two after the patient has been carried to a state of drowsiness or of mental sluggishness. Subsequently, he gives doses of twenty grains or more three times a day.

Most authorities advise the continuance of the bromides for two years at least. A late author says, "Patients may take sixty to ninety grains of bromide a day for six or ten years without injury." I would caution the reader against so extreme a statement. I have seen patients brought to a state of insanity and idiocy by very heavy doses, and all patients become more or less affected in mental power by its long-continued use. Personally, I am inclined to believe that the apparent benefits derived from the use of bromides is more than counterbalanced in most cases by their disastrous effects upon the nervous system.

If a skin eruption (acne) appears, arsenic may be judiciously given in connection with the bromides. The dose of Fowler's solution is from three to five drops for such a purpose.

The bromide of lithium is said to be tolerated by weak stomachs better than the other salts mentioned.

In connection with the bromide treatment, it is oftentimes important that patients take iron, cod-liver oil, and quinine. It is also well to stop

indulgence in alcohol, tea and coffee to excess, highly-seasoned and indigestible food (especially of an animal kind), and cold bathing. A regular movement of the bowels should be had daily. This may be aided by the drinking of mineral waters, or by the hot-water treatment (p. 248).

In nocturnal epilepsy, the largest dose may be advantageously given at night.

Hydrate of chloral is often combined with bromides. It should not be continued too long, as it affects the general health. The dose should not exceed fifteen grains, and it is well to combine digitalis with it, to prevent the possibility of heart symptoms.

Belladonna was first recommended highly by Trousseau in the treatment of epilepsy. Atropine is now generally used in doses of one one-hundredth of a grain. This dose may be given three times a day until the pupils become dilated and the throat unnaturally dry.

The *salts of zinc*, chiefly the oxide and the bromide, have been extolled as a cure for epilepsy. The bromide may be given in simple syrup and water, commencing with one-grain doses and increasing the amount gradually as long as nausea is not produced. The oxide is given in doses of from five to ten grains. If an eruption follows its use, combine it with Fowler's solution of arsenic.

The *nitrate of silver* is liable to produce a permanent blue staining of the skin. Its supposed benefits hardly warrant so great a risk; hence, it should not be continued long, in case it be employed.

Curare has been given by the hypodermic method to epileptic patients in doses of 0.03 grammes by Kunze and others. It is not to be administered oftener than the fifth day. It shows its toxic effects early in the eyes, sight being rendered dim and indistinct.

Osmic acid (in doses of 0.002 grammes) may be advantageously administered in combination with the bromides, according to the late publications of Wildermuth.

Finally, *nitrite of amyl* may be used by epileptics as a means of warding off impending attacks. It is best carried in small bulbs of thin glass (amyl-pearls) which may be crushed in the handkerchief and the fumes inhaled by the patient as soon as the aura is perceived. Two to five drops of this agent will generally prevent an attack for a while.

Nitro-glycerine as a therapeutic agent in epilepsy was first employed by Hammond and Weir Mitchell. The favorable results claimed for this agent seem to have fallen far short of general acceptance. The late report of Osler respecting its effects in epilepsy seems to show that the remedy rapidly lost its influence in those cases that, for a time, appeared to improve.

It may be given in pilules of $\frac{1}{100}$ of a grain, or in a one per cent. solution in doses of ♏v. The susceptibility of different patients to this drug varies, according to my experience; hence, it is wise to begin with small doses, and to slowly increase the dose until its physiological effects are manifested by a flushing of the face, a sense of fullness in the head, and a peculiar glow of the whole body.

Finally, when the condition known as the "*status epilepticus*" develops, during which the convulsive attacks are practically constant, it is well to treat the patient, according to circumstances, by ice to the spine, inhalations of nitrite of amyl or chloroform, repeated doses of chloral, or subcutaneous injections of morphia.

CHOREA.

This form of functional nervous disturbance is most commonly encountered in children. It may, however, begin in adult life; and it has been known to develop in advanced age. It is commonly known among the laity as "St. Vitus' dance."

Etiology.—This disease may be congenital. It is particularly common in the offspring of tuberculous parents. The period of second dentition (the sixth and seventh years) is one that is apparently very susceptible to these attacks. I have several times observed it in adults between the fifteenth and twenty-fifth year of age; and in old age it has been often known to follow grief, fright and violent mental emotions. A predisposition to chorea seems to exist in subjects that have had isolated cases of hysteria, epilepsy, insanity and neurasthenia among different branches of their family.

Most authorities mention, among the exciting causes of this disease, excessive joy or grief, severe fright, traumatisms to the head or back, infectious diseases, rheumatism, anæmia, onanism, reflex irritation arising from the genitals, the intestine, neuromata, dentition, pregnancy, etc.; and, finally, certain atmospheric conditions. It seems to be somewhat more common among girls than boys. It may be acquired by imitation.

In the light afforded by the latest researches in reference to the existence of eye-defect as a cause in producing chorea, as well as the so-called "predisposition" thereto, the following deductions of Dr. Stevens' seem to prove quite conclusively that *hyperopia* (often latent) *exists in an enormous proportion of choreic subjects, and that muscular error in the orbit frequently coexists.* From the prize essay of that author the following deductions are taken:—

In 118 cases of chorea, 78 had simple hyperopia (or about 67 per cent.); 13 had hyperopic astigmatism (or about 11 per cent.); 5 had mixed astigmatism (or about 4 per cent.); 6 had unequal myopia in the

two eyes (or about 5 per cent.); 11 had myopic astigmatism (or about 9 per cent.); 5 had no marked refractive error, but a marked museular defeet existed in the orbit (or about 4 per cent.).

It will be seen from these statements that nearly eighty per ecnt. of the subjeets examined by this author (and they were eonsecutive eases) exhibited either simple hyperopia or hyperopic astigmatism, and that four per cent. more had mixed astigmatism, whieh entitles them to belong to this elass of refractive error. Only fourteen per cent. had myopia or myopie astigmatism. Only four per cent. had no refractive error; and these had a suffieient degree of ocular insuffieieney to justify the view that reflex irritation from the visual apparatus was markedly present. In not a single case were the eyes perfeetly emmetropie and the ocular muscles in the state of physiologieal equilibrium.

My own personal experienee with ehoreie patients of a persistent type leads me to sustain in a general way the aecuracy of these observations. They are oftentimes difficult subjeets to examine with satisfaction, partly on aceount of their age, and partly on aeeount of the spasmodie movements of the head and body. I have found "latent" hyperopia of a high degree in all of my later eases whose eyes I have examined, not infrequently assoeiated with esophoria and hyperphoria. This subjeet will be more fully diseussed when the treatment of this disease is reviewed.

Morbid Anatomy.—The changes whieh exist in the brain or spinal cord in connection with chorea are unknown. It is very doubtful, to my mind, if any exist. All theories in relation to it are either pure assumptions, or are based upon insuffieient data.

Symptoms.—Various prodromal symptoms of this disease are mentioned by authors. Among these the following may be given: Anorexia, a disinclination toward mental or bodily pursuits, headaehe, restlessness, pains in the limbs and joints, irritability of temper, weakness of the memory, and many others.

As the disease develops, the patient gradually begins to exhibit a eertain *awkwardness of movement* in the extremities. Objects fall frequently out of the grasp. The child spills his food while eating. It becomes diffieult for the ehild to stand still. Attempts to write, sew or draw are imperfectly performed. Sueh ehildren are very often punished for supposed ill behavior or careless habits.

Later on, the symptoms beeome so unmistakable that the presence of aetual disease is no longer doubted. The patient may beeome incapable of dressing. The limbs or faee are no longer under the eontrol of the will. Involuntary movements of extension and flexion of the fingers, pronation and supination of the hand, shrugging of the shoulders, dancing of the legs, grimaees of the faee, and distortions of the body

become more or less constant. These patients may be unable to sit upon a chair without feeling a sense of danger from falling. I have known a child to be thrown out of bed by the violence of the spasmodic movements.

Speech, mastication, and swallowing may be seriously embarrassed, and the teeth have been known to be broken by the uncontrolled movements of the jaw.

The thoracic and abdominal muscles may, in rare instances, be so seriously affected as to produce cyanosis and a sense of impending suffocation; but I have never seen any affection of the heart, the bladder, or the rectum in chorea. Even the glottis may participate in the convulsive movements.

During sleep, the convulsive movements usually entirely cease. They are, however, sometimes very severe just preceding repose; hence these subjects often regard bed-time with a peculiar horror.

Strange as it may seem, these movements seldom create fatigue; although they may be incessant.

The whole body is not usually affected until late in the disease. Often a condition (known as "hemi-chorea"), in which the muscles on the right or left side are alone uncontrolled, is developed. Again, one arm and the face, or the face alone, may be affected. The left side is usually more severely attacked than the right.

The mental state of these subjects is generally far below the normal standard. They are prone to laughter over trivial things, or to attacks of weeping without apparent cause. They may develop idiocy or mania. Irritability of temper, a stupid demeanor, and impairment of memory are frequently observed.

Any unusual excitement of the mind or body is apt to intensify the muscular twitchings. Severe mental application, the reading of exciting books, the witnessing of dramas or other entertainments of that character, excessive indulgence in sports, etc., are to be discountenanced, therefore, in cases of chorea.

Choreic subjects are apt to be pale and anæmic. It is not uncommon, therefore, to detect an anæmic murmur over the heart and the jugulars. Sometimes the second sound of the heart is intensified.

It is difficult to carefully observe the pulse in chorea on account of the spasmodic movements. It has been stated that the arterial tension is diminished in the height of the disease.

I have never observed any change in the bodily temperature which might be regarded as directly due to chorea. An elevation of the temperature of the parts affected has been noted by some observers in hemi-chorea; but, in my experience, this is not always present.

In serious cases, insomnia may develop.

Complications.—Basedow's disease may be developed in connection with chorea. Aphasia has been observed as a complication of this affection; and, in rarer instances, paralysis of a transient charaeter has been known to develop. The joints may become swollen and painful. The pupils may be rendered sluggish to the effects of strong light, and be unnaturally dilated. Disturbances of the intellectual faculties have been known to occur during chorea, and to cause permanent idiocy or insanity.

Diagnosis.—This disease may be confounded with athetosis, post-paralytic chorea, hysteria, convulsive tremor, paralysis agitans, multiple spinal sclerosis, and some other forms of organic disease of the brain and spinal cord. The table on the following page will aid the reader in distinguishing between chorea and most of these affections.

The age which is generally attacked in choreic subjects practically removes all the difficulties of diagnosis. It is only in adult cases that we are called upon to discriminate closely between it and hysteria, convulsive tremor, paralysis agitans, and multiple selerosis.

The chief point in all of these discriminations bears upon the relation of choreic movements to willed muscular movements of the extremities. In chorea, willed muscular action is unexpectedly opposed by spasmodic movements which carry the part in some unnatural direction. In this respect the disease simulates incoördination of movement (which is prominently encountered in locomotor ataxia). Thus, for example, a choreic subject while eating will jerk a spoon away as it is about to enter the mouth; or, he may possibly strike the ear or nose with the fork. Such accidents are seldom observed when tremor exists, or when even in athetosis.

Prognosis.—Although fatal cases of chorea have been recorded, the prognosis is, as a rule, extremely favorable. Quite a large proportion of cases recover under judicious medication within ten weeks. Obstinate insomnia is a symptom of evil import. I have seen some very persistent cases of chorea in the adult which have withstood all medication, and have subsequently made a complete recovery when the eye-muscles have been properly balanced by partial tenotomies; and I regard the correction of refractive errors in all cases of chorea as a step of vital importance. It certainly tends to prevent relapses, which are particularly prone to occur in chorea. I believe the mental impairment often noted in connection with this disease to be due in a large proportion of the cases to "eye-strain" uncorrected. A hyperopia or hyperopic astigmatism in a child is certainly a defect of sufficient importance to merit early recognition and correction.

	CHOREA.	HYSTERIA.	ATHETOSIS.	MULTIPLE CEREBRO-SPINAL SCLEROSIS.	CONVULSIVE TREMOR.	PARALYSIS AGITANS.	POST-PARALYTIC CHOREA.
CONSCIOUSNESS..	Never lost.	May be lost.	May have been lost at onset of disease.	May be lost.	Never lost.	Unaffected.	May have been lost when the paralysis developed.
HALLUCINATIONS.....	Absent.	May exist.	May have existed.	May exist.	Absent.	Absent.	May have existed.
MEMORY.....	Usually impaired.	Seldom seriously affected.	May be enfeebled.	May be impaired.	Unimpaired.	Generally unaffected.	May be impaired.
PARALYSIS.....	Absent.	May have existed.	Absent.	Paresis and contracture.	Absent.	May develop late in the disease.	Has existed before the choreiform movements.
GENERAL SENSIBILITY.....	Normal.	May be diminished or abolished.	Normal in parts attacked, as a rule.	Markedly impaired.	Not affected.	Seldom impaired.	Anæsthesia may exist.
SPASMODIC MOVEMENTS....	Independent of voluntary movements. Are more or less incessant except during sleep.	Paroxysmal.	Are generally regular and constant, even during sleep.	Always excited by some voluntary movement.	Not continuous. Paroxysmal and more orderly.	More or less rigidity of muscles exists.	Unilateral, and due to attempts at voluntary movements.
NYSTAGMUS.....	Generally absent.	Absent.	Absent.	Often present.	Absent.	Absent.	Seldom present.
SPEECH.....	May be lost in rare cases.	Seldom affected.	Aphasia may develop.	Generally impaired.	Hoarseness and aphonia in some cases.	Generally unimpaired.	May be impaired by brain-lesion.
TREMOR.....	Absent.	Absent.	Generally absent.	Very marked when voluntary movements are attempted.	Absent.	Tends to subside when limbs are supported. Is otherwise constant.	May exist.
AGE OF PATIENT.	Most common in children.	Young adults—after puberty.	Adults.	Adults.	Adults.	Old age.	Adults.
GAIT.....	Jerking in case legs are affected.	Peculiar (see p. 165).	Not affected.	Peculiar (see p. 165).	Not affected between attacks.	Very peculiar (see p. 163).	Generally that of hemiplegia (see p. 162).
HANDS.....	Movements of are greatly embarrassed if affected by spasm.	Voluntary movements are seldom seriously embarrassed by spasms.	Fingers in continued motion.	Embarrassed by tremor and directed with difficulty.	Often convulsed during attack.	Deformed.	May be affected by contracture.

1.

2.

See page 499.

PHOTO-COLLOTYPE.

The following case illustrates the importance of correction of anomalies of the visual apparatus:—

Chronic Chorea of Thirty-one Years' Duration, affecting the Head, Face, and all the Extremities.—Female, aged thirty-three, unmarried.

Family History.—The father had pulmonary hemorrhages for many years. One paternal aunt died of "hasty consumption." Sick-headaches are very common among both paternal and maternal ancestors. Neuralgia is a frequent complaint among the paternal ancestors.

When two years of age, this girl developed chorea. The spasmodic twitchings steadily grew worse, in spite of the fact that her father was a physician, and that she had the services of the most skillful medical men from time to time. The twitchings began on the right side; but they subsequently involved the left side, and also the head and face.

She has suffered some from sick-headaches, as has also her sister. The *hands have gradually become so contractured* that all attempts to use them are more or less distressing. Her fingers could not be extended farther than would suffice to grasp small objects.

When I first saw this patient she was unable to write except by grasping the pencil with all the fingers and the palm of the left hand, and holding the left hand with the right hand as the spasmodic movements of writing were made. She walked with a peculiar unsteady and crab-like gait, ate with difficulty, and suffered great pain between the shoulder-blades and over the first lumbar vertebra (two points, by the way, which are very frequently attacked, in my experience, when eye-strain is present). She had never written with ink. Prior to menstruation (which occurred at seventeen years of age) the patient had experienced attacks (probably epileptic) which she describes as "those of numbness, followed by a loss of consciousness." She has had chronic constipation all her life. The memory and mental faculties are perfect.

When I first saw this patient the spasms were very violent, especially about the face and neck. The limbs were jerked about, the fingers too tightly clinched at times to grasp anything, and the speech was rendered peculiarly spasmodic and almost unintelligible at times. She sputtered, and at times ejected drops of saliva, when endeavoring to converse.

At the first examination she exhibited no refractive error; but, under atropine, a high degree of hyperopia (1·75 D.) was detected, and proper spherical glasses (+ 1 D.) were at once provided. In order to test her eye-muscles, the services of Professor J. Williston Wright, of New York city, who saw her with me by invitation, were invoked to hold her head. This he did with no small effort by clasping the head on either side, and firmly pressing her head against his body as he stood behind her chair. During this examination she whistled shrill notes on two occasions, and underwent the most violent facial and body contortions.

The results of this imperfect examination (necessarily so under such conditions) indicated to me that a high degree of hyperphoria existed; and, as I could not again see the patient for some months, I decided to perform a free but incomplete division of the left inferior rectus muscle. I then instructed the patient to try and get a photograph taken, if possible, before she saw me again. She laughingly said that she had never been able to have a picture taken, but she would do so if she could. She then departed for home with instructions to return to me for treatment in the autumn. The first picture received of this case was one that she was able to have taken three weeks after the operation, when her head and shoulders had become comparatively calm, as a result of the relief afforded by it. This photograph was deemed at that time a great success by herself and friends. You can see in it the blurred outlines which indicate that the movements were still somewhat active.

During the past autumn this patient has been under my care for some eight weeks. I have partially divided the right superior rectus and both externi in order to overcome a high degree of left hyperphoria and exophoria, and I have administered static sparks daily to the spine and limbs. The second picture will give, better than words can describe it, an idea of the wonderful improvement which has taken place. Prior to her departure for home (some weeks since) she could thread the finest cambric-needle, and pass her fare to the conductor of a street car without attracting the notice of passengers, or throwing it out of the window, as she certainly would have been apt to do two months previously. She can fully extend her fingers, walk several miles a day, write with far greater certainty and ease, and eat at a boarding-house table without exciting comment. Her limbs still twitch somewhat immediately before going to sleep, and, in the presence of strangers or when unduly excited, she still shows some spasmodic movements of the face and shoulders. When calm she is, however, perfectly composed, and almost entirely free from convulsive movements. She considers herself as practically cured; but I suspect that time and some further operative work upon the eye-muscles will be demanded before complete restoration to health is effected.

As I regard this case as one of the most distressing and typical cases of chronic chorea ever reported, it may be well to state that the patient is well known to Professor A. M. Phelps and Professor J. W. Wright, of this city, and Professor Woodward, of Burlington, Vt., and that she has been seen by many members of the profession, both from this city and distant States, during her treatment in my office.

During the whole treatment of this patient no drugs have been employed, and the photographs are from untouched negatives. I attribute to the static applications the rapid relief of the contractured state of the fingers and the improvement in her general strength; but, from many facts observed during my treatment of her, I am convinced that the relief of the eye-strain is alone deserving of whatever credit may be claimed for her recovery. Four weeks before she was dismissed from my care she read and sewed continuously for several days, and was immediately precipitated into a relapse, which as rapidly subsided when the cause was ascertained and its recurrence prevented.

Treatment.—My remarks concerning the causes of chorea and those which treat of the surgical relief of epilepsy bear strongly upon the cure of this form of functional nervous disturbance. I have not yet encountered a case for the past three years in my private practice where I have failed to find either a refractive error or an insufficiency of some of the eye-muscles. It has been my custom for some time past to carefully examine the eyes of these cases under atropine, and to correct all refractive errors (so found) subsequently with a glass that the patient could comfortably wear after the effects of atropine had subsided.

In previous pages of this work, I have combated the view (too commonly held by oculists) that the ophthalmoscope furnishes as positive information in respect to hyperopic defects as the ordinary type-tests made when the eye is fully under the effects of atropine. I have time

and time again found the best experts to be in error, when they have too implicitly relied upon ophthalmoscopic tests.

All that any one can determine with this instrument is based, of necessity, upon the presumption that the *patient's accommodation* as well as *that of the observer* is relaxed. This factor in the case is therefore two-fold; and is not always overcome, on the one hand, by an "acquired faculty" of the oculist, or, on the other hand, by directing the patient to look at an object over twenty feet from the patient's eye. To be sure of your results you must be able in any case to state positively that no accommodative efforts are made by the patient. This is positively ensured by a free use of atropine, and by no other recognized method.

Again, the tests for suspected errors in the eye-muscles are valueless, unless the refractive errors be intelligently corrected first. Moreover, if any muscular defect is detected, and confirmed by repeated examinations, the use of prismatic glasses does not fully meet the indications; because we have no way (yet known to science) of estimating the amount of "latent" insufficiency which may exist in each case.

The results obtained by partial tenotomies prove the truth of this statement beyond the possibility of its denial.

Respecting the *relationship of chorea to anomalies of the visual apparatus* I would make the following suggestions:—

(1) Choreic subjects belong to one of two classes: (*a*) Those who tend to get well under almost any treatment or even without treatment, and (*b*) those who fail to get relief from any medicinal aid. The latter tend to run a chronic course, usually one of unfavorable progression.

(2) The chronic form of chorea is one of the most serious and hopeless of nervous maladies. It is not infrequently associated with epilepsy or with mental impairment.

(3) Both forms of chorea are based, as a rule, upon a well-marked neuropathic or tubercular predisposition.

(4) The pathology of chorea is not known. No one has ever proved that it was a "constitutional disease," in the sense that an organic lesion was essential to its development.

Now, the remarkable case which I report belonged, without question, to the class which I think is generally regarded by neurologists as incurable, and as offering but little hope of marked improvement under any form of medication. In this girl, at least, all such attempts at relief had proved of no benefit. The convulsive movements had persisted for over thirty years, and the condition of the patient has steadily grown worse in spite of the best medical care. She had probably had a few epileptic seizures in girlhood, but her mind had remained unimpaired.

When Dr. G. T. Stevens read his paper on the relationship between refractive errors and chorea in 1876, he advanced views that were new

to the profession. Within a year, a paper on the same subject was published by another,* in which the view of Dr. Stevens, that hyperopia constituted an important element in most cases of chorea, was very strongly combated. The latter paper has been quite extensively quoted. It may not be inappropriate for me, therefore, to carefully analyze the paper referred to in this connection, as I feel that the conclusions of the critical reviewer are misleading, and certainly not in accord with my own observations.

This observer drew his conclusions from an examination of thirty-one cases of chorea, most of which, if not all, were taken from dispensaries. It is safe to infer, therefore, that the patients were not well educated. They may not have even known their letters sufficiently well to be regarded as accurate in reading test-type.

In the second place, the ages of the thirty-one patients reported show that twenty-two were less than twelve years of age. Four were six years of age, and one was only three and a half; one was seven, three were eight, three were nine, four were ten, and six were eleven years old. The question naturally arises whether (at these ages) the tests of vision usually made by the aid of test-types, when the patient is well under atropine, are reliable in children that are presumably ignorant.

In the third place, seventeen out of the thirty-one patients were found to be emmetropic in one eye or both when atropine was used by this observer. This is certainly a very remarkable fact, as it is a proportion which is contradicted by statistics gathered by equally competent observers from the examination of children's eyes under atropine.†

In the fourth place, the percentage of hyperopia and hyperopic astigmatism combined constitutes, according to this observer, about 55 per cent. of the total number. No myopia or myopic astigmatism was detected in any of the thirty-one cases. The latter fact is remarkable, and seems to cast further doubt upon the cases reported as "emmetropic."

Again, nineteen of the thirty-one patients are reported as having had "insufficiency of the interni." Now, I have examined within the last three years the eyes of a very large number of patients who were afflicted with various nervous disorders, and I have given special attention to the state of the eye-muscles detected by appropriate tests in these cases. I have found the condition of "insufficiency of the interni"

* Dr. C. S. Bull, *Med. Record*, June, 1877.

† Cohn shows that, in 299 eyes under atropine, no case of absolute emmetropia was detected. Hausen found but 26 emmetropic eyes in 1610, and Dürr but 30 in 414. A. Randall states, in his article on "The Refraction of the Human Eye, a Critical Study of the Examinations of the Refraction, especially among School-children" (*Am. Jour. of the Med. Sci.*, July, 1885), that only 7 $\frac{35}{100}$ per cent. of 1834 eyes of infants and school-children were found to be emmetropic.

to be a comparatively rare one when Graefe's test was employed with the test-object (preferably a candle-flame), at twenty feet from the eye. It is reasonable to infer, therefore, that the tests made by this observer were such as to require accommodative efforts (probably the line-and-dot test at fourteen inches). Such tests, if made under atropine, are certainly open to criticism and probable correction. Even if not made under atropine, this form of test is only of value in connection with the other.

The critical reviewer mentions a certain "Martin family" as a proof to his mind that a "neurotic taint" exists in choreic subjects. Now, the four choreics of this family were all hyperopic, while five who were not so had no chorea. This fact would seem to confirm Dr. Stevens' view. No one disputes the fact that a "neurotic predisposition" is present in most choreic subjects; but the view that eye-defect tends to create this tendency seems to be less generally accepted.

Finally, the paper here referred to notes a failure to relieve the chorea by the use of glasses, in a few cases where the patients were able to purchase them. If other serious defects existed besides the hyperopia (to the extent shown in the examinations reported by this observer), this is not to be wondered at. Hyperopic glasses will not relieve "insufficiency of the interni" (frequently noted by this observer in his choreic subjects); and the latter is certainly a well-accepted cause of reflex disturbance when it exists, as well as the latent hyperopia that was alone corrected.

In preparing this section, I have looked carefully over the records of all cases of chorea which I have personally tested for anomalies of the visual apparatus. I have not found a single case where either "manifest or "latent" hyperopia did not exist. I do not mean to assert that this statement proves anything—but it certainly seems a very strange coincidence, if such it is.

Respecting the "neurotic taint" to which this reviewer attributes the origin of chorea, I would respectfully refer my hearers to a study of this question and its dependency upon anomalies of the visual apparatus in a paper previously quoted from,* and also to tables of a similar purport published (since that article was read at the International Medical Congress) by G. T. Stevens, in his work an "Functional Nervous Diseases."†

Every patient whom you examine for defective equilibrium in the eye-muscles instinctively strives (not by mere volition) to get binocular vision, under the nearest approach to physiological conditions of which he is capable.

We are forced to admit, therefore, that what we detect in any patient and record as an error is in reality only what the *patient cannot*

* *Med. Register*, November 19, 1877. † D. Appleton & Co., N. Y., 1887.

conceal, not necessarily all the defect in the muscles that actually exists.

Because a patient can momentarily perform a feat of eye-balance which approaches the normal state, by the aid of his reserve power, it is by no means proved that the eyes are habitually in equilibrium. An over-taxed muscle may, and often does, become preternaturally shortened or "contractured," so long as its utmost exertions are habitually demanded. Why, then, may this condition not exist in the eye-muscles, when the antagonistic forces are unequally balanced? If it may do so, does this view not tend to shed some light upon the fact that patients often show a higher degree of "insufficiency" after a tolerably free division of the stronger tendon than before the operation? May not a contractured muscle relax when relieved of the irritation caused by the over-taxed condition of that muscle? May not the development of "latent" insufficiency be attributed (in part at least) to the relaxation of a muscle in the orbit, which has been thrown into a state of abnormal spasm by its efforts to overcome an antagonistic force disproportionate to its inherent strength? I propound these questions because many facts have been observed by me after partial tenotomies upon the eye-muscles, which seem to me to add confirmatory evidence in support of this view. If there exists in any case a tendency on the part of the visual axes to deviate from their normal condition of parallelism when the eyes are directed at an object twenty feet or more from the eye, may it not indicate that an inherent defect (probably congenital) exists in the weaker muscles, either in respect to their actual development or their contractile power? If this view is admitted, why may not such a muscle, by endeavoring to antagonize a stronger muscle, become contractured in consequence of the development of the state of excessive nervous irritability?

Clinically, as I have remarked before, we are forced to recognize two classes of choreic patients,—those who get well within a short time (usually in less than four months) by the aid of tonics, good food, etc., and possibly without any medication; and those of a chronic type, in whom the choreic manifestations persist in spite of every form of medication.

There is no nervous disease known which has apparently been cured by so many different and often antagonistic lines of treatment. Some get well under iron, some under arsenic, some by the aid of good diet and good air. All the remedies which have been extolled as curative agents would, if compiled, exceed even those suggested for the relief of epilepsy; the difference between the two diseases being that one generally gets well and the other seldom if ever does, no matter what drug is used.

Now, it may pertinently be said in this connection that the cure of a typical chronic case of chorea without the use of drugs is a fact worthy

of record. If it can be shown that correction of a refractive error in the eye, or the relief of an existing "insufficiency" of some eye-muscle by partial tenotomy, has cured most obstinate cases of chronic chorea where medication and all other lines of treatment have proved of no benefit, the value of the method must be recognized.

I have seen choreic symptoms disappear in several instances within a week or two when an existing hyperopia has been relieved by convex glasses, with the frames well fitted to the child, so that each pupil is opposite the centre of the glass. It is not enough to tie a pair of glasses with frames made for an adult onto a child's head, and expect that comfort to the child will follow such a procedure. Neither is it right to expect that glasses are all that is demanded, when a child has esophoria, exophoria, or hyperphoria, in addition to a hyperopia or hyperopic astigmatism or refractive errors of the myopic type. The case reported on page 499 was one of the worst that I have ever encountered, and yet a practical recovery ensued when the muscles of the eye were properly adjusted. The photographs of some cases of chronic chorea published by Dr. Stevens illustrate more forcibly than words the results of such treatment when skillfully employed.

Finally, certain precautions are to be exercised in reference to the child by its parents and associates. Study should be interdicted, plenty of good food and fresh air should be provided, and encouragement and praise should be freely bestowed as aids to the child in its attempts to conquer the choreic habit.

Anything which disturbs and annoys the patient does harm,—such, for example, as mimicry, confinement to the house, deprivation from reasonable pleasures, etc.

Arsenic is a valuable remedy, in a large proportion of cases. Fowler's solution may be given to a child after eating, beginning with doses of three drops three times a day, and gradually increasing the amount by the addition of one drop each day until the patient takes ten drops after each meal, provided that nausea, œdema of the eyelids, or other toxical effects of the drug do not appear. I do not believe that arsenic should ever be pushed to the poisoning point, in spite of views advanced to the contrary. Iron, cod-liver oil, and quinine may be employed in a combination with the arsenic, if the condition of the patient is anæmic, or if good results do not follow the use of arsenic alone.

The judicious use of chloral, combined with digitalis, may be of great benefit in cases where persistent insomnia exists.

Ice-bags or ether spray to the spine, static electricity administered by the insulation or spark methods, and general galvanization are worthy of a trial in refractory cases.

A few months ago, I was asked by Dr. Stevens to meet a patient then under his care, whose subsequent recovery possesses great clinical interest in this connection. I give the details of this case as the patient stated them to me at our first meeting:—

Mr. C., minister of the gospel. Mother died of phthisis. No nervous diseases had existed among his direct ancestors, or in remote branches of his family.

About twelve years before this interview, his family had noticed frequent facial contortions which he was unable to control. A trip to Europe, and parish labors in a district where he spent most of his time in a carriage and wrote but little in his study, prevented its increase for about four years. He then became the pastor of a church and began active labor in his study. The facial contortions grew rapidly more aggravated in character. Every feature would become horribly distorted; the eyes would close, the forehead become terribly wrinkled, and the nose and mouth would assume attitudes which no one could possibly imitate by volition, and which it is difficult to describe. The hour of retiring was particularly dreaded, because the facial spasms would become terribly persistent and severe as soon as the eyes were closed and a recumbent posture was assumed. The facial contortions were always least severe in the morning, and grew more severe as the day progressed. No medicinal treatment had ever benefited the patient.

An examination showed hyperopia of a high degree, esophoria of 6°, and hyperphoria of right eye of 3°. He had been wearing *prisms with the base inward*, as the suggestion of an oculist.

A partial tenotomy was first performed to correct the hyperphoria. The facial spasms *ceased within an hour;* and no *sign of chorea was observed for two entire days.*

On the third day a very slight twitching about the mouth developed. A partial tenotomy of the internal rectus of the left eye was then performed. This completely corrected the esophoria.

Subsequent to the second operation, the patient had few, if any, choreic movements. He stated to me that "unless excited his face remained absolutely quiet," and that for the first time "he had that day been able to attend a meeting of ministers and look them in the face without facial spasms while discussing church matters." During his recital of his various symptoms, etc., to me, his face only showed one very slight convulsive movement. Thus, in less than one week, were the convulsive spasms of his face almost completely arrested by correcting a hyperopia and two muscular defects associated with the eye.

Such a case is rarely encountered. The patient was an adult. The duration had exceeded twelve years. The spasmodic movements were terribly severe. All medication had failed even to ameliorate them. They became greatly aggravated as soon as the patient was compelled to use his eyes in study or writing. He could not even "look out of a car window" without being thrown into a most distressing state; yet, in spite of all these unfavorable facts, he was apparently perfectly well when I last conversed with him.

HYSTERIA.

A form of functional disturbance of the brain, spinal cord, or the sympathetic nervous system, in which the patient gives evidence of "an abnormal susceptibility to external impressions, and a deficient

power of the will to restrain its manifestations," is generally termed "hysteria."

It is encountered chiefly in women. In the male, cases are somewhat rare.* The allied conditions known as "catalepsy" and "hystero-epilepsy" will be discussed also under this head.

Etiology.—A very large proportion of cases of this type develop symptoms of nervous derangement at the age of puberty (twelfth to twentieth year). Girls reared in luxury and idleness, especially in cities where excitement and dissipation are cultivated, suffer more than those who have to labor and those who enjoy country life. Cases of genuine hysteria are sometimes encountered in children under twelve years of age. It is very uncommon for hysteria to develop after the age of forty.

Psychical influences frequently seem to excite this condition, especially when the patient is predisposed to hysteria. Among such influences, fear, jealousy, love, disappointment, anxiety, care, remorse, etc., are more liable to cause hysteria than pleasurable emotions or states of mind.

The *sexual organs* are liable to be found deranged in many hysterical females. Displacements of the womb, ulceration of the cervix, diseases of the ovaries or vagina, scanty menstruation, or irregularity of the periods, leucorrhœa, excessive irritability of the vulva or clitoris, etc., may be often detected on examination. Self-pollution, or the frequent occurrence of erotic dreams, in females is not uncommonly met with in hysterieal subjects.

Heredity plays an important *rôle* in hysteria. A phthisical predisposition is extremely common. Again, hysteria may be directly transmitted, or it may alternate with epilepsy, insanity, sick-headaches, neuralgias, chorea, and allied conditions.

In this connection, my remarks concerning eye-defects and muscular insufficiencies in the orbit when discussing epilepsy and chorea might be repeated here. Hysterical subjects are almost invariably thus affected, and an examination of the eyes and the eye-muscles will generally shed light upon this disease, as well as upon its allied diseases. The reader is referred to the introductory pages of this section, and to the views advanced respecting the causes of epilepsy and chorea.

Finally, *imitation* has been known to cause hysterical attacks in schools and convents. In such cases the afflicted probably suffered from one or several of the predisposing causes mentioned, the attacks being actually developed by the mental impression made by witnessing an attack in another.

* According to Briguet about fifty males were attacked to nine hundred and fifty females.

Morbid Anatomy.—The existence of any organic lesion of the nerve-centres may be considered extremely doubtful in any case of hysteria, unless some other symptoms of a strongly diagnostic character are detected. I have on my case-book the records of one case of hysteria where a calcified state of the falx cerebri and the adjacent dura was found after death. A few cases have been reported where other organic changes have been shown to have existed, and to have unquestionably caused the hysterical phenomena. These cases must be regarded, however, as exceptional.

All authorities seem to be in accord in the statement that no pathology of hysteria has, as yet, been recognized as proven.

Symptoms.—These are so varied as to require classification. The following table will bring the more common symptoms of hysteria prominently before the mind of the reader:—

HYSTERICAL PHENOMENA.

A. Disturbances of the Sensory Apparatus.	1. Hyperæsthesia. (Rarely of the whole body.) It may affect...	One entire side. Occipital region. The back, thorax, or abdominal walls. Individual joints. The muscles. The special senses.
	2. Anæsthesia.	Generally of left side. May affect *touch*, *temperature sensations* and pain (*analgesia*).* Sensibility may remain intact in spots scattered over the anæsthetic area. One or more of the *special senses* may be lost or impaired. *Joints* and even *bones* may be deprived of sensation. *Muscles* may lose their electro-contractility.
	3. Loss of Muscular Sense.	Hysterical patients sometimes cannot tell if they move a limb, or, if it is moved, in what direction, unless they observe it with their eyes.
	4. Neuralgias (of various nerves).	*Coccydynia* (at tip of spine). *Sciatica* (in lower limb). *Intercostal* (most often near seventh rib). *Lumbar* (in small of back). *Brachialgia* (arm). *Omalgia* (neck). *Cephalgia* (headache). This is a very common symptom. *Hemicrania* (confined to one side of head). *Cardialgia* (pain over heart). *Ovarialgia* (pain over ovaries). This is a valuable diagnostic sign. *Rachialgia* (spinal pain).
B. Disturbances of the Motor Apparatus.	1. Clonic Hysterical Spasms.	Facial spasm. Of larynx, pharynx, and œsophagus (the so-called "*globus hystericus*"). Paroxysms of uncontrollable laughter. Paroxysms of uncontrollable weeping or convulsive cries (barking, howling, etc.). Paroxysms of uncontrollable hiccough (due to diaphragmatic spasm). Hysterical asthma (due to bronchial spasm). Hysterical yawning (due to spasmodic action of inspiratory muscles). Hysterical cough (due to irritation of the laryngeal nerves).

*Anæsthesia of the larynx and pharynx is a very common symptom of hysteria. In many cases, the finger or a probang may be introduced into the larynx without exciting coughing or vomiting.

HYSTERICAL PHENOMENA (*continued*).

B. Disturbances of the Motor Apparatus (*continued*).

- **2. Tonic Hysterical Spasms.**
  1. Vaginismus (often preventing sexual intercourse).
  2. Spasm of bladder and rectum.
  3. Goose-flesh (due to erection of the papillæ of the derma).
  4. Hysterical contractures of the limbs (resulting in deformity—usually at the knee, wrist, fingers and toes).
  5. Torticollis or "wry-neck."
  6. Strabismus or "cross-eye." This may be permanent or transient.
- **3. General Convulsions (hysterical type).**
  - Constituting "hystero-epilepsy." These may be partial or general, and with or without a loss of consciousness).
- **4. Motor Paralysis (hysterical type).**
  1. Of *face*. May exist in combination with hemiplegia of the same side.
  2. Of *eye*. Ptosis and alternating strabismus are occasionally observed.
  3. Of *œsophagus* and *pharynx*.
  4. Of *larynx*. (Causing "hysterical aphonia.")
  5. Of *diaphragm*. (The voice is lost. The thorax contracts during expiration and the abdomen rises.)
  6. Of *bladder*. (Usually accompanies hysterical hemiplegia or paraplegia.)
  7. Of *rectum*. (Accompanied by constipation, tympanites and rectal anæsthesia.)
  8. *Hemiplegia* or *paresis*. (Usually developing after excitement or a general convulsion.)
  9. *Paraplegia* or *paresis*. (Usually associated with paraanæsthesia.)
  10. *Monoplegia* or *paresis*. (Generally affects arm or leg.)

C. Visceral Disturbances.

1. Abnormal respiration. (Usually increased in frequency.)
2. Abnormal heart's action. (Palpitation or anæmic murmur.)
3. Impaired digestive functions. (Capricious appetite, fasting, vomiting, belching, etc.)
4. Unnatural craving for food. (*Boulimia.*)
5. Irregular or scanty menstruation. It may be suppressed (*amenorrhœa*).
6. Vicarious menstruation by the lungs or the rectum.
7. Retention of urine. (Requiring the regular use of a catheter.)
8. Abolition of sexual excitement.
9. Increase of sexual excitement. (Nymphomania.)

D. Psychical Disturbances.

- ***Premonitory* manifestations**
  - Morbid desire for sympathy or for attracting attention.
  - Apathy to external surroundings.
  - Obstinacy to all influences exerted upon the patient.
  - Sudden transition from gayety to sadness, or *vice versa*.
- ***Acute* manifestations.**
  - Hallucinations.
  - Delirium.
  - Ecstasy.
  - Mania.
- ***Chronic* manifestations.**
  - Melancholia.
  - Ecstasy.
  - Somnambulism (usually followed by convulsions, if the patient is awakened while out of bed).
  - Nymphomania.
  - Lethargy or stupor. (Has been known to last for months without any interruption.)
  - Trance. (It may simulate death very closely, in some cases.)

E. Vaso-motor Disorders.

- Elevation of temperature. (Sometimes preceded by a chill.)
- Salivation. (Probably due to irritation of the central origin of the chorda tympani nerves.)
- Polyuria. (The urine being very light in color and deficient in salts.)
- Œdema. (Usually appearing suddenly without cause and disappearing as suddenly.)

After a perusal of this table, the thought may occur to the reader that it would have been easier to mention the symptoms which do not occur in hysteria than those that may be encountered. It is safe to say that no nervous disease (if such a term is applicable to hysteria) presents a greater variety of forms, or may more closely simulate the effects of organic lesions of the nerve-centres. The diagnosis of hysteria is

almost invariably made by the exclusion of more serious conditions which the symptoms exhibited by the patient might lead a physician to strongly suspect. The best diagnosticians are sometimes misled by hysterical subjects.

Diagnosis.—To the practiced eye, there are certain symptoms in almost every case of hysteria which materially aid in making a diagnosis; although it is difficult to state in a general way exactly what the particular points in a given case may be. Certain hints may be given, however, in this connection with advantage to the reader.

In the first place, the *history* of the case may aid you. If the patient has from childhood been very impressionable; if she has been subject to periods of unnatural excitement; if the existence of similar conditions in the family can be traced; if epilepsy, insanity, chorea, neurasthenia, severe and recurrent headaches, or neuralgias have existed in her relatives; if anæsthesia, hyperæsthesia, painful points, or a sense of compression in the region of the epigastrium persist after the suspected hysterical paroxysm has passed away, and if transient paralyses have appeared at any time, the diagnosis of hysteria is more than probable.

Again, whenever the paroxysm assumes the *convulsive type*, the irregular character of the fit, the length of its duration, the occurrence of hiccough or of laughing or weeping after the convulsions have subsided, the fact that the convulsions seldom occur at night or when removed from the possibility of sympathetic attention, and the passage of large quantities of pale, clear urine, deficient in salts, point to hysteria or hystero-epilepsy. Energetic pressure upon the ovaries may also modify hysterical convulsive seizures, while in epilepsy this test is negative in its results (Charcot). If the larynx or pharynx is anæsthetic, so that coughing or vomiting cannot be induced by introducing the finger or a probang after the attack, hysteria is almost positively indicated.

The so-called "*globus hystericus*," the sensation of a ball in the throat, the absence of any appreciable rise in temperature (when taken in the rectum), and the absence of albumen or casts in the urine point rather to hysterical attacks than to true epilepsy, uræmic convulsions, or organic lesions of the nerve-centres.

Hysterical paralyses may generally be distinguished from the paralyses of cerebral or spinal lesions by the gait (page 165), the history of its onset, the absence of tremor, the testing of the reflexes (page 174), the results of electrical tests (page 194), and the history of the case.

The diagnosis from *trismus* or *true tetanus* is easily made by the absence of a history of some injury received, the method of extension of the convulsions, the facial expression, the attitudes assumed, and the typical relaxation and termination of attacks of tetanus.

The *diagnostic table* given on page 498 will aid the reader in making a discrimination between hysteria and other diseases which it may closely resemble. As Hammond very aptly remarks, "careful watching, with thorough skepticism, will either result in detection, or in the patient's defeat from sheer weariness."

Let us now pass to the consideration of hystero-epilepsy, catalepsy, and ecstasy, which belong to the hysterical type of nervous affections. The prognosis and treatment of these conditions will be considered later.

HYSTERO-EPILEPSY.

This condition is characterized by peculiar combinations of the symptoms of hysteria and epilepsy.

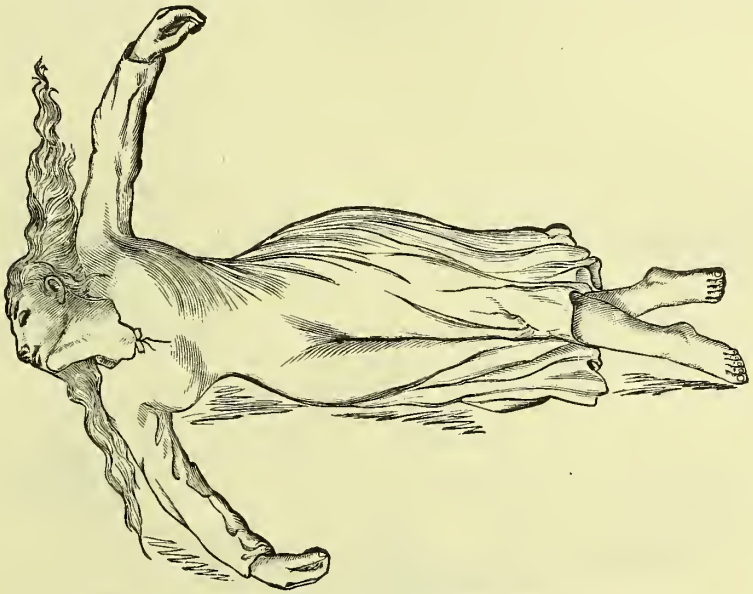


FIG. 121.—HYSTERO-EPILEPSY. (From a photograph. Case of C. K. Mills.)

These attacks are usually preceded by some of the prodromal manifestations of hysteria.

The various manifestations of this form of attack have been classified by Richer into four distinct periods, as follow:—

(1) The epileptoid period.

(2) The period characterized by contortions.

(3) The period of emotional attitudes.

(4) The period of delirium.

The symptoms of each of these states may be arranged in a tabular form, which admits of a contrasting of the chief peculiarities of each by the reader.

SYMPTOMS OF THE FOUR PERIODS OF HYSTERO-EPILEPSY.

The First or Epileptoid Period.

1. Premonitory symptoms.....
   - Tremor.
   - Pupils contracted.
   - Rapid winking of the lids.
   - Rapid respirations.
2. Convulsion, characterized by.....
   - Pupils dilated.
   - Face pale at first and congested later.
   - Loss of consciousness.
   - Rigidity.
   - Slow bending of body and twisting of head.
   - Distortion of the features.
   - Pronation of the hands.
   - Adduction and slow movements of the legs.
   - Inversion or eversion of the feet.
3. Stage of secondary rigidity (patient lies in a fixed attitude).
4. Stage of clonic convulsion.....
   - Partial or General
     - Unilateral.
     - Confined to one limb
     - Resembling an epileptic attack.
5. Stage of recovery.....
   - Stertorous breathing.
   - Frothing at mouth.
   - Stupor.

The Second or Contortion Period.

In this stage the following symptoms exist and the movements are very forcibly made.....
- Incomplete loss of consciousness.
- Extreme opisthotonos.
- Piercing shrieks.
- Forcible and rapid movements of the limbs (usually of flexion and extension).
- Striking of the body.
- Tearing of the clothing and the hair.
- Face not turgid.
- No foaming at the mouth.
- Duration, 5 to 10 minutes.

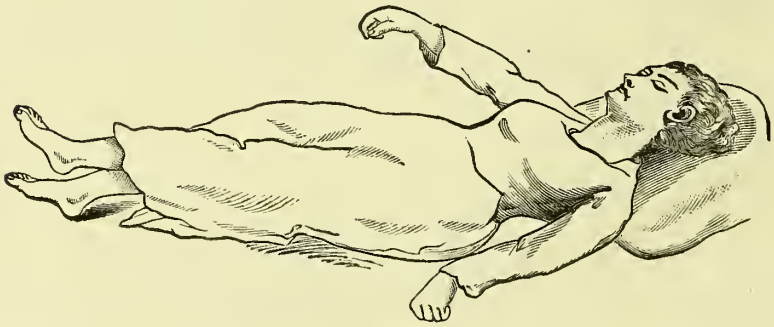


Fig. 122.—Another Attitude of same case.

The Third or Period of Hallucinations.
- Abolition of general sensibility to touch, pain, or temperature usually exists.
- The special senses may be in abeyance.
- Various forms of hallucination exist.
- The patient may answer questions unconsciously.
- Various expressions are uttered and certain gestures are made which indicate the form of hallucination that exists (usually that of sight).
- Eyes still anæsthetic.
- Pupils may be contracted or dilated.

The Fourth or Period of Delirium.
- The patient gradually passes into this stage.
- Pupils may be dilated.
- The patient wanders, laughs, weeps, or shows mental excitement in other ways.
- The patient frequently passes large quantities of urine.

The second and third periods described are of especial interest. The contortions observed in the second stage are often horrible to witness. The arms and legs may be placed in the most revolting of attitudes.

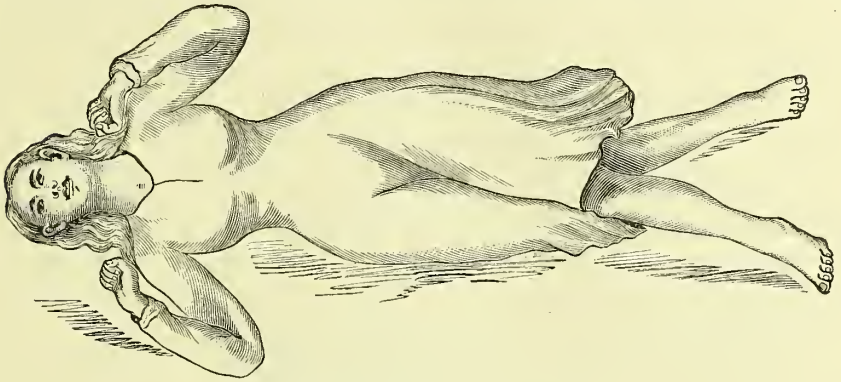


Fig 123.—Third Attitude of same case.

In the third stage, the expressions of the patient often indicate the greatest alarm. Hallucinations of sight are very common and the patients see horrible sights. To quote from Hammond, they "hurl invectives at imaginary persons,—Scoundrels! robbers! brigands! Fire, fire! Oh, the dogs, they bite me!"

Sometimes it becomes necessary to feed these subjects through a tube and to draw the urine at regular intervals; because the pharynx and the bladder are occasionally paralyzed after the attack.

Fig 124.—Convulsion of Hystero-Epilepsy. (From a photograph. Case of C. K. Mills.)

During the intervals which elapse between these attacks of hystero-epilepsy, many of the symptoms enumerated as hysterical develop; such, for example, as paralysis, anæsthesia, hyperæsthesia, and diminution of the special senses.

CATALEPSY.

Catalepsy can be classed as a condition closely allied to hysteria. It is a functional neurosis, with no recognized pathology. It is characterized by attacks of partial or complete loss of consciousness, which are accompanied by a peculiar rigidity of the muscles. During these attacks, the limbs *remain in any position in which they chance to be at the onset*, unless they are passively moved into some other position by outside influence or until the limb falls from exhaustion of the muscles.

Although the muscles appear tense and unyielding, a slight amount of force suffices to *cause them to yield and to assume any posture* which an investigator may desire. They *will then remain fixed until the attitude is again changed* in the same manner. This state of the muscular system has been termed "waxy flexibility."

These attacks usually begin suddenly, but they are frequently preceded by prodromal symptoms, as, for example, by yawning, eructations,

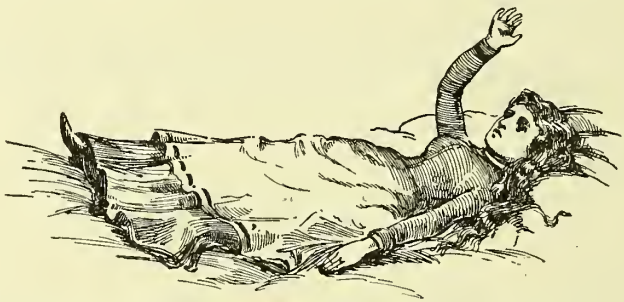


FIG. 125.—CASE OF CATALEPSY, EXHIBITING THE PROLONGED MAINTENANCE OF AN ARTIFICIALLY INDUCED ATTITUDE. (From an original sketch.)

a sense of pressure in the head, palpitation of the heart, vertigo, a change in the disposition, etc.

When the attack develops, the patient is suddenly rendered incapable of altering the position in which he or she may be at the moment. The eyes are either open or closed. The patient may occasionally understand questions and yet be unable to respond or move. The features are immobile, and the whole body remains as if suddenly petrified.

Sometimes one limb is affected at first, but the rigidity soon extends over the entire body. The respiration is often slower than normal. The heart's action is regular. The power of swallowing is preserved. The sensibility of the skin is greatly diminished or absolutely lost. The pupils are usually dilated and respond slowly to light. Occasionally, the temperature falls below the normal point and the skin is pale and cold to the touch. The reflex excitability of the muscles is abolished in some cases.

Either sex may be affected. I observed with great interest in 1872 an attack in the male, which came under my notice while a resident surgeon in Bellevue Hospital. The details of this case are given in full by Hammond, from notes furnished him by my friend, Dr. Early. The cataleptic state persisted in this case for several days. The temperature rose to 100° F.

The duration of cataleptic attacks varies from a few hours to several days. They generally subside with sighing and a desire for food. A tendency to recurrence of these attacks is often observed.

Among the reported causes of this condition the following may be mentioned: Pregnancy, mental excitement, grief, anxiety, mental disease, hysteria, chorea, fevers, narcosis from ether or chloroform, and anæmia.

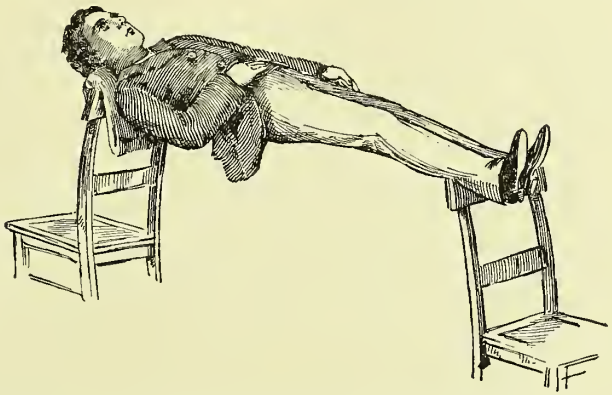


FIG. 126.—A CATALEPTIC PATIENT SUPPORTED BY HEAD AND FEET. (From a sketch made at the time.)

These patients can be easily nourished if food is made to pass the fauces. Death may be simulated during cataleptic attacks; but the heart's beat and respiration can be easily detected by a careful observer.

Catalepsy may be induced in "hypnotic" subjects by suggesting to their minds the state of rigidity of the muscles. Charcot has supported a patient in this state by resting the nape of the neck and the ankles upon the backs of two chairs placed about five feet apart. I witnessed a similar condition of muscular rigidity in a cataleptic during my connection with Bellevue Hospital in 1872, and also in another cataleptic subject during the winter of 1885. (See Fig. 126.)

TREATMENT OF THE HYSTERICAL STATES.

The management of hysteria, hystero-epilepsy, and catalepsy will now be considered. I would suggest, in the first place, that before any medication is begun a thorough search be made for eye-defects

which have been discussed in the introductory pages of this section, and also in Section II, and in relation to epilepsy and chorea. In case they be found, they should be corrected by glasses or partial tenotomies. My records do not show any cases of hystero-epilepsy and catalepsy in which such examinations have been scientifically made; hence, I cannot give any personal experiences in reference to the beneficial results of this treatment except in hysteria. Among the latter class, I have observed several very marked examples, where a close relationship between eye-strain and the attacks existed.

I could quote many cases from my case-book if I deemed it necessary. The records in all go to show that eye-defect existed, and that partial tenotomies gave marked relief in a very large proportion of the whole number operated upon by me.

The *examination of the eyes* of hysterical subjects tends, in my experience, to lead me to the conclusion that a defect in the eye or its muscles is too often disregarded or unrecognized. It is well known that heredity plays a very important part in hysterical cases, as well as in other functional diseases; hence, it is advisable to investigate this possible factor in any case early, and, if it exists, to remedy it without delay.

In *hysterical anæsthesia*, the employment of the secondary coil of a faradaic machine by means of the wire brush often acts in a magical way. Daily applications over the entire area of anæsthesia with as strong a current as the patient can comfortably bear will generally cure this symptom in a short time. The negative pole of a galvanic battery may also be employed with decided benefit upon the area of anæsthesia.

Hysterical paralysis yields in my experience most quickly to heavy indirect static sparks. Strychnia and phosphorus are valuable aids in effecting a rapid recovery, in some cases. Persistent daily attempts to use the limbs should be urged upon the patient.

Hysterical contractures are often cured by static sparks and passive motion. The galvanic current applied through the positive pole to the affected muscles (with the cathode on some neutral point—see p. 186) often gives excellent results. I prefer "stabile" applications to "labile" in case the positive pole is indicated. Massage may be employed with marked benefit in certain forms of hysterical contracture. It should be practiced daily by one well instructed in the art; and it is well in some cases to use a faradaic current for from three to five minutes daily in connection with massage.

Hysterical paroxysms, when particularly severe, are controlled and often entirely relieved by the inhalation of ether or chloroform. These inhalations may be repeated as often as the symptoms seem to warrant. It is generally advisable to push the administration of anæsthetics at first to a point where the patient is rendered insensible by them. This

rule of treatment applies with equal force to emotional paroxysms as well as to those where spasm is the prominent feature of the attack.

Hysterical vomiting is sometimes obstinate. Hydrocyanic acid, subcarbonate of bismuth, blisters over the epigastrium, strychnia, phosphorus, and cocaine have been used by me with great benefit in such instances. The valerianate of caffeine in three-grain doses, repeated in a half hour if necessary, has been highly recommended by Paret, Hammond, and others.

According to most authors, the so-called "*hysterical state*" may be controlled or eradicated, in some subjects, by long-continued medication. While I am inclined to believe that some form of reflex irritation exists in a very large proportion of hysterical subjects, and that its detection and removal constitutes the first duty of a physician, still I am not prepared to state that there may not be a certain proportion of this class of subjects that must be medicated before the hysterical tendencies are fully eradicated. I do not believe, however, in any medicinal specifics for hysteria.

Among the REMEDIES which have been suggested for the relief of these cases, the following may be cited:—

(1) The *monobromide of camphor* may be given either in an emulsion or pill in doses of from three to five grains every hour or two as the symptoms seem to indicate (Hammond). This drug seems to be particularly of service when the inhalation of ether or chloroform is contraindicated during emotional paroxysms or attacks of hysterical convulsions.

(2) The *antispasmodics*. Musk, valerian, asafœtida, and the various bromides are highly recommended by authors generally. Valerian is best administered in the form of the tincture, extract, or as the valerianate of zinc. The nauseous taste of the latter preparation is best overcome by giving it in capsules.

Castoreum is held in high favor by some authors. It is somewhat expensive, however, and is not always procurable. Ten drops of the tincture may be given on a lump of sugar.

The bromides of sodium, potassium, calcium, or zinc may be given in large doses until the full effects of the drug are obtained in cases where hyperæsthesia or mental disturbances are prominent manifestations of the hysterical state.

(3) The *mineral tonics*. Arsenic stands at the head of this list, in my opinion. It may be administered in pills of the arsenate of iron, or as Fowler's solution. It acts well when anæmia is markedly exhibited by the patient.

Various combinations of zinc, copper, silver and gold have been given to some cases with apparent benefit.

(4) The *narcotics*. This class of drugs has been quite extensively employed by some observers. Opium may be administered cautiously

by the hypodermic syringe or by the mouth or rectum. Belladonna must be administered with extreme caution; especially if its active principle (atropine) is employed. It sometimes acts well when hiccough exists, or when convulsive seizures are frequent.

In closing, I would mention certain SURGICAL PROCEDURES which have been suggested as a means of cure of hysteria:—

(1) Cauterization of the clitoris, according to Friedreich, often yields good results in hysteria.

(2) Removal of the ovaries is now frequently practiced in severe forms of hystero-epilepsy (Battey's operation).

(3) The removal of the clitoris has been reported as having led to good results.

(4) Partial tenotomies may be performed upon the ocular muscles, in case any abnormal tendency to deviation of the visual axes inward, outward, or upward can be detected in the patient. I am able to bear strong testimony to the efficacy of this step in several instances in which I have deemed it wise to operate.

(5) An application of the actual cautery may be made to the spine and the nape of the neck. This procedure, in my experience, has given very marked relief to several patients of the hysterical class. Such applications may be repeated as often as the symptoms seem to demand them. The heated platinum-tip should not be allowed to blister or burn the skin. It should simply be brushed lightly over the surface.

Before we leave the subject of hysteria and its allied conditions (hystero-epilepsy and catalepsy), it may be well to give some general directions respecting the general steps which should be taken in the management of such a case.

It is very essential, in the first place, that the doctor has the respect and full confidence of the patient. The impression made upon the mind of the patient by the personal characteristics of her medical attendant has often a great deal to do with the efficacy of the steps employed for the relief of the symptoms. Possibly this accounts for the fact that recoveries have occurred under lines of treatment which could have had little, if anything, to do with the benefits derived. We are all too apt to think that any given patient recovers in consequence of some medicinal agent that we may have administered, when perhaps the mind of the patient has simply been forcibly influenced.

In the second place, we must make such subjects believe that their symptoms are perfectly understood, that all the points of the case have been thoroughly noted and examined, and that there is strong reason to hope for a complete recovery. Ridicule or the implication that the symptoms are purely imaginary is apt to destroy the physician's influence over the patient.

Again, the use of the term "hysteria" is often construed by the patient or her friends as a slur upon the patient's integrity of character; hence it is well to avoid it in discussing the case outside of medical circles.

Patients of this class generally do better when removed from the influence of sympathizing friends, or surroundings that tend to recall the original exciting cause of the attacks. Pleasant and cheerful surroundings, travel, amusements of a non-exciting character, etc., are often of great advantage to such patients. A married woman will frequently improve faster when taken away from her husband; and it is often well to free her also from the care of her household by removing her from her home.

No effort should be spared to improve the general health of the patient. Tonics, good hygiene, regular exercise or massage, and even the so-called "rest treatment" (first suggested by Mitchell) may be required to establish this end. Some cases of hysteria are close to the border-lines of insanity, and must be treated with due regard to their condition.

Above all, do not fail to examine for all possible conditions which may exert (through reflex irritation) a delcterious influence upon the nervous centres of the patient. Every organ in the body should be examined before this question be decided in the negative.

Children of hysterical mothers should be brought up by a healthy wet-nurse, and given every possible chance to grow strong and robust.

Ice-bags or a pressure-pad may be placed over the ovaries (if decidedly hyperæsthetic) with benefit to some patients. The use of cold should not be employed for more than an hour at a time to the ovary. The actual cautery or blisters have been suggested as a substitute for the ice-bag, since both tend to contract the blood-vessels by influencing the vaso-motor system of nerves.

Static insulation (see subsequent section) often acts charmingly with hysterical patients, in my experience. The patient should be very highly charged with static electricity for twenty minutes daily.

NEURASTHENIA.

By the term *neurasthenia* we have been taught to include all manifestations of the condition commonly known as "*nervous exhaustion.*"

It may be manifested in a variety of ways. Its symptoms will depend upon the type which exists—cerebral exhaustion or spinal exhaustion—and also upon special idiosyncrasies of the patient.

Neurasthenia has been brought, in my opinion, too strongly into prominence as an independent condition, chiefly through the writings of Beard, Mitchell, Playfair, and Clark.

To my mind, neurasthenia, while not in reality a disease in itself, constitutes the basis of many of the functional diseases which have already been described in this section; hence, much that has been said in relation to the causation of epilepsy, chorea, hysteria, hystero-epilepsy and catalepsy might pertinently be repeated here.

Patients probably always develop the neurasthenic state, to a greater or less degree, prior to the appearance of certain symptoms which are characteristic of the special functional diseases already discussed.

When the condition of neurasthenia is not accompanied or followed by convulsive or emotional manifestations, it tends to manifest its presence by many other morbid phenomena. These have been classified by Beard, who, in various monographs which he issued upon this subject prior to his death, has minutely described the abnormal conditions most frequently encountered.

Etiology.—The more common evidences of functional nervous derangements which we so often encounter to-day (among which may be mentioned sleeplessness, muscular twitchings, nervous dyspepsia, sick-headache, hay-fever, morbid fears, sexual debility, melancholia, etc.) were uncommon, and are still so, in certain climates and among certain classes.

The reasons why functional nervous derangements are more common now than of old, and why the American race is particularly disposed to them, has been made the subject of much scientific thought and discussion. It may be well to refer to a few of the causes which tend to promote nervous debility. Practical suggestions may be afforded by so doing.

Dryness of the atmosphere is one of these factors.—In all cold climates the humidity of the atmosphere is less than in the warmer latitudes. This is because cold air condenses moisture, while warm air will carry a large amount of it without depositing it as rain. It is well known that dry climates predispose to nervous excitability by absorbing the natural fluids of the body. Since dry air is a poor conductor of electricity, it tends, moreover, to cause the body to become overcharged with electricity, and thus to render the nervous organization abnormally susceptible to any form of external or internal irritation. In very dry climates, the hair becomes stiff and brittle on account of a want of the natural moisture and oil. Sparks of electricity may be elicited under such circumstances from the hair by drawing a comb through it, and even from the clothing in some instances. Men and animals of all kinds become fretful and irritable when exposed for any length of time to dry, cold winds, so often encountered in the Western States. The vegetation is frequently shrivelled and parched by an abstraction of its moisture, and its vitality is quickly destroyed.

Extremes of heat and cold tend to cause nervous diseases.—In the southern climates, and in the small islands surrounded by salt water which are known as health resorts, marked extremes of temperature are uncommon; hence we find less susceptibility to nervous excitability in the inhabitants of these climates, when contrasted with those of the Northern States, in which bitter winters are followed by a high range of temperature during the summer months. The freezing blasts of winter compel the inhabitants of the Northern States to live in-doors, in homes filled with a dry and over-heated atmosphere. On the other hand, the heat of the summer months does not encourage out-of-door exercises and athletic sports as a popular pastime. Now, in England, for example, the climate is more equable and moist than in America. Athletic exercises can be indulged in there during all seasons of the year, and are of the greatest benefit to the inhabitants. While I am glad to see a growing love for similar sports on this side of the water, the peculiarities of our climate will never permit of the highest development and general popularity of the hunt, tennis, cricket, foot-ball, etc., with the masses. Thousands may attend exhibitions of this character, but those who participate must of necessity be few.

The heating of our houses is an innovation upon the past.—Our grandfathers brought up their families to rely on food and exercise for warmth. In the Northern States the log-fire on the hearth was the only way of keeping warm when in-doors. Now, any one who has had experience in that style of heating will accord with the statement that in extremely cold weather it is impossible to heat a room to a temperature above 60°. I have personally known water to freeze in a corner of a room in New England, which was illumined by the blaze of a roaring fire. The bed-rooms in olden times were cold, and feather-beds with an abundance of clothes were used to protect the body during sleep. The hostess of olden time was accustomed, moreover, to have the bed warmed for the guest immediately before his retiring.

In some parts of the country this method of heating is still employed; but, as a rule, the use of stoves, furnaces, and steam has superseded the hearth—much to the injury of the inhabitants. Most of us are now baked and dried all winter in a temperature which varies between 70° and 80°. We shiver when a slight draught enters the door or window casement. We pass into the air with our skin-circulation active, our pores open, and our bodies lacking the proper amount of fluid, since it has been abstracted by the heat of our houses. Is it to be wondered at, therefore, that an extreme of cold checks our perspiration, drives the blood from the surface to our lungs and digestive viscera, whose vessels become thus over-filled, and causes pneumonia, pleurisy,

liver and kidney diseases, and thousands of ills to the nervous organization whose development is not at once perceived?

Our habits of eating are often detrimental to health.—It was the custom of our ancestors of a century ago, if inhabitants of a northern climate, to eat salt pork three times a day and nearly every day in the year. I vividly recall, during a residence of four years in New England, the dish of salt pork that was invariably put upon the table. It had a constant claim to recognition as much as its companion, the castor. A prominent medical author humorously remarked, in an address delivered some years ago before a convention of doctors, that "Pork, like the Indian, flees before civilization." He says, furthermore, "The history of the rise and fall of pork as a food is itself instructive in relation to the first effects of civilization upon the nervous system. In all the great cities of the East and among the brain-working classes of our large cities everywhere, pork in all its varieties and preparations has taken a subordinate place among the meats of our tables, for the reason that the stomach of the brain-worker cannot digest it. This dethronement of pork has had, and is still having, a disastrous effect upon the American people; for, as yet, no article of food with a sufficient amount of fat has been generally substituted. Fat in our dietaries is one of the most imperative hygienic needs of our time, which has come to be felt, both instinctively and rationally, and which, on all hands, we are trying to meet by the use of cream, cod-liver oil, eggs, fish, and the fats of fresh meat."

It has been my custom for years to allow babies suffering from nervous debility to chew upon a well-cooked rind of salt pork. I believe that this form of food must of necessity always be the main article of animal food for the commmnity at large.

I would call attention, in the second place, to a habit which is common among brain-workers, viz., of eating irregularly and too rapidly, and the drinking of large quantities of fluid during their meals. It needs no argument to prove that both conduce to destroy or impair the powers of digestion. Who would think of placing a bucket of water by the side of a horse every time it was fed. If food is properly masticated, the saliva should suffice for all lubrication necessary to the act of swallowing.

Our systems of education may conduce toward ill health.—It is a well-recognized fact among scientific medical men that a defective construction of the eye is present from birth in quite a large percentage of children. As long as the child and its parents are ignorant of such a defect, or until the defect is remedied by glasses properly adjusted to the eye, serious harm may be done to the nervous system by the strain to which that important organ is constantly subjected. Most children in the larger cities are now compelled to spend from five to six hours each

a day, for ten months in the year, in a school-room; and to use their eyes as well as their intellect after school hours in preparation for the exercises of the ensuing day. It is not infrequent for medical men to encounter adults who have been rendered victims of countless nervous maladies by defects in vision which have never been corrected by the use of glasses.

A far-sighted child becomes easily fatigued when attempts at reading, writing, or study are made; hence he quickly develops tastes for out-of-door amusements in which he usually excels. Children of this type are often punished for a willful neglect of their studies, whenever impaired vision from a tired ciliary muscle renders it impossible for them to accomplish the allotted task. On the other hand, near-sighted children cannot indulge in out-of-door sports because their vision either prevents it entirely or makes them awkward in their attempts; hence they are generally fond of reading, and are too often regarded as precocious beyond their years. The steady increase in the functional disorders of the eye, which is proven by all of the carefully prepared statistics, may be attributed, in part at least, to the neglect of parents in having the eyes of children examined by a competent oculist before they are sent regularly to school.

One of the most frequent symptoms of nervous exhaustion in adults is a weak condition which manifests itself in a sense of pain and weariness whenever the eyes are used. This condition is known as "asthenopia."

Our hygienic surroundings should be conducive to health.—A very large proportion of the patients who are referred to me for advice respecting functional nervous maladies owe their troubles in part to defective hygiene. Most of the large cities are imperfectly sewered, and few dwellings are built with a proper regard for the requirements of health. The business offices in cities are often so dark as to be constantly damp and filled with the vapors arising from the consumption of illuminating gas. They are, as a rule, over-heated, ill-ventilated, deprived of sunlight, and often imperfectly protected against sewer gases. This statement is true also of all of our stores, and of some of our dwellings. It is one of the natural results of economy of space entailed by the high value of land.

While it is difficult to obviate this element of disease in cities, it is well to impress the minds of laymen with the fact that sunny bed-rooms, perfect ventilation, and pure air are of vital importance to health. Large cities are not the best places for people of moderate means to live in. Their business offices may be in the city, but their homes from choice should not be. A person may breathe impure air for a few hours each day with comparative impunity, if he can have its effects counteracted by pure country air during his hours of rest and the Sabbath.

Alcohol, tobacco, and other stimulants are often used to excess.—There is a deeply rooted opinion among some of the laity, and medical profession also, that tobacco and alcohol are the most prominent factors in producing the steady increase of nervous maladies which is generally recognized as existing. It is not my intention to discuss the question of temperance here from a social or moral aspect. What remarks I feel myself obliged to make on this point are of a purely medical character.

From information gathered by personal observation and careful inquiry and research, I am convinced that our ancestors were fully as indulgent in the consumption of tobacco and alcohol as are those of the present generation, and probably very much more so. Many of our grandfathers and grandmothers can be shown to have used both alcohol and tobacco to excess, without developing any of the functional nervous derangements of to-day. Ladies of the Southern States formerly indulged to excess in the habit of snuff-dipping. Men and women were habitually addicted to the use of snuff as a stimulant to the mucous membrane of the nose, during the epochs when it was fashionable. The open sideboard is certainly less common to-day in private residences than in the past. People do not now, as a rule, take their "night-cap" before retiring; but it was once the universal custom. Bulwer happily remarks that "it requires a very strong constitution to dissipate."

Now, it is very common for me to have nervous patients tell me that they have been obliged for years before they sought my advice to discontinue the use of alcohol and tobacco. Some of my patients are unable to drink tea or coffee; others are abnormally susceptible to stimulating narcotics, such as opium; while a few are unable to tolerate many of the drugs which they could previously use with impunity. On the other hand, I am satisfied that nervous exhaustion is a cause of confirmed inebriety in many instances.

It seems to me that we are forced by what has thus far been said, without further illustration, to the following conclusions: (1) that the tendency of the age is toward nervous excitability and debility; (2) that the brain-workers (in contradistinction to the muscle-workers) are more susceptible to external and internal disturbances than in past generations; (3) that many of the factors enumerated can be justly included among the elements which have produced this result; (4) that the American race is particularly prone to nervous derangements; and (5) that we are ourselves partly responsible for the modifications which have taken place in the constitutional vigor of man as civilization has progressed.

Before I pass to the consideration of the more prominent symptoms of functional nervous derangements, I may state that the *premature decay of the teeth* has been brought forward by Beard as one of the most

striking evidences of the steady increase of the constitutional impairment which has followed our present methods of living. It is common to hear dentists account for this fact on various grounds, among which may be mentioned the use of acids, the eating of sweet things to excess, a lack of scrupulous cleanliness, and the elimination of such foods as require thorough mastication. While I do not deny that there may be a justification for these views, still I would draw the attention of my readers to the fact that the teeth of the negro race, of the Indian, and of all semi-barbarian tribes are proverbially perfect. This is the case in spite of the circumstance that they are extremely fond of sweets. It is also true that they seldom if ever clean their teeth, and that they never suffer from cavities except in old age. Neither are the teeth of these races nor of animals irregular; yet how common are such deformities among the descendants of intellectual and refined ancestors! We should remember in this connection that the nutrition of all parts of our frame is controlled by the nerves. Is it not rational, therefore, to regard imperfect bone nutrition and development as a result, in some instances at least, of an impairment of the nervous functions?

Morbid Anatomy.—In the early stages no pathological lesions can be detected in many neurasthenic subjects. Later on, sclerosis may possibly be developed in exceptional instances, and many other varieties of structural change. Doubtless, these abnormal conditions are either induced or hastened by a state of nervous debility; but they cannot be said to be more than mere coincidences.

Symptoms.—Neurasthenia may affect the cells of the brain or of the spinal cord separately. Hence we are forced to clinically recognize two types of neurasthenia,—the cerebral and spinal.

Cerebral neurasthenia (*brain exhaustion*) may be indicated, according to Beard, by one or more of the following symptoms: Tenderness of the scalp; pains in the head; fleeting neuralgias; sleeplessness; vertigo; a tenderness and pallor of the gums; abnormal sensitiveness of the teeth; blanching of the hair; flushings of the face; dilatation of the pupils; idiosyncrasies in regard to food and external irritation; mental depression and melancholia; defects in memory; a morbid craving for alcohol; a decrease in intellectual capacity; a buzzing or ringing in the ears; specks before the vision; abnormal and imaginary impressions of taste or smell; morbid fears of various kinds; sick-headache; dryness of the skin and the mucous surfaces; weakness of the muscles; numbness in the limbs; thickness of speech; and mental excitability, irascibility or loss of emotional control.

These symptoms, in many cases, are but the manifestations of weakness. The electric batteries of the brain (the minute organs known as the "brain-cells") are feeble or uncertain in their action. They are

incapable of performing the offices for which they were created. They are not diseased (in a medical sense), but they are weak and liable to become so sooner or later. I have known sufferers of this type to be precipitated into a condition approaching incurability by mental alarm; excited, in some instances, by an opinion of an unfavorable kind made by physicians respecting a prospect of recovery. Again, it is well known that insanity may arise as a consequence of the loss of sleep often experienced by these subjects, and by "brooding over their symptoms" whose significance they fail to properly understand. I recall several cases where a patient was with difficulty convinced that some special type of malady was not about to attack him, because in reading a medical work his attention had been called to the significance of some special symptom, which he was sure he had personally experienced. If medical students who possess vivid imaginations can become (as they often do) victims to imaginary diseases whose symptoms they have been studying, is it to be wondered at that the weak and highly-organized sufferers from neurasthenia are especially prone to become impressed by this form of delusion?

Spinal neurasthenia (*spinal exhaustion*) signifies an exhausted state of the cells which help to form the spinal cord. The cord itself, although only about the size of an ordinary lead-pencil, is composed of millions of nerve-cells and distinct bundles of nerves. Some of these nerves pass through it to reach the brain above, while others become united to the spinal cells and pass no further. The cells of both the brain and spinal cord are practically electric batteries; and the nerve fibres are the wires by which they are connected with the different organs of the body, the muscles, skin, joints, and viscera. This wonderfully constructed organ is under the control of the brain; but is capable of exerting, under certain circumstances, a control over all acts, which are termed "reflex acts" because they are to a greater or less extent independent of the will.

Now, when the cells of the spinal cord become exhausted, the symptoms produced differ markedly from those already mentioned as indicative of brain-exhaustion. Among its chief manifestations, may be mentioned the following: A general tenderness of the skin to touch or pressure; tenderness along the spine or over certain limited portions of the spine; irritability of the breasts, ovaries, and the womb in females; fleeting pains of a neuralgic type in various parts of the body; an extremely rapid or slow pulse, which fluctuates widely during periods of excitement or fatigue; attacks of palpitation of the heart; dryness of the skin, or in many cases the reverse; excessive perspiration of the hands and feet; sudden startings on going to sleep; muscular twitchings in one muscle or a group of muscles; chilliness and creeping sensations along the spine; numbness or abnormal sensations of heat in the skin

of the body or limbs; itching of the skin; eruptions upon the skin, chiefly of the type of eczema; frequent gaping, yawning or stretching; frequent seminal emissions; weakness of the bladder and rectum; and disturbances of the digestive functions.

The distinction between cerebral and spinal neurasthenia, which has been stated by many observers to exist, cannot be made in each and every case, because various combinations of the symptoms of the two are often encountered in the same individual. A prominent author upon this type of diseases very aptly compares the nervous system of man to certain mountainous regions,—since it causes so many echoes and reverberations. He says, "An irritation at one point may be transferred to any other point, following the paths of least resistance, and making itself felt in those parts that are least able to resist molecular disturbances. Thus, for example, seminal emissions and spermatorrhœa, when they arise through abuse or through spinal-cord disease, almost uniformly react on the brain,—robbing the sufferer of courage and manliness, exciting various phases of morbid fear (of which I shall speak), with aversion of the eyes and countenance."

I have known a decayed tooth to cause persistent earache, and in one case to cause the corresponding eyebrow to become white. In male children, a tight foreskin not infrequently creates sufficient irritation of the sexual organs to induce spasms or paralysis of the lower limbs by an indirect effect upon the spinal cord. I have frequently cured patients who have come to me for relief from persistent and excruciating attacks of neuralgia, by a correction of some defect in their eyes. The extraction of a tooth has frequently, in my experience, relieved facial and orbital neuralgia.

It may be well to consider a few of the more prominent manifestations of nervous exhaustion separately. Among these, sleeplessness, a defect in vision known as asthenopia, sexual weakness, headache, an unnatural dryness of the skin and mucous surfaces or profuse sweating of the hands, and morbid fears or melancholia, deserve special mention.

Insomnia.—Sleeplessness may assume different forms. Some of those afflicted have difficulty in getting asleep; some awake after a few hours of slumber and remain so until daylight; a few find themselves overpowered with a desire for sleep during their working hours, when their business will not admit of it, and at night cannot obtain sleep except under narcotics. I have had patients who have told me that they spent most of their nights for years in writing to friends, riding in the horse-cars, or walking the streets for amusement because they could not sleep. It is safe to assert that persistent insomnia, extending over a period of weeks or months, indicates in most cases a persistent cause of neurasthenia or organic disease of some kind.

In patients who have passed the age of fifty, or in younger persons who have indulged to excess in alcohol, it is often due to a type of kidney disease, to detect which repeated examinations of the urine are required. This form of trouble is known as the "granular" or "contracted kidney;" and insomnia, frequently combined with headache, is one of its most prominent symptoms. Obstinate sleeplessness is the cause of many a suicide, too often the starting point of the opium and chloral habit, and surely the destroyer. I would caution my readers against allowing this symptom to remain uncontrolled in a patient for any length of time; to avoid the use of all forms of narcotics as long as possible; and to keep the patient from acquiring a habit of using them without medical authority. The chains of intemperance are but silken threads when compared to those of the opium or chloral habit.

Asthenopia.—This type of defective vision cannot be relieved by ordinary glasses; nor does it respond quickly to the customary suggestions of gymnastics, horseback-riding, etc. It is due, as a rule, to a defective equilibrium in the muscles which control the movements of the eyeballs, and it manifests itself chiefly as a sense of extreme weariness when the eyes are steadily employed for short periods of time. It is an indication of neurasthenia, and is of great diagnostic value. In many cases it becomes necessary to partially divide the tendons of the stronger muscles of the eye, in order to relieve the weaker ones of a strain. I have seen patients who could not sew for five minutes at a time from this cause, and others who would be made sick by attending a theatre, picture gallery, or other places of amusement. The reader is referred to preceding pages, which deal with this subject at greater length.

Headache.—Many attacks of this character are undoubtedly to be attributed to imprudences in eating, exposure, or fatigue. But I believe that most of those who are periodically afflicted in this way owe their suffering to a lack of tone in the muscular coat of the blood-vessels of the brain, consequent upon some of the causes of neurasthenia mentioned. I have seen a large number of instances where the *eyes were the cause of such headaches*, and where the adaptation of glasses has brought immediate relief. The medical profession are rapidly becoming enlightened upon this fruitful cause of pain. It is well also to examine the urine when persistent or periodical headache occurs, as it may be a symptom of kidney-disease. Some neurologists believe that the so-called "sick-headaches" are to be regarded as but a modified form of that condition which produces epilepsy. This condition will be considered separately in subsequent pages.

Dryness or Unnatural Moisture of the Skin.—Some nervous patients suffer from an unnatural dryness of the skin, the throat, and the nose. They are also liable to experience dyspeptic symptoms at the same time,

which are probably due to similar changes in the lining of the stomach. This dryness may be accompanied also by an itching of the affected parts or an attack of eczema. A burning sensation is sometimes produced. I was once consulted by a patient who had been in the habit of encasing himself in flannel and putting on flannel stockings before he retired for years, in order to overcome a sense of burning in the skin which followed the contact of cotton or linen with any part of his body. I recall a case where the feet were once frost-bitten, and the patient has never been able since to walk upon a carpeted floor on account of a burning sensation which immediately follows. He takes off his shoes as the last step before retiring.

On the other hand, many patients afflicted with neurasthenia suffer from a profuse sweating of the palms of the hands. This is accompanied in some instances by a flushing and redness of the face, neck, and ears. The nails may become unnaturally soft or brittle.

Morbid Fears.—This peculiar manifestation of nervous exhaustion may assume one of several types. Attempts at classification of these morbid fears have been made by some authors, such as fear of lightning, of places, man and society, solitude, accident, etc., and special names have been applied by them to each of these types. Fears of this kind may be present without any other manifestation of mental impairment. They are usually uncontrollable, in spite of the fact that the patient may exhibit a knowledge that they are groundless and absurd. They seem to take full possession of a being, and to cause mental torture of an extreme kind.

Finally, melancholia is not an infrequent symptom of neurasthenia. It may be accompanied by paroxysms of laughing, weeping, and hysterical phenomena.

Prognosis.—I have never observed a case of neurasthenia where death has occurred, save through some intercurrent disease; and I doubt if such an occurrence has ever been noted by a competent observer. I have, however, seen organic disease develop in the brain and spinal cord (apparently as a sequel to the neurasthenic state) in several instances. Melancholia, delusions of various kinds, and even mania have been known to develop in subjects who had previously suffered from symptoms of nervous exhaustion. I am not inclined to believe that these cases are extremely uncommon. Quite a large proportion of insane subjects give a history which leads to a different conclusion than that given by most authors.

A very large proportion of neurasthenic cases drag out a miserable existence for years; become hopeless invalids; and eventually die of some intercurrent disease. It is a daily experience with me to encounter both males and females who are totally unfitted for the active duties of

life from neurasthenia. To many of them the possibility of death would be robbed of its terrors, because life has ceased to be a source of comfort or of usefulness.

Treatment.—In this class of cases, my experience has convinced me that "eye-strain" constitutes one of the most important factors in the causation of the symptoms, and that the detection and relief of the defect which exists in any individual case is of the greatest importance. I could quote case after case, if space would permit, to prove this assertion. One of the most remarkable instances of this kind which ever came under my personal observation may be worthy of record here:—

Case I. *Cerebral Neurasthenia, Gastralgia, Persistent Tremor, etc.*—Mrs. ——, aged forty-three. Her father and mother died of phthisis, as well as several blood relatives. Has two children; was "very nervous" as a child. For the past sixteen years has been more or less of an invalid. For some years the patient has not been able to spend her evenings with her family or to see company. She reads without fatigue, has suffered but little with headaches, and has never had marked asthenopic symptoms. She had suffered greatly for some years with recurring attacks of gastralgia, a loss of emotional control, severe palpitation of the heart, and an inability to endure physical exertion or any form of excitement. Has always been regular in menstruation.

When I first saw the patient, she trembled violently, had moist palms, and could not without great effort control her emotions. Her eyes were constantly averted while in conversation, being generally directed toward the floor. Her voice was feeble and tremulous, and at times scarcely audible. Under any excitement, she would experience a choking sensation. During one interview in my office, she was so attacked, and suffered like a person in the height of a paroxysm of asthma.

An examination of her eyes under atropine showed:—

Emmetropia.
Esophoria of a high degree (6°).
Power of abduction 4°; of adduction, 28°.

Daily applications of static insulation gave her but little relief; although they were persisted in for some weeks. Prismatic glasses annoyed her and yielded negative results. Her tremor and emotional excitability persisted, and her condition remained practically unchanged. Finally, she consented to allow me to perform partial tenotomy upon her internal recti muscles. Within twenty-four hours, *her trembling ceased* and has not returned. In less than two weeks she was able to attend places of amusement, entertain her friends and perform all the duties of life as well as when a girl. She has remained absolutely free from attacks of gastralgia and cardiac palpitation since the last operation (nearly two years ago), and looks at least ten years younger than when I first saw her.

This case illustrates admirably the following points: (1) that no asthenopic symptoms had ever existed, in spite of the eye-defect; (2) that prisms failed to relieve the patient; and (3) that tenotomy cured her of tremor, gastralgia, and cardiac palpitation, besides a host of other neurasthenic symptoms. It is needless to add that the patient had tried medicinal agents without number during the many years that she had been an invalid.

Another equally striking case of cerebral neurasthenia is illustrated in the following history:—

Case II. *Cerebral Neurasthenia, Constant Pain in the Head of Five Years' Duration, Asthenopia, etc.*—Female, unmarried, aged twenty-one years.

Family History.—Maternal aunt and five paternal relatives died of phthisis; two cousins had chronic chorea.

Eye-defects.—Patient had hyperopia (latent) of 1.25 D. and exophoria (manifest) of 2°. A latent hyperphoria of 2° was subsequently discovered.

This young lady was brought into my office by two assistants, who were obliged to carry her from the carriage. For several years she had been carried daily from her room to the library of her father's house, and, after reclining in a chair for a few hours, she would be again carried to her bed-room. She could manage with difficulty to walk slowly across a room. She had not been able to write, read, sew, or see her most intimate friends for five years on account of a constant pain in her head, which was rendered intolerable by any use of the eyes or excitement. Her symptoms began while at boarding-school, from which she was removed to her home in a recumbent posture and by easy stages.

I used static electricity upon this patient for some weeks with a slight improvement in her power of walking, but no relief to her head. I then persuaded her to consent to a relief (by partial tenotomies) of her abnormal eye-tension. Tenotomies were then performed upon her left superior rectus and both externi within the space of two weeks. From that date her improvement was very rapid. She was sent home a few weeks later practically cured.

A letter from her physician, lately received by me, says:—

"Your patient is the wonder of this region. She rivals the 'Jersey Lily' in her feats of walking."

Before this patient was sent home she ascended and descended five flights of stairs daily, and averaged over a mile's walk each day without a companion to assist her.

In case the eye be found to be entirely free from abnormalities of refraction or accommodation, and the ocular muscles to be normal, I would advise rest as a valuable step toward a cure. The plan first suggested by Dr. S. Weir Mitchell (now generally known as the "rest-cure") has proven of great value in some of my cases. It consists of perfect isolation, a prolonged confinement to bed, and a total cessation of bodily or mental pursuits, combined with regular and systematic feeding, massage, and electrical applications. It is especially adapted for females; but, in a modified form, is useful in male subjects.

With males, it is usually well to advise early a total withdrawal from business for a time, and retirement to some quiet place where out-of-door sports can be indulged in. I seldom advise travel, because it involves too much excitement.

Sleeplessness may usually be controlled by a judicious use of the bromides, some preparations of valerian, chloral, hyoscyamus, conium, or opium. I seldom employ any drug, however, for the relief of this symptom until I have thoroughly cauterized the back of the neck (over

the point of entrance of the vertebral arteries) with the white-hot platinum tip. Such an application is often immediate in its effects.

Sweating of the hands and feet is generally relieved by atropine and friction of the parts after bathing them in very hot water.

Disturbances of the digestive functions are often greatly benefited by a prolonged use of hot-water drinking (see p. 248) and the administration of tonics. Stimulants may be given in some cases directly after eating. In occasional instances I am compelled to restrict patients for a time exclusively to a milk diet.

Static electricity, by the spark method, the static breeze, or simple insulation often acts as a great aid in the cure of these cases. General faradization or general galvanization are excellent methods of employing electricity upon these subjects, in case a static machine is not attainable, or as a substitute for static applications when the patient fails to respond to its influence.

MIGRAINE—SICK-HEADACHE—MEGRIM.

This distressing condition is characterized by paroxysmal attacks of pain in the head (usually unilateral, but not always so), accompanied by nausea, eructations of gas from the stomach, and vomiting. Between the paroxysms, the patient generally enjoys more or less perfect health and is free from pain.

There is perhaps no form of pain that is more commonly encountered by the physician than this; unless it be "neuralgia" in the common acceptation of that term. Sufferers from sick-headaches are to be found in every large city and country town, especially among the brain-workers in contra-distinction from the muscle-workers. No clime or atmospheric condition seems to confer immunity from the attacks to those who are congenitally predisposed to them.

I approach the discussion of this subject with more than ordinary interest, because for nearly thirty years I was personally a victim to the most severe form of these attacks. Within the past ten years, I have treated a very large number of these cases with the most satisfactory results; hence, my convictions are strong in reference to their causation and their treatment.

Etiology.—*Migraine runs in families.* Hereditary predisposition is perhaps more strongly marked in this affection than in most of the so-called functional nervous maladies. In all of this class such a predisposition is very marked, and particularly so in migraine.

Most authorities classify migraine under the "vaso-motor neuroses;" but, in the light of my own observations in this special field, I prefer to discuss it under the head of functional nervous diseases, because I believe that *reflex irritation of the oculo-neural type* lies at the bottom of the attacks.

It is quite remarkable how often the family history of this class of patients points to a *tubercular predisposition*. It is, in my experience, very rare to find typical attacks of migraine in a male or female who has not had some relatives die of phthisis.

For many years I have taught in my lectures that I had yet to meet a case of typical migraine in which an examination of the eyes or the eye-muscles would not show the existence of a marked error. All observations to date confirm me in this view. *Latent hyperopia* is an extremely common cause of reflex disturbance in these cases; and *esophoria* or *hyperphoria* (p. 143) are not infrequently found.

No one in the profession, as far as my information goes, has ever kept such careful records of the condition of the eye and its muscles in descendants of "consumptive" ancestry as has my friend, Dr. G. T. Stevens. His investigations in this particular direction have been noted carefully in several thousand patients. They go to confirm my own researches in the conclusion that a hyperopic eye exists in an extremely large percentage of these subjects, and that ocular insufficiency is also very common among them.

To avoid repetition, I would refer the reader to some pertinent remarks upon the generally accepted views respecting the causes of sick-headache which I have made in the second section of this work (p. 127).

The age varies at which patients experience their first attack. Generally they develop during childhood, provided the occupation of the child requires much eye-work, as in the case of educational pursuits. If they begin after the twenty-fifth year, I generally am led to suspect some intercurrent disease in addition to "eye-strain."

Symptoms.—Attacks of sick-headache are always paroxysmal and peculiarly "explosive" in character. They come without known cause, as do epileptic seizures; although all persons so afflicted, as well as their doctor, usually think that they know the particular exciting cause of each paroxysm. (See p. 128.)

Premonitory symptoms, as a rule, warn the patient of the approach of an attack. They awake with a peculiar "heavy feeling" in the head, or a sense of languor, or a disinclination to eat, or to smoke (if addicted to tobacco). They are often constipated, and feel a peculiar aversion to mental effort or to gayety. Sudden movements of the head or body are often followed by pain in the head. Stooping is liable to produce unpleasant sensations. They are apt to yawn frequently, and eructations are not uncommon. In rare instances, these attacks are preceded by a marked buoyancy of spirits on the preceding day.

As the day progresses, pain of a decided character begins to be felt in the head. It may be unilateral or bilateral. If unilateral, the left or

the right side are equally apt to be the seat of pain. The special senses become abnormally acute. The temporal arteries are swollen on the affected side and feel like whip-cords beneath the skin. The patient begins to experience pulsations within the head, which send shooting pains with them of a severe character. The region of the stomach often feels unpleasantly, as if distended with gas. Light becomes painful, and the eye of the affected side gradually changes in its appearance. It is usually dull, retracted, and not widely opened. The pupil may be slightly dilated.

The seat of pain may be at first in the occipital region or over the forehead or temple; but it tends to spread gradually over the affected side as the paroxysm increases in severity. The cervical region of the spine is apt to be more or less affected with an aching sensation.

Toward the close of the day the patient is obliged to give up business and retire to absolute quiet and a dimly-lighted room, provided the attack is a severe and typical one. The pain becomes more and more intense until it culminates in vomiting. This often closes the attack and the patient falls into a heavy sleep closely resembling that which follows an epileptic seizure. Several fits of vomiting are generally experienced, accompanied by retching, sweating, pallor and extreme physical prostration, before entire relief from pain comes.

No one who has never experienced a severe attack of this kind can conceive of the torture which such a patient endures. It deprives him of all power of performing the simplest mental processes and takes away the ability to endure suffering. In addition, the patient is harassed by all forms of visual disturbances, even when the lids are closed. Such patients may be rendered partially or totally blind during the attack. The hearing and smell are so acute as to detect things which would otherwise be unnoticed, and to cause them to suffer acutely in consequence of such perceptions. Bright zigzag lines of light often seem to flit across the vision; or luminous spots may appear, which grow in size and brilliancy till they become intolerable. Sensation may be abolished in some well-defined area of the skin.

The frequency of the paroxysms varies in different individuals. Some have them once a week, others once a month, others much less frequently.

Between the attacks the patient usually feels perfectly well and generally suffers from no nervous symptoms. There are exceptions to this rule, however, which are well illustrated in the few cases cited. These are taken from my own records. They represent but a small fraction of similar cases which I am constantly observing in my practical office work.

Case I. *Inherited Sick-headache, Vertigo, Loss of Emotional Control*, etc.—Mr. ——, aged twenty-eight; has had severe headaches at least once each week since the age of six. Consumption very frequent in his maternal ancestry and immediate relatives. Has suffered with diplopia at times; especially when fatigued by the use of the eyes for near-work. Has lately had a marked increase of the headache. The attacks are excruciatingly severe and almost constant. Has tried almost every known drug, but has found all more or less unsatisfactory. Within the three months preceding the examination of the eyes has had, in addition to the headache, "extreme nervousness," with a loss of emotional control, attacks of vertigo, which have been distressing, and an inability to use the eyes steadily for any length of time without great fatigue. Has used a + 1.00 D. glass at times for reading.

Examination of the eyes shows:—

Without atropine:

- O. D. V. . . . . $\frac{20}{20}$
- O. S. V. . . . . $\frac{20}{20}$
- Binoc. V . . . . . $\frac{20}{15}$
- External insufficiency (esophoria) . . . . . 7°
- Adduction . . . . . 20°
- Abduction . . . . . 4°
- Visual field . . . . . normal.
- Reading power: No. 1 type read easily by + 1.50 at ten inches.

With atropine . .

- O. D. $\frac{20}{100}$ . . . . . made $\frac{20}{20}$ by + 3.00
- O. S. $\frac{20}{100}$ . . . . . made $\frac{20}{20}$ by + 3.00
- Binoc. $\frac{20}{100}$ . . . . . made $\frac{20}{20}$ by + 3.00
- External insufficiency 5°, but totally relieved by + 3.00 glasses, without prisms.
- Reading power: test type, No. 1, read with ease with + 4.00.

Ordered + 1.75 glasses for each eye, in spectacle-frames; these to be worn constantly, for both distant and near vision.

The correction of this ocular defect *made a complete cure.* The patient did not have a headache for over eight months, and all the nervous symptoms disappeared immediately. During the past five years, the attacks of headache have not exceeded two a year. They invariably follow excessive mental effort and a protracted use of the eyes. This case illustrates well the acuteness of vision which existed before the use of atropine, in spite of a high degree of hyperopia.

Case II. *Headache, Mental Depression, and Insomnia.*—Mr. ——, aged forty-nine; lawyer. Phthisis common among maternal ancestry. Has had more or less headache, but no periodical attacks of migraine. Has begun to suffer for the past two years with extreme headaches, insomnia, inability to use the eyes, great mental depression, and vertigo. Has never required glasses even to read.

Examination of the eyes shows:—

A "manifest" myopia of — 0.75 existed before atropine was used.
Hyperopia under atropine corrected by + 1.50.
External insufficiency (esophoria) of 1°.
Adduction, 10°; abduction, 5°.
Marked ciliary spasm (as shown by the effects of atropine).

An uncontrollable trembling of the head, and severe pain at the occiput invariably occurred during the testing of the eyes for insufficiency, before atropine was used.

This was one of the most interesting cases I have ever seen. Any attempt at convergence of the eyes gave intense occipital pain and a shaking of the head which would dislodge the spectacle-frame. Although he exhibited, under atropine, a latent hyperopia of a marked degree, he was apparently myopic before its use, on account of an existing ciliary spasm. The total muscular power of adduction and abduction was extremely low.

The use of atropine arrested all the reflex nervous phenomena in this subject at once. He was kept under its influence for a week; was given + 1.50 glasses for reading and all near-work. By the use of prismatic, daily exercise for the muscles, he recovered the normal power of convergence and divergence in about three weeks. In this case, static sparks were administered daily to the occiput and cervical region of the spine for several weeks, as it gave the patient a great sense of relief in the head. Although all pain had ceased from the first administration of atropine, a sense of "fullness in the head" remained, which was overcome entirely by the static spark and ergot.

Case III. *Hysteria and Morbid Fears, associated with Headache.*—Mrs. ——, aged twenty-seven; wife and mother of two children. Has a well-marked phthisical ancestry. Patient has had scrofulous joint-disease. Prior to the examination she had been growing hysterical, with a tendency to cry constantly. Has had repeated "sinking feelings." Has an aversion to crowds, and morbid fears. Suffers greatly from sick-headaches. Has had amenorrhœa for many years at intervals.

Examination of the eyes shows:—

Without atropine, O. D. V.=$\frac{20}{20}$ . . . . . with atropine, $\frac{20}{70}$ imperfect.
" " O. S. V.=$\frac{20}{20}$ . . . . . " " $\frac{20}{70}$ "
" " Esophoria, 4°.
" " Adduction, 12°.
" " Abduction, 6°.

Hyperopic astigmatism of a high degree (+ 1.50) in each eye.

This patient did not wait in the city to have her visual apparatus corrected. The case, however, illustrates well the existence of a marked hyperopic defect and muscular insufficiency as a cause of headache.

Case IV. *Excruciating Headaches, Chronic Diarrhœa, and Neuralgic Paroxysms.*—Female, aged twelve.

Family History.—The father is somewhat eccentric and intemperate; pulmonary consumption was extremely common among the paternal ancestry; one brother is an epileptic and is partly idiotic; all the paternal side of the family are very excitable and nervous people.

This little patient was a great sufferer. Whenever attempts were made by her parents to send her to school, she would "break down" at once with peculiar attacks, characterized by obstinate vomiting, chronic diarrhœa, intractable headache, and neuralgic pains in the spine, limbs, and chest. All medical treatment had proved of no permanent benefit. So long as study was not attempted, the child suffered only at intervals with these severe attacks, but she remained weak and delicate.

Eye-defects.—An examination of her eyes showed a *latent hyperopia* of nearly 3 D. and *esophoria* of 8°. Prior to the use of atropine she had normal vision ($\frac{20}{20}$). The wearing of spherical glasses (2 D.) and prisms (4° over each eye with the bases out) caused all her distressing symptoms to disappear within two weeks.

A letter, lately received from her mother, says: "I thank you next to the dear Lord for removing this burden of anxiety about my child, which was becoming unbearable."

Prisms were ordered in this case because the parents decided to postpone a radical correction of the muscular error. This step I expect to undertake very soon.

CASE V. *Orbital Neuralgia and Sick-headaches.*—Mr. ——, aged forty-seven. Patient is a lawyer. Mother has had sick-headaches every week for her life-time, confining her to bed. Sister is "very nervous." Patient has suffered personally either with orbital neuralgia or sick-headaches nearly every week for nearly thirty years. The attacks usually last two days and require from one to three grains of morphia to control them. They seem to be independent of any known exciting cause. No remedy has ever been found. Patient has consulted several of our best physicians.

Examination of the eye shows:—

Normal vision before atropine was instilled.

A latent hyperopia of the right eye, corrected by a + 1.50 glass
A latent hyperopia of the left eye, corrected by a + 1.50 glass } under atropine.

Reading test for No. 1 Jaeger's type, under atropine = { O. D. = + 4.25 / O. S. = + 4.25

External insufficiency (esophoria) of 3°.

I ordered this patient at first glasses of + 1.00 for distance, reading, and all near-work. The insufficiency at first appeared to be corrected without prisms by his "distance" glasses. Subsequently I was obliged to add 3° of prism, as he refused surgical interference.

Patient experiences great relief from the glasses and is still under galvanic treatment. The neuralgic attacks are apparently occurring at less frequent intervals.

CASE VI. *Excruciating Neuralgic Headaches.*—Mr. ——, aged thirty-nine. Patient for years had had attacks of neuralgic headache, usually hemicranic. They have occurred with increasing frequency and severity for the past few years. They have of late become almost constant and have rendered the patient entirely incapable of following his profession with pleasure or profit.

Examination of the eye shows:—

A latent hyperopia of each eye, which requires a + 2.75 glass to correct it when the eye is under atropine. Without atropine, V. = $\frac{20}{20}$ in both eyes.

External insufficiency (esophoria) of 6°.

With corrected vision, by means of + 1.50 glasses and 3° of prism over each eye, this patient has almost entirely regained his health. His neuralgic attacks are very infrequent and are usually traceable to excessive use of the adductor muscles. Patient still wears prisms and refuses surgical relief for his esophoria.

CASE VII. *Cerebral Neurasthenia, Pseudo-ataxia, and Chronic Headache.*—Male, married, aged forty-three.

Family History.—Two brothers died of phthisis, one uncle of paralysis; maternal ancestors are nervously predisposed; two children of the patient suffer from nervous disturbances.

Eye-defects.—Hyperopia (latent) of 2 D. Esophoria of 7° (manifest).

This gentleman was a great sufferer. Had been forced to give up his business from constant distress in the head, which was aggravated by any mental labor. He walked

with difficulty on account of a feeling of great insecurity, and closely simulated the gait of an ataxic. He ate poorly and slept badly. No organic disease could be found, and electrical treatment was thoroughly tried, and proved of little benefit. Within twenty-four hours after a tenotomy of his internal rectus was performed he ceased to have pain in his head, walked with greater ease than for many months, and left for his distant home within a week, provided with + 1 D. spherical glasses. The last report from him noted a slight tendency toward a return of his bad feelings, and he was advised to have his eyes again examined to ascertain if any latent esophoria remained to be corrected.

In his last letter he says: "I have not had a sick-headache since leaving New York, and much less of the neuralgic pain in the neck and other parts."

Cases VIII and IX. *Asthenopia and Sick-headaches.*—Mr. —— and Mr. ——, unmarried, aged respectively twenty-one and twenty-two. Both were college students.

Family Histories.—Headaches were common in their immediate relatives and ancestors, and in one phthisis was found to have affected the ancestry.

Eye-defects.—Both had slight latent hyperopia and esophoria (manifest) of a moderate degree.

These were typical cases of asthenopia and sick-headache. Both wore prisms for a while with benefit, but they found that a latent esophoria showed itself, and required a change of the strength of the prisms. The stronger prisms caused them some distress in walking. They both underwent an operation for the radical correction of the defect, and have remained entirely free from asthenopic symptoms and headache up to the present time. The necessity of wearing glasses, which was thus dispensed with, is a matter of delight to both.

In one of these cases, a marked tendency to dyspepsia and chronic constipation has apparently been entirely overcome.

The last tests made of the eyes of these patients showed an entire absence of muscular defect in the orbits.

Case X. *Chronic Neuralgic Headaches of Twenty-six Years' Standing.*—Mrs. ——, widow, aged forty; has had two children.

Family History.—Father died of paralysis. Mother had "suffocating spells" all her life One sister was treated for vaginismus. Dyspepsia is a "family ailment." No phthisical tendencies, as far as known. Her oldest child is "very nervous."

Eye-defects.—Myopia (—2.50 S.); hyperopic astigmatism (+ 0.75 c. and + 1.50 c.); hyperphoria of 4° (manifest); adduction, 24°; abduction, 8°.

Since a child, this patient has had terrible headaches. They are often preceded by blindness. Since her marriage they have increased in frequency and severity. She has (on an average) two each week. She has frequently been in complete coma for twenty-four hours from pain, and "it generally takes forty-eight hours to recover her strength sufficiently to get about the house."

She has had twenty-six teeth drawn, in the vain hope of getting rid of the headaches, which have been pronounced as "neuralgic."

Prior to her deliveries, while pregnant, she would oftentimes become suddenly unconscious several times a day. These attacks were apparently "epileptoid" in character. No uterine disease or deflection has ever been found, although repeatedly sought for.

This patient was treated at the first interview by a partial tenotomy of one superior rectus muscle, as her hyperphoria was of a very marked character, even after the correction of her refractive error by glasses.

On the following day, some two degrees of latent hyperphoria developed and was corrected by a second operation. She was sent to her home in a distant state within a week, provided with proper glasses for constant wear.

Since her return, some six months have elapsed and she has had but two headaches, both of which she attributes to very marked excitement and physical fatigue. She states that her physical condition has improved to a remarkable degree.

Such cases as the last one narrated certainly justify an earnest inquiry regarding the cause and proper methods of cure of chronic headache. This hopeless sufferer recovered without drugs and with little or no inconvenience.

Treatment.—In my opinion, the first step toward a cure in these cases is to examine the eyes when fully under the influence of atropine, and to carefully ascertain the amount of *refractive error* which exists. (See p. 146.)

If a high degree of *hyperopia*, with or without hypcropic astigmatism, be detected, it is often well to keep the patient's eyes under atropine for a period varying from one to several weeks.* During this period, the patient should wear constantly a glass that corrects the hyperopia (less one dioptre—a glass of about thirty-six inches focus†); and a stronger glass may be ordered while the patient is under atropine for such near-work as he is obliged to perform with his eyes. This glass takes the place of the normal accommodation, which is paralyzed by atropine. For example, if a hyperopia of three dioptres is found, the patient should wear a + 2.00 glass for distance, and while under atropine his power of accommodation must be temporarily furnished by the use of a convex glass of sufficient strength to enable him to read ordinary type with ease at fourteen inches. When atropine is discontinued, the latter glass must be dispensed with as soon as the ciliary muscle begins to assert itself; hence, these patients, unless presbyopic, need but one glass, which answers both for distance and near-work after the effects of atropine have passed away. If the patient be simply astigmatic, the full correction will generally be tolerated by the patient, after the effects of atropine have subsided. These cases do not require a prolonged use of atropine, as a rule.

If the *muscles* of the eye are found to be *out of balance*, prisms may be added to the glass which is selected to correct the refractive error; in case the patient absolutely refuses to have a partial tenotomy performed. I am personally opposed to the constant use of prisms, save as a *dernier ressort;* partly because all patients cannot wear them with

* This step is taken to ensure a decrease in the power of a hypertrophied ciliary muscle, which would oppose attempts to correct a high degree of hypcropia which had been "latent."

† A glass of one dioptre is in reality about 40 inches focus. Practically, the subtraction is necessary of 1-36 from the glass, which affords full correction of the refractive error. It is tolerated by the patient if he wears such a glass constantly, and affords great comfort when the ciliary muscle ceases to exert its action in viewing objects beyond the 20-foot radius.

comfort, and also from a knowledge of the fact that the "manifest" insufficiency which the prisms correct is not usually all that actually needs correction. Prismatic glasses frequently render the appearance of the sidewalk or floor apparently concave to a patient when first worn. This effect soon tends to pass away. It is very annoying to some subjects, and they should be prepared for it by timely explanation.

Considerable difficulty may be experienced in getting patients to persevere in the wearing of glasses for a latent hyperopia, after the effects of atropine have already subsided. The ciliary muscle of each eye invariably fights the glass to a greater or less extent, because it has long been accustomed to excessive action in bringing the eye to a focus both for distant and near objects. This accounts for the fact that these patients often experience a dimness of vision at times through the glasses (which passes off as soon as the glasses are removed). I have known vertigo, nausea, and even vomiting to occur from this conflict between the ciliary muscle and the glass.

Patients should be told that these difficulties are apt to arise for some weeks or months at intervals, and that they must persevere in wearing the glasses ordered, even if they have to remove them for a few minutes at a time whenever they become particularly annoying. It is not enough for them to use their glasses for reading or writing only. The "eye-strain" must be overcome without interruption, if we are to expect a cure. These patients will very often tell you "that they can see better without their glasses than with them;" and they may narrate in a complaining tone all the inconveniences of wearing a glass constantly, such as irritation of the nose, the trouble of cleaning them, the accumulation of moisture or snow upon them, the disfigurements, etc. With such complaints I constantly have to deal by impressing them with the benefits derived from the glasses, the difference between seeing without effort and seeing with eye-strain, the necessity of strict compliance with my directions, etc. The wearing of spectacles or nose-glasses constantly is at best a nuisance, and to ladies they are sometimes very repulsive; but when the choice between sick-headaches and glasses is intelligently discussed before this class of sufferers, it is very uncommon to find a rebellious patient.

During a paroxysm, what is to be done for the patient? In the prodromal stage, give a good purgative or an emetic early. If you delay it until late in the day the paroxysm will reach its height. I frequently give compound cathartic pills (three or four at a dose), and follow it in two hours with warm water as an emetic.

Sometimes full doses of bromo-caffeine, or the fluid extract of guarana in drachm doses (repeated if necessary), or large doses of quinine (ten to twenty grains), or copious draughts of very hot water,

will cut an attack short. Antipyrine, in doses of two to five grains every hour, will often cure an attack, even when excessively severe. These remedies, however, are liable to prove ineffectual when most needed.

When the pain becomes very severe, immersion of the entire body in a very hot bath will almost always give relief. A bag partially filled with hot water placed at the back of the neck after the bath is very agreeable to most sufferers of this class and frequently induces sleep.

The patient should be placed in a darkened room and all noises should be excluded. Finally, a full dose of morphia may be required in extreme paroxysms. Of all the remedies that my personal experience has proved to be effectual, a brisk purgative and the hot bath are the most liable to relieve the patient promptly.

I have never observed any decidedly curative effects from medication or electrical applications during the intervals between the paroxysms. The intervals may be prolonged somewhat, in some cases, by tonics, out-of-door sports, or exercise, a regular life, care in the diet, etc.; but the paroxysms tend to return with severity in spite of every known precaution, until the eye-strain is removed.

Of late, considerable attention has been given to the dependence of headache in certain cases upon a partial or complete obstruction of the nasal passages due to hypertrophy of the mucous covering of the turbinated bones or deflections of the nasal septum. These conditions are more commonly encountered than one would at first imagine.

Probably deflections of the septum are due in part to blows received upon the nose during childhood.

The explanation of the development of headache, in consequence of an obstruction in the nares of sufficient extent to interfere with the oxygenation of the posterior nares in the vault of the pharynx, seems to be that of reflex irritation of the nerves distributed to that part, which probably induces vaso-motor disturbances within the cranium.

While my experience in the removal of such obstructions by means of the nasal trephine, the nasal saw, and applications of chromic acid or the actual cautery is not extensive, I can bear testimony to the fact that I have encountered cases where this step has led to marked relief of headache. I believe that in typical sick-headache occlusion of the nares is less frequently observed than in those cases which suffer from the so-called "catarrhal" headaches. These are less paroxysmal and severe than typical attacks of migraine.

NEURALGIA.

In view of the fact that different authors include under this head widely varying conditions which are associated with paroxysmal pain, it may be well to state early what my conception of the term embraces.

It seems to me that the term "neuralgia" cannot be properly applied to paroxysms of pain which are clearly traceable to organic changes in the brain or spinal cord. On the other hand, it is equally clear to my mind that typical attacks of neuralgia of a very persistent and obstinate kind *may* occur without the existence of organic changes in the nerves themselves.

I have for some time regarded most neuralgic attacks rather as a symptom of an existing "neuropathic predisposition," which has been either created by or subjected to some marked reflex irritation, than as clinical evidence of existing disease in the nerves. This condition of reflex irritation is encountered not alone in subjects who have fleeting attacks of pain in various nerves at different times. It is met with often in those who suffer from paroxysms of severe pain with more or less periodicity in some special nerve-trunk or its branches. It is my opinion that a very large proportion of neuralgias are totally independent of pathological lesions either in the nerves or the nerve-centres; and that but a small proportion are the direct result of morbid changes. This subject will be discussed further when the causes of neuralgias are considered.

Neuralgia must not be confounded with *neuritis*. The latter condition is an inflammatory one; in which the symptoms are not paroxysmal but constant while they last, and are not infrequently accompanied by marked disturbances of motility and sensibility, and also of muscular nutrition.

Although the existence of certain tender points in the course of a nerve (where pressure is acutely felt and usually accompanied by pain along the nerve—the so-called "puncta dolorosa" of Valleix) is generally considered as peculiarly diagnostic of neuralgia, there may be cases of typical neuralgic paroxysms, in my opinion, where these tender points are not to be detected after a careful examination.

The "*puncta dolorosa*" are to be sought for, as a rule, near the orifices of bony canals or foramina through which the affected nerve escapes, or where the nerve subdivides, anastomoses, or pierces a fascia. In my work, "The Applied Anatomy of the Nervous System," I have described the situation of these tender points for the more important nerves of the body. Space precludes a full discussion of this field here.

Etiology.—The causes of neuralgia may be classified as follow: (1) The predisposing; (2) the modifying; and (3) the exciting.

The *predisposing* causes of neuralgia (like those of other functional neuroses) comprise all constitutional or acquired conditions which tend to create the so-called "neuropathic tendency."

It is generally recognized that in this particular class of nervous subjects, hereditary influences may be observed in a very large proportion

of cases when carefully sought for. We find, not infrequently, that neuralgia has been present in different branches of the family for several generations; or that some other evidences of a nervous taint have occasionally appeared,—such, for example, as hysteria, epilepsy, chorea, insanity, etc. Again, a tubercular tendency is very commonly detected in the ancestry of neuralgic subjects. Finally, neuralgia may be shown in some cases to be indirectly a result of general anæmia, chlorosis, neurasthenia, and other similar states, which may have been induced by excesses, over-exertion of the mind or body, prolonged anxiety, diarrhœa, or lactation, and many other similar causes.

Now, I believe, from a somewhat extended research into the probable factors which tend to induce the "neuropathic tendency," that "*eye-strain*" and "*abnormal eye-tension*" are perhaps more closely related to this obscure and imperfectly understood condition than any other factors which have as yet been observed. *Dental* and *ovarian irritation* are also peculiarly liable to induce neuralgic paroxysms.

It is safe to assert that comparatively few cases of neuralgia are ever subjected during their lives to eye-tests made in a scientific manner by competent observers. It can be shown, I think, that until recently the tests made by many of the best observers in this field have been more or less superficial in reference to the state of the eye-muscles; and that the method described in this volume (p. 145) is far more complete and thorough than that given by most authors on ophthalmology. It is not hard, therefore, to understand why this statement is not more generally accepted by the profession at large. The medical mind is now rapidly becoming awakened to the necessity of such tests in nervous disturbances: as well as to the importance of more care regarding the details of the methods employed by those who make the tests. Many of the profession (who are not oculists) are to-day beginning to make their own tests for suspected refractive and muscular anomalies. To such we shall soon owe perhaps more valuable information respecting the relationship between errors in the orbit and functional nervous maladies than to oculists, because nervous diseases are more commonly observed in general practice.

The *modifying* causes of neuralgias come next in order. They comprise all forms of infectious, toxic, and depressing conditions which tend to exert a deleterious influence upon the general health of the patient. In this way, malarial influences, the germs of the various fevers, poisoning by lead, mercury, alcohol, tobacco, etc., syphilis, exposure to cold and dampness, over-fatigue, and many other similar causes may have a bearing upon the causation as well as upon the relief of neuralgias.

It should be borne in mind, however, that these causes are usually engrafted upon some one or more of the predisposing causes mentioned.

It is rare, in my experience, to observe cases of neuralgia where a predisposing tendency cannot be demonstrated. I think we are all apt to attach too much importance to the modifying and exciting causes of functional nervous attacks. I believe future investigation will justify this conclusion, and that the views of the profession must undergo a marked reformation respecting this painful and distressing malady.

The *exciting* causes of neuralgias are sometimes very difficult to determine. I quote from Rosenthal, who aptly says: "The arrangement of the central mosaic (*i.e.*, the intermingling of nerve-roots and cells) determines the law of peripheral manifestations." It is often a matter of difficulty to ascertain (even by a careful examination of the patient) whether some local cause may not exist; or, if it is not discovered, whether some form of reflex irritation is not helping to keep the neuralgic manifestations active.

Trouble with the teeth may start up either a trigeminal neuralgia, or an earache, or some other reflex disturbance, which often proves obstinate until the cause is removed or ameliorated. Perhaps a periostitis near to a foramen through which a nerve passes may light up a neuralgia in that nerve. Again, in inflammatory exudations, cicatrices, tumors of all kinds, and other conditions may occasionally press upon a nerve and cause neuralgic manifestations. Finally, reflex irritation from the eye, nose, mouth, uterus, ovaries, intestine, bladder, clitoris, or rectum may prove to be exciting causes of paroxysmal pain far removed from the actual cause.

Of all these reflex causes, I regard the eye as probably the most common, and certainly the most important one to search for early in the examination. So generally do I find palpable evidences of eye-defect in these cases, that I have many times questioned whether this factor does not surpass in its frequency all others combined. Congenital errors of refraction and anomalies in the eye-muscles are, in my opinion, the chief factors in producing the "neuropathic predisposition." Their continuance without relief tends also under certain physical conditions to act as a reflex cause for pain; as well as for the more serious neuroses, when the subject becomes unable to endure the eye-strain which such congenital defects entail. That this conclusion is not irrational, the effects of removal of this factor in many cases which I have personally observed clearly demonstrates.

Morbid Anatomy.—As a rule, it may be stated that neuralgias are seldom dependent upon pathological changes. In a few exceptional cases, however, the nerves and the nerve-centres may reveal in a variety of ways the existence of a morbid state. These morbid conditions may usually be diagnosed after a thorough examination of the case and a careful study of the symptoms has been made.

In the *nerve-trunks*, the following lesions have been observed in reported cases where neuralgia has been a marked symptom: (1), congestion and thickening of the sheath of the nerve; (2), neuromata; (3), sclerosis and atrophy of the nerve; (4), granular degeneration of the axis-cylinders; (5), inflammation or degeneration of the ganglia on the posterior spinal nerve-roots; (6), similar changes in the ganglia of the sensory cranial nerves; (7), simple atrophy of the nerve, following pressure upon it from one or more of the causes previously mentioned; and (8), capillary hemorrhages in the nerves themselves.

In the *spinal cord*, Anstie has claimed theoretically that morbid processes (atrophy or degeneration) in the posterior nerve-roots or in the gray matter of the posterior horns probably exist in neuralgia. Unfortunately for this view, pathological research has failed to verify the hypothesis. Like many pure hypotheses, it is probably without foundation.

In the *brain*, we find that tumors, softening, inflammatory exudations, tubercle, hemorrhagic clots, and many other morbid conditions may, under certain circumstances, irritate the roots of some cranial nerve, or the trunk of the nerve itself. These morbid conditions are, however, in no way to be considered as pathognomonic of neuralgia.

The *cranial nerves* may in some instances be independently affected with any of the changes already mentioned in connection with the nerves.

My friend, Dr. C. L. Dana, of New York, has lately contributed an article to the literature of neuralgia based upon the clinical study of 453 cases. Some of his deductions are of value in this connection, and therefore quoted.

Respecting the *percentage of the various types*, his statistics show that 41 per cent. were of the trigeminal type; 23 per cent. of sciatic; 13 per cent. of the intercostal; 4.5 per cent. of cervico-occipital; 2.5 per cent. of lumbo-abdominal; 2 per cent. of articular; and about 2 per cent. of brachial.

Respecting the *season of the year* which is most frequently attended with neuralgic attacks, he shows that winter and fall have a larger proportion than spring and autumn.

Regarding the *age of patients* so afflicted, he shows that 45 per cent. developed before the thirtieth year.

The *influence of sex* seems to be apparent. Females are shown to be attacked more commonly than men in the proportion of 5 to 3.

In this paper, migraine is classed as a type of neuralgia. Unfortunately, in my opinion, while admitting "the neuropathic predisposition" and marked "family-tendency" to neuralgia in any or all of its forms, this author does not bring into prominence the common

relationship between eye-strain and these attacks which is now being very generally recognized.

In summary, it may be said that we are forced to admit that the pathology of neuralgia is as yet undetermined. It has been sought for by a host of enthusiastic pathologists without any satisfactory results.

The morbid conditions which have been previously enumerated as having been found in exceptional cases are probably the last to be clinically suspected, whenever the paroxysms of pain are unassociated with motor, sensory, or trophic disturbances between the paroxysms. All of the later observations of Dr. Stevens and myself go to show that neuralgic attacks are curable in a large proportion of cases when treated by the relief of eye-strain. This fact is incapable of explanation if the presence of pathological lesions of the brain, spinal cord, or the nerves affected with neuralgic paroxysms is admitted. Like other purely functional neuroses, the detection of the cause and the removal of an irritation (generally of a reflex type), which a thorough examination of the case will usually reveal, results in permanent benefit to the patient and a more or less complete cessation of the attacks.

Symptoms.—The chief symptom of this affection is *pain*. This symptom is characterized by the following peculiarities :—

(1) It is very acute, paroxysmal, and usually intermittent.

(2) If remittent, the remissions are very distinctly marked.

(3) The pain is generally unilateral.

(4) It follows the course and distribution of a nerve.

(5) Tender points (*puncta dolorosa*) are generally present.

(6) The general health is but little affected.

(7) Inflammatory symptoms are absent.

(8) Abnormal phenomena of a sensory, motor, and trophic kind may be associated with the attacks of pain, but do not exist between the paroxysms.

The table on the opposite page will aid the reader in distinguishing the diagnostic points which are to be clinically observed in cases afflicted with various types of neuralgia. Some other diseases which simulate neuralgia will be contrasted with it later.

Clinically we are forced to recognize two great classes of neuralgic patients, viz., those who have mild and infrequent attacks, and those whose sufferings are almost without intermission.

A careful scrutiny of this table will make the prominent features of tic-douloureux, cervico-occipital neuralgia, cervico-brachial neuralgia, intercostal neuralgia, mastodynia, lumbo-abdominal neuralgia, and sciatica more apparent than a long description. Such a table aids the reader, moreover, in contrasting the chief symptoms of the special types of neuralgia most frequently encountered in medical practice.

SYMPTOM.	TRIGEMINAL (*Prosopalgia*) TIC-DOULOUREUX.	CERVICO-OCCIPITAL.	CERVICO-BRACHIAL (*Neuralgia of Brachial Plexus*).	DORSO-INTERCOSTAL (*Intercostal Neuralgia*)	MAMMARY (*Mastodynia*).	LUMBO-ABDOMINAL.	SCIATICA.
PRESSURE-POINTS	*Sup. Maxil. Ophthalmic.* Supraorbital foramen. Parietal eminence. Inner angle of eye. *Sup. Maxil.* Infraorbital foramen. Malar region. Upper lip. Alveolar processes. *Inf. Max.* Mental foramen. Side of tongue.	*Between mastoid process* and *1st cerv. vertebræ.* *Cervical spinous processes.*	Vary with the branches of the brachial plexus attached. Near *internal condyle* Near *head of ulna.* *Bicipital depression.* *Lower ang of scapula* *Axillary fossa.* Spinous processes.	*Close to spinous processes* of the vertebræ (vertebral point). *Axillary line of chest* over affected nerve (lateral point). *Edge of sternum* (sternal point).	Are *inconstant.* Nipples very tender. Sore spots may be detected over the breasts.	Near spines of lumbar vertebræ. Middle of crest of ileum. On scrotum or labium.	Apt to be inconstant or wanting. Lower border of gluteus maximus. Behind trochanter of femur. Middle of back of thigh. Bend of knee. Below head of fibula. Behind the malleoli. Dorsum of foot.
REGION OF PAIN (During paroxysm.)	Depends on *branch of nerve involved*—the ophthalmic, supramaxillary or inframaxillary.	Occiput and back of neck. May extend to ear, scapulæ, and chest (in severe cases).	May be confined to area of distribution of the *median, ulna, musculo-spiral, subscapular,* and *supraclavicular* branches of brachial plexus.	Rarely in *dorsal branches* of the intercostal nerves. *5th to 9th nerves* most often attacked. May extend into arm.	Most marked in the breasts. *2d to 6th intercostals* are attacked. Nipple very sensitive. May radiate into neck, shoulder or back.	Affects areas of distribution of the *lumbar plexus*:— 1. Region of hip and hypogastrium. 2. Groin and inner part of thigh. 3. Scrotum and testicle. Several nerves often attacked simultaneously.	May affect *individual branches* of the nerve. If *whole nerve* is attacked, pain is felt in buttock, posterior part of thigh, leg, and all the foot but the inner side.
CLINICAL HISTORY OF THE ATTACK	Most frequent in females. *Ophthalmic branch* most often affected. *Entire nerve rarely attacked.* Generally unilateral.	Most frequent in females from twenty to fifty years of age. *Occipitalis major nerve* generally attacked. Generally unilateral.	Most frequent in males. Right side usually attacked. May follow traumatism, lead poisoning, joint diseases, etc. Generally unilateral.	Most frequent in females. Follows malaria, injury, chlorosis, exposure, pleurisy, phthisis, typhoid, lead poisoning, etc. Most frequent on left side. Generally unilateral.	Exclusively in females. Develops after puberty. Nodular indurations are not infrequent during paroxysm. Is apt to be persistent and obstinate. Often bilateral.	Affects both sexes equally. May follow spinal caries, spinal meningitis, etc. Most frequent on left side. Generally unilateral.	Most frequent in males (20 to 60 years of age). Follows traumatism, exposure, gout, rheumatism, syphilis, malaria, etc. Generally unilateral.
OCCASIONAL TROPHIC AND VASO-MOTOR EFFECTS	Skin eruptions; blanching of hair; glaucoma; iritis; choroiditis.	Redness of a lateral half of the face; injection of conjunctiva; contracted pupil; excessive secretion of tears.	Pallor or redness of skin; profuse sweating; skin eruptions; excessive growth of hair; changes in nails; glossy skin.	Herpes zoster. Offensive perspiration Cardiac neuroses. Localized anæsthesia. " hyperæsthesia.	Secretion of milk after paroxysms, in some cases.	Profuse sweating. Skin eruptions.	Abnormal redness of skin: excessive growth of hair; excessive sweating; Herpes zoster; boils; paræsthesiæ; atrophy of muscles.
MOTOR PHENOMENA (Which may coexist with the pain.)	Convulsive spasms of the face, and occasionally of the neck. The limbs may be convulsed in rare cases.	Movements of head seriously interfered with during paroxysms.	Fibrillary twitchings; tonic or clonic spasms; paresis or paralysis; permanent extension of the fingers; contracture of muscles.	Body often bent toward painful side. Breathing superficial. Voice feeble.	Vomiting frequent (at height of paroxysm).	Seminal emissions. Spasm of cremaster muscle (retracted testicle). Vesical tenesmus.	Vomiting (at height of paroxysm). Tonic or clonic spasms of leg. Paresis. Posture in bed (usually on healthy side, with affected leg flexed at thigh and knee).

A few points may be mentioned separately, however, which have more than ordinary diagnostic importance in these affections.

(1) The discrimination between neuralgic attacks which follow the development of some *organic cerebral lesion* and ordinary neuralgia is clinically of great importance. It modifies materially the prognosis and the treatment.

(2) The pains of the *first stage of locomotor ataxia* are very often mistaken for neuralgia and rheumatism. In my experience, it is rarely my privilege to encounter a case of this spinal disease where the subject, through an incorrect diagnosis, has not been medicated for the relief of one of these conditions for a longer or shorter time before he is brought to my notice. The peculiarities of ataxic pains have already been quite fully described.

(3) *Organic spinal lesions* of the "non-systematic" type are very apt to be mistaken for neuralgia.

(4) *Vertebral caries* is a prolific source of pain in the young which may be referred to parts which are remote from the spine. I recall a case where persistent pain over the stomach (produced indirectly by caries of the spine) was treated for many months by medication, to the serious detriment of the child.

The table opposite may aid the reader in making the discriminations mentioned.

In *trigeminal neuralgia* the situation of the pain and the locality of the "pressure points" varies with the branch of the nerve which is affected. It is very rarely bilateral, and seldom alternating in character. The ophthalmic branch is most often attacked. When accompanied by spasm of the facial muscles, it is known as "*tic convulsif.*" Muscular movements increase the pain of neuralgic attacks; hence, chewing, laughing, talking, etc., are apt to be studiously avoided during these attacks.

In *sciatica* we have one of the most common and rebellious types of neuralgia. Exertion of any kind, such as sitting or walking, is apt to increase the severity of a paroxysm or to hasten the approach of an attack. It is most common in middle life, and very rarely encountered in childhood. It may be associated with a neuritis. If so, the intervals between the paroxysms are apt to be associated with an aching, burning, or bruised feeling in the leg. The muscles not infrequently undergo atrophy in cases of long standing.

The *skin eruptions* which follow or accompany neuralgic attacks comprise herpes, pemphigus, psoriasis, erythema, and urticaria.

The *duration of neuralgic attacks* varies from a few minutes to several days. Paroxysm generally follows paroxysm with more or less rapidity during the attack; while pain of a less severe type is often felt

SYMPTOMS.	CEREBRAL NEURALGIAS.	LOCOMOTOR ATAXIA.	SPINAL NEURALGIAS.	VERTEBRAL CARIES.
PAIN	*All the branches* of the fifth nerve are liable to be simultaneously affeeted (infrequent in neuralgia). No puncta dolorosa exist. Headaehe is apt to exist.	The pain of this disease has *typical peculiarities* (p. 397). No puncta dolorosa exist. Head-pain is absent as a rule.	Paroxysms of pain may attack different nerves from time to time. Pain in the baek may be constant.	Neuralgic pains are apt to be more or less constant as the deformity develops. Pain in baek is marked.
SPECIAL SENSES	These are liable to show more or less impairment. The seat of the lesion modifies these symptoms. "Choked disk" may exist if intracranial pressure is excessive. Pupils may be affected.	*Robertson's pupil* (p. 120) may exist. *Diplopia* may be developed.	Intercurrent diplopia may develop. Pupils may be unequal.	Not affected.
SYMPTOMS OFTEN ASSOCIATED WITH PAROXYSMS OF PAIN	Vertigo. Mono-anæsthesia. Hemi-anæsthesia. Monoplegia. Hemiplegia.	*Incoördination* of movement. *Anæsthesia* (an early symptom). *Extreme susceptibility to east winds* or *damp atmosphere.* *Decrease* or *abolition* of *reflexes.* *No actual paralysis.*	Paræsthesiæ generally exist. Anæsthesia. Hyperæsthesia. Altered reflexes. Paralyses of various kinds. Girdle-sensation. Bulbar symptoms may develop.	Deformity. Paralyses. Various disturbances of sensation. Altered gait. Psoas abseess.
TROPHIC DISTURBANCES	Are infrequent.	Common, late in the disease.	May develop.	Are generally absent.

during the intervals. Sometimes the approach of an attack is indicated by certain prodromata, such as formication, tingling, or a feeling of stiffness in a part.

The *frequency of neuralgic attacks* depends upon the exciting cause and the susceptibility of the individual to reflex irritation. In malarial cases they occur with marked periodicity, and sometimes begin with a chill and end with sweating.

Neuralgic attacks, when once developed, are very apt to be more or less *frequent and obstinate* to treatment. If the attacks are very severe and prolonged, the general health of the patient may become impaired. The digestion is apt to be imperfect in these subjects, the hours of sleep cut short, the ability to take exercise lessened, the mental power more or less weakened, the disposition rendered irritable, and many other manifestations of the neurasthenic state may become apparent to the patient or his friends. Personally, I regard these symptoms as due, in most cases, rather to the exciting cause of the neuropathic predisposition than to the attacks of pain.

Extremely protracted or very frequent neuralgic attacks tend to impair the *functions of the nerve* so affected. In some instances, we encounter hyperalgesia, anæsthesia, paræsthesiæ, paresis, motor palsy, etc., as a result. Twitchings, tremor, or convulsive spasms may also precede or accompany severe neuralgic attacks. The trophic functions of the nerve will be shown to have been disturbed in case the skin gets glossy or affected with eruptions, when the hair becomes excessively long or thick, when the nails appear to be altered in appearance, or when the muscles undergo marked atrophy.

Diagnosis.—Sufficient has already been said to aid the reader in clinically recognizing the various forms of neuralgia from each other, and in distinguishing typical neuralgic attacks from organic lesions of the brain, spinal cord, or vertebræ, which may induce paroxysms of pain that may closely resemble such attacks.

The tables previously given may prove of utility in making these discriminations.

Prognosis.—Like other functional disease, neuralgia of a mild or severe type is seldom if ever a cause of death. Still it must be admitted that the terrible sufferings of many of its victims not infrequently lead either to suicide or the morphine habit, because many cases are extremely intractable to medicinal treatment, and the vital forces of the sufferer become exhausted in consequence of the recurring paroxysms of pain. When the treatment of this malady shall have been discussed, it will be shown conclusively, I think, that the correction of its reflex causes (chief among which I would mention eye-strain) leads to happier results in the treatment of the obstinate type of

neuralgias than any of the remedial agents commonly extolled by authors of repute.

Treatment.—My views upon this head are somewhat at variance with those which have previously been advanced by most authors. If the fact can be substantiated, that serious eye-defects exist in a very large proportion of those subjects who suffer from frequent and obstinate neuralgic attacks, we must naturally look to the eye for beneficial results which may be anticipated from the relief of this exciting cause. Both of these conclusions are, to my mind, susceptible of proof which is conclusive.

In the prize memoir which Dr. Stevens presented before the Royal Academy of Belgium, he reports careful deductions drawn from eight hundred and eighty-five neuralgic subjects which he had examined with special reference to the existence of eye-defect or muscular errors in the orbit. The effects of his treatment (directed exclusively to the correction of such errors) are shown to be as follow in one hundred consecutive cases selected from this large number:—

After eliminating fifteen cases (in which, for various reasons, he had been unable to obtain complete records of the results of such treatment) eighty-five cases are reviewed by this author in which the *eye alone was subjected to remedial measures* for the relief of neuralgic paroxysms of a more or less persistent and obstinate type. Of these eighty-five subjects, seventy-one, or $83\frac{1}{2}$ per cent., were absolutely cured; ten, or $11\frac{3}{4}$ per cent., were markedly benefited; and four, or $4\frac{3}{4}$ per cent., were not materially relieved.

Although an analysis of so small a number of cases is not sufficient to warrant any positive deductions respecting percentages of cases in which permanent relief may be expected by this method of treatment when skillfully employed by competent experts, it cannot be denied that the results obtained by this author are very remarkable and entitle his views to thoughtful consideration. The total number of persons examined by him for eye-defect, in which neuralgia was a prominent symptom, was a very large one. His observations go to show that it is a rare occurrence to find a typical case of neuralgia in which anomalies of refraction or a lack of equilibrium in the eye-muscles does not exist, provided the history of the case renders the presence of organic changes in the nerve-centres highly improbable.

In my own practice I have examined during the last three years over fifty victims to neuralgia with reference to the existence of eye-defect as an exciting cause of the attacks. I have found in almost every case satisfactory evidence of anomalies, which I have deemed of sufficient importance to require correction. In some, the correction of a latent hyperopia by convex glasses has been followed by immediate relief; in others, astigmatism has existed to a high degree and cylindrical

glasses have been required; in a third class, the eyes have been dissimilar in reference to their refractive conditions; in a fourth class, the eye has given undisputable evidence of a tendency to deviate from the condition of physiological equilibrium. Persons of the fourth class have, in the majority of cases noted by me, given no external evidences of strabismus or a tendency thereto. They have, however, been greatly benefited in almost every instance in respect to their neuralgic habit, after partial tenotomies have been performed upon the eye-muscles for the correction of the abnormal muscular tension. In several instances I have been unable to persuade a patient to submit to surgical relief for this defect. Under these circumstances I have resorted to prismatic glasses. The results so obtained have not been as satisfactory as I might wish.

The following table will give to the mind of the reader a clear conception of the results of these observations:—

A TABLE SHOWING THE RESULTS OF EYE-TESTS* MADE IN FIFTY CASES OF TYPICAL NEURALGIA.

ASTIGMATISM.			MYOPIA.	HYPEROPIA.	EMMETROPIA.	MUSCULAR ERRORS.		
Myopic.	*Hyperopic.*	*Mixed.*				*Esophoria.*	*Exophoria.*	*Hyperphoria.*
3	5	2	10	24	9	26	5	16

Such a table shows conclusively to my mind the necessity of subjecting neuralgic subjects to a systematic method of eye examination as well as a complete physical examination (prior to medicinal treatment, if possible); and it certainly offers some forcible suggestions respecting certain steps which may be taken for its relief.

If, after a satisfactory correction of all existing defects so discovered, the neuralgic paroxysms persist, further search should, in my opinion, be made for conditions which may create reflex irritation; such, for example, as nasal, dental, rectal, uterine and ovarian disorders, which might have escaped notice in the earlier examinations of the patient.

Probably no nervous malady depends, in a larger proportion of cases, upon reflex disturbances than does neuralgia; hence, neither time nor trouble should be spared in ferreting out the cause. That this cause is not always to be sought for along the course of the affected nerve, all authorities agree. That it may be far removed from the seat of pain is indisputable. The admirable work of Hilton on "Rest and Pain" may be read with profit by those who seem to despise scientific methods of research, and who are ever ready to grasp at any new medicinal agent which is presented to them.

* Many of the cases had more than one error. For example, one case had hyperopia, astigmatism, esophoria, and hyperphoria.

I would not be construed as denying *in toto* the influence of debilitating influences in certain cases of neuralgia, such as malaria, rheumatism, syphilis, gout, anæmia, etc. Any of these may constitute a factor in certain cases, which certainly demands our best efforts at its removal; yet, on the other hand, our first duty should always be to detect and remove, if possible, any local cause which may materially assist in prolonging if not in creating the neuralgic tendency.

If *malaria* exists, quinine in large doses, arsenic, or Warburg's tincture will usually aid in the recovery.

When *rheumatism* is present, salicylic acid in some of its various preparations, iodide of potash, the carbonates of soda and potash and the oil of wintergreen may be employed with good results. Colchicum may be given if gout is present.

If the existence of *syphilis* is strongly suspected, mercury by the mouth or skin and the employment of the iodides and tonics will be indicated.

In *anæmic* subjects, it is well to give cod-liver oil, iron, arsenic, and strychnia.

Among the special drugs which have been highly recommended for the relief of neuralgia *per se* may be mentioned: (1) phosphorus, in large doses every four hours for forty-eight hours (Thompson); (2) gelseminum, in doses of from five to twenty minims for trigeminal neuralgia; (3) aconitia (Duquesnil's crystallized alkaloid) in doses of one one-hundred-and-fiftieth ($\frac{1}{150}$) of a grain (Seguin); (4) atropia, by the mouth or hypodermically in one-fiftieth ($\frac{1}{50}$) grain doses (Anstie); (5) osmic acid, in a one per cent. solution by the hypodermic method (Eulenberg); (6) carbolic acid in solution of two parts to two hundred of distilled water, by the hypodermic method in doses of half a drachm (Schultz); (7) syrup of hydriotic acid; (8) oil of turpentine, in doses of a half ounce.

Many of these drugs are exceedingly poisonous; hence, they must be administered with extreme caution, and their effects should be carefully noted while being given to the patient.

During the paroxysms of pain, or following them, the following methods of treatment may be tried in case previous suggestions prove of no value:—

(1) *Blistering* over the nerve. This may be readily done by holding chloroform upon the nerve under a watch-glass. Several spots should be blistered simultaneously.

(2) The *administration of morphia* (by the mouth or by means of a hypodermic syringe). This is our main-stay during the paroxysms.

(3) The *application of ice-bags to the spine* (Chapman). They may be employed for from a half hour to an hour several times a day.

(4) An *application of the actual cautery* over the affected nerve at several points. This gives less pain and better results than blistering.

(5) The *employment of the "static spark,"* or the *positive pole* (anode) of a *galvanic battery* over the seat of pain (see Section VII). I have seen wonderful results follow these methods of treatment.

(6) *Rapid percussion over the puncta dolorosa* by means of the rubber percussion hammer. This will not be borne well by patients during the paroxysm.

(7) *Stretching the affected nerve.* Opinions differ respecting the permanent value of this procedure.

(8) *Acupuncture* or *electro-puncture* over the affected nerve. A needle is thrust slowly and carefully through the skin and as near to the nerve-trunk as possible. When the point touches the nerve (told by a peculiar feeling on the part of the patient) it is best to withdraw the needle. If the galvanic current be employed, care must be taken to prevent electrolysis of the nerve.

(9) Application of *aconitia ointment* (one grain to one ounce of lard) to the seat of pain during a paroxysm. This is very dangerous if the skin is abraded. It should be applied only with gloved hands and kept away from the eyes, nose, or mouth.

(10) Continued applications of *hot water* to the part affected. This remedial measure is often of great benefit during the paroxysms.

(11) Hot water may be injected subcutaneously over the nerve (as a substitute for morphia) during the paroxysm.

SECTION VI.

TOXIC AND UNCLASSIFIED NERVOUS DISEASES.

SECTION VI.

HYDROPHOBIA.

(*Rabies in the Human Race.*)

This disease tends to occur in the human subject after inoculation with the saliva of an animal affected with rabies. Of all animals, the dog is most commonly affected with rabies; although the wolf is frequently so attacked, as may also be cats, cows, and probably other domestic and wild animals.

Morbid Anatomy.—In the spinal cord of patients who have died of this condition, as well as in the brain, the blood-vessels appear to be dilated, and their walls more or less thickened. Amyloid degeneration and an increase of the nuclei in the cells of the neuroglia have also been observed. Small hemorrhages may be occasionally detected in the cord. According to Ross, the median and central groups of cells in the anterior horns of the cord may be shrunken and atrophied; and the nuclei of the pneumogastric and spinal accessory nerves in the medulla may be similarly affected.

Marked congestion and a tendency toward hemorrhages seem to be the morbid phenomena most generally detected. A tendency to the formation of cavities in the brain and the substance of the spinal cord (probably due to degeneration of the nerve-tissue) has been noted by several observers.

It must, however, be confessed that we are as yet more or less ignorant of the exact character of the morbid changes which are peculiarly typical of this disease, as well as of the nature of the poison which seems to excite it. It has even been claimed by some late observers that hydrophobia cannot be regarded as a distinct disease.

Etiology.—From a study of reported cases, the saliva of a rabid animal seems to be the vehicle by which the poison is conveyed in most cases of hydrophobia. Patients who are bitten upon portions of the body which are not protected by the clothing are apparently more liable to develop the symptoms of hydrophobia than when the teeth of the animal pass through the clothing. It has not, to my mind, been positively proven as yet that the poison of rabies can be transmitted by any other secretion than the saliva; not even by inoculation with the blood of the infected animal. The saliva of hydrophobic patients has, however, been known to induce a similar condition in other persons, after coming in contact with a wound or an abraded mucous surface. Cases are recorded, also, where the symptoms of this disease have occurred from a rabid dog licking the hand or face of a human being on which there were pimples or sores (Hammond).

Symptoms.—A wound made by a rabid animal usually tends to heal in the ordinary way. An interval then occurs, which is rarely shorter than a month, and probably never longer than five years, during which time the patient may be totally free from any evidences of ill health. In warm climates, hydrophobia has been known to occur within a few days after the bite. In the vast majority of cases recorded, however, the symptoms have occurred within a period of six or seven months.

During the *period of incubation*, there may occasionally be some unnatural sensations in the region of the wound. These sensations commonly include shooting pains in its vicinity, or a peculiar sense of distress in the parts which the patient can with difficulty describe. It is stated that the scar may, occasionally, become more or less livid. The respiration may become sighing in character and somewhat irregular as the onset of the attack approaches. There may also be a sense of oppression or of constriction in the chest; very often accompanied by a sense of anxiety and disturbed sleep. The patient's disposition may show a change. As a rule, these subjects become more or less moody and irritable. The skin may be dry; and chilly sensations, followed by flashes of heat, are occasionally observed.

The *onset of the attack* is usually accompanied by a sense of uneasiness in the epigastrium, accompanied by a feeling of constriction in the throat and a difficulty in swallowing. On attempting to swallow, the muscles of the throat are attacked with irregular spasms. The patient cannot be induced on account of these spasms to partake of liquid or solid food. Speech may be embarrassed in some instances by a peculiar and painful stiffness of the tongue. I have seen food ejected from the mouth with considerable force, during an attempt to swallow, by one patient whom I was called upon to attend.

The tendency to spasm and the reflex excitability of the patient soon becomes intensified. The whole body may become convulsed from the mere suggestion of food or drink, or by any sudden sound, a draught of air, a bright light, the sight of water, etc. The patient generally shows physical exhaustion early. He may also be affected with trembling, weeping, excessive perspiration, delirium, hallucinations and delusions.

The *spasmodic phenomena* generally tend to increase in severity and frequency as the disease progresses. Vomiting may occur; and intense headache is not infrequent. A severe pain in the spine and muscles of the back and abdomen may develop. The mouth often becomes excessively dry and parched. The saliva may be very tenacious and frothy, and expectorated with difficulty. Moistening of the lips or mouth not infrequently tends to excite convulsions. In one of the cases which I observed some years since, the constant attempts of the patient to rid the mouth of tenacious mucus was the first symptom observed. It

preceded the convulsions, and lasted several hours. Within five hours from the time when I first saw the patient, general convulsions of a severe character developed and continued uninterruptedly until death occurred.

In three patients which I have personally been called upon to treat, I have never observed any efforts on the part of the patient to snap or bite or to make any noise which could be compared to the barking of a dog. I am inclined, therefore, to regard published statements that such symptoms have occurred as due rather to the imagination of the attendants than to accurate observation.

The *termination* of this disease is usually due either to physical exhaustion, the frequent convulsive attacks, the loss of sleep, the inability to take food, or to great emotional excitement. It is stated that paralyses have been known to occur during an attack of hydrophobia, but I have never observed it.

The *temperature* of the body is markedly elevated, as a rule, at the beginning of the disease, and is usually highest at the height of the paroxysm or during their subsidence. Hammond states that it may rise to 110°.

The *duration* of the disease seldom exceeds three days, when the symptoms are well established. Death generally occurs during a spasm. It is stated that the power of swallowing sometimes returns before death.

Diagnosis.—Some of the manifestations of hysteria (usually following severe fright occasioned by the bite of some animal not affected by rabies) may closely simulate genuine hydrophobia. It is stated that such cases can usually be distinguished by the fact that the convulsions do not invariably follow slight forms of external irritation, such as those already mentioned. Moreover, attempts at swallowing are not always accompanied by spasmodic action of the muscles of the throat. Hammond states, for example, that "the hysterical patient is apt to be loud in the expression of apprehension, while the real hydrophobic one, though intensely anxious and terrified, endeavors to prevent others perceiving the state of his mind."

The diagnosis of hysteria will be rendered probable if the attack comes on too soon after the bite to allow of a period of incubation; and if the patient can be shown to possess hysterical tendencies. Simulation of hydrophobia is not confined to females. It may prove fatal.

An *attack of tetanus* may be confounded with hydrophobia. It should be borne in mind, however, that tetanic spasms are of the tonic variety; that they affect chiefly the jaws and the back (not the throat primarily); that the mind is unaffected at all times; that atmospheric influences often predispose to tetanus; that the facial expression shows less terror on the part of the patient; and that there is no relationship between the convulsive attacks and excitability of the special senses.

Prognosis.—After hydrophobia has once developed, there are, in my opinion, no well-authenticated cases of cure. On the other hand, the prevention of attacks by the method of inoculation lately advocated by Pasteur, seems to have been quite well proven in some of his cases. The bite of the rabid wolf seems to be more uniformly followed by hydrophobia than that of the rabid dog. Those subjects who are bitten by a rabid animal first are more liable to develop hydrophobia than are those who are subsequently bitten; a fact that tends to prove that the poison becomes to a certain extent exhausted, even in the rabid animal. Moreover, a wound which bleeds very freely is less liable to be followed by hydrophobia than one in which the poison is not washed away.

Treatment.—When any one is bitten by a rabid animal, or even by one that is suspected to be rabid, the wound should be at once sucked by the patient; or, when not so, by an attendant as soon after the injury as possible. There is no danger in this step, if the mucous membrane of the mouth or lips be not broken or abraded.

The wound should also be thoroughly cauterized, either by caustic potash, the nitrate of silver, or by the ignition of gun-powder in the wound. A complete excision of the wound may also be performed immediately after the accident, the part being tightly constricted after the accident to prevent the circulation carrying the poison away from the wound.

During the attack the treatment is purely symptomatic. If ether or chloroform be inhaled by the patient, food may be introduced into the stomach by means of a tube. Milk is preferable to any other form of nourishment, combined with alcoholic stimulants. Stimulants and nourishment may also be given to the patient as enemata.

Among the drugs which have been employed during these attacks, the following may be mentioned: (1) Hypodermic injections of morphine and atropia during the paroxysms. (2) Large doses of chloral (twenty grains), and one ounce of brandy with two ounces of beef-jelly every three hours as enemata. This treatment resulted in an apparent cure, in the experience of Broadbent. (3) The tincture of cannabis indica has been administered in doses of from six to fifteen drops every few hours.

A continuous application of the galvanic current from the soles of the feet to the forehead during the attack has been suggested by Schivardi, of Milan. In one of his cases, such a current was maintained for fifty-eight consecutive hours from twenty-two Daniel's cells. Life was thus prolonged for over seven days.

The late researches of Pasteur seem to have been attended by some extremely favorable results, although inoculation by his methods have not thus far been proven to be an absolute specific against the poison of rabies.

MULTIPLE NEURITIS.

Before we consider in detail the various forms of toxic neuroses, it may be advisable to discuss a condition which has attracted considerable attention of late, first, because its existence seems to be positively determined, and, furthermore, because its pathology tends to shed much light upon many reported cases that have heretofore been probably incorrectly interpreted. I refer to the disease now known as "multiple neuritis."

Under this term may be classed all toxic and a few spontaneous cases in which a peculiar combination of abnormal motor, sensory, and trophic phenomena may have existed during life in consequence of morbid changes in the peripheral nerve-trunks.

Morbid Anatomy.—In multiple neuritis, the ultimate nerve-fibres appear to undergo certain alterations in their structure which vary somewhat in their degree and type, but which are embraced under the general terms "*degeneration*" and "*regeneration.*"

During the former process, the myelin and axis-cylinder of the affected fibre becomes at first segmented; subsequently more or less disintegrated and studded with new nuclei; and finally absorbed to a greater or less extent, leaving the sheath comparatively empty save the presence of scattered nuclei and some débris. The terminal plates in the muscles are simultaneously affected. They tend to become granular and to undergo absorption. It is also probable that the terminal sensory organs become simultaneously altered in their structure; but the changes which they undergo are less positively determined than those in the muscular apparatus.

Should the process of regeneration follow after the extensive changes already enumerated have taken place, a new axis-cylinder and myelin sheath is slowly formed. This is probably effected either by the aid of the nuclei which remain in the sheath of Schwann or by a direct growth of the axis-cylinder from the proximal end of the nerve. Regeneration is, as a rule, an extremely slow process. From six to eighteen months may be required to insure a complete restoration of a nerve after the fibres have been seriously impaired by a degenerative process.

In multiple neuritis, the morbid changes observed in the nerve-trunks attacked are so closely allied to those which experiment has proven to result from compression of a nerve-fibre with destruction of its axis-cylinder—the process of "nerve-degeneration"—that they may be considered as practically identical.

The dependence of these changes in mutiple neuritis upon a spinal lesion seems to be disproved by the fact that the nerves are not affected throughout their entire length, but only in their distal portions. M. A. Starr, in his late lectures upon this condition,* before the New York

* *Medical Record*, February 5, 1887.

Pathological Society, lays great stress upon this point, as well as upon the fact that the sensory nerves as well as the motor are affected (centrifugal degeneration of sensory nerves being never observed as a sequel to a spinal lesion). He regards the morbid condition as one of "primary parenchymatous inflammation of the nerve-fibres."

This author classifies the conditions observed in the nerves of subjects attacked with multiple neuritis as of three types :—

In the first, the nerve-fibres exhibit no fatty metamorphosis. They undergo degeneration in their distal segments and terminal plates according to the normal formulæ.

In the second, the presence of fatty changes is very apparent. He regards this class as possibly dependent upon the pressure exerted upon the nerve-fibres by the exudation resulting from a primary interstitial inflammation.

The third class recognized by him is that originally described by Gombault as typical of lead-poisoning. It consists of a segmental degeneration of a nerve, with normal nerve-segments being interposed. He states that this condition has been observed also in diphtheria by Pitres and Vaillard.

Finally the muscular fibres exhibit an increase of their nuclei, a fatty degeneration of the sarcous elements, and a progressive atrophy proportionate to the destruction of the nerve-plates.

Etiology.—The many conditions which have been imperfectly recognized and interpreted in times past, and which the light of recent investigations seem to connect strongly with an existing multiple neurosis, offer some practical deductions respecting the etiology of this morbid condition. There is, probably, no doubt that many cases which have heretofore been reported as dependent upon a spinal lesion (chiefly those of supposed polio-myelitis anterior), and, in addition, some of the so-called "functional neuroses" have at times been wrongly diagnosed. The following classes of cases are enumerated by Starr as particularly liable to be induced by inflammatory conditions of the peripheral nerves :—

(1) The "*numb fingers*" observed in women about the climacteric period (first described by I. I. Putnam as a disease); *intermittent paralyses;* and the endless train of obscure *subjective symptoms* clinically encountered; such, for example, as pain, formication, numbness, muscular weakness, which is not far removed from paresis, tremors and slight convulsive attack, flashes of heat and cold, etc.

(2) *Toxic Cases.*—Among these may be prominently mentioned the paresis and tremor encountered in chronic alcoholism, and the symptoms of arsenic, lead, and bisulphide of carbon poisoning.

(3) *Infectious Cases.*—These include the results of diphtheria, the

eruptive fevers, tubercle, malaria, and "beri-beri" (an epidemic disease due to a recognized bacillus).

(4) *Spontaneous Cases.*—These apparently follow exposure to dampness, cold, or over-exertion; but their exciting causes are not, as yet, well understood.

Of the toxic class, alcohol and lead are the most prolific causes. Paralysis is, as we all know, not an infrequent result of chronic alcoholism; and it is also a typical symptom of lead-poisoning. The question whether syphilis can directly excite multiple neuritis does not seem to be yet well determined.

Symptoms.—In multiple neuritis, we are apt to encounter a peculiar train of symptoms in which are combined abnormal motor, sensory, and trophic phenomena. This clinical fact is of great value, since it may enable the observer to exclude the existence of a spinal lesion that might otherwise be strongly suspected to exist. In the main nerve-trunks the motor, sensory, and trophic fibres are so intermingled that a lesion of the entire nerve could hardly fail to involve each, and thus cause three distinct sets of symptoms.

From an analysis of all the reported observations to date, the deductions made by Starr relative to the symptomatology of this imperfectly understood disease are of special interest. The following tables will present the views of this author in the fewest possible words :—

Sensory Symptoms.	*Paræsthesiæ* of various kinds occur at the onset. These tend to disappear as the disease advances. *Occur only below elbows and knees*, save in very rare instances. *Moderate pain.* Not continuous. Less severe than in ataxia. *Tenderness in nerves and muscles is constant.* It renders manipulation painful and interferes with massage and electrical applications. *Anæsthesia* is rarely complete, but occurs late in the disease. *Delayed transmission* of pain-sensations and temperature-sensations is generally observed. *Impaired pressure-sense* is usually to be detected. *Muscular sense* may be impaired, but is often normal. *Special senses* are not affected, as a rule.
Motor Symptoms....	*Progressive muscular weakness and a sense of fatigue* occur early. This becomes true paralysis within the space of a few weeks, as a rule. The paralyzed muscles are chiefly those which *move the feet and hands.* The distribution of the paralysis depends on the nerve-trunks which are most involved. *Facial muscles* and *cranial nerves* are not usually attacked. *Spasmodic phenomena* are rarely observed. *Tendon reflexes* are abolished. The muscles lose their natural tone and tend to atrophy. The so-called "*reaction of degeneration*" is detected by galvanic tests, and *very strong galvanic currents are required* to cause muscular contractions early in the disease. This fact is in contrast with the condition observed in poliomyelitis. *Faradaic contractility* is often abolished early in the disease. It may not be entirely lost, however, in some cases. The so-called "*drop-wrist*" and "*drop-foot*" are characteristic deformities. They are very often encountered. The "*claw-hand*" and *various forms of talipes* may also be developed.
Trophic Symptoms.	*Œdema* may be a marked and early symptom. It may affect the feet, hands, and joints. It is usually temporary. *Excessive perspiration* of the feet and hands, possibly with an offensive odor. *Glossy skin.* This may develop early and be persistent. Its disappearance usually indicates regeneration of the nerves. Ulcerations, bed-sores, and eruptions are rarely encountered.

The *onset* of multiple neuritis, like that of some spinal disease, may sometimes be sudden and characterized by marked febrile symptoms. The temperature may occasionally rise to 103° or even 104.5°, but it probably has a lower range, as a rule. The pulse is generally somewhat accelerated, but it may rise in exceptional cases as high as 140. Starr states that such an increase with marked irregularity probably indicates degeneration of the vagus nerve.

The sphincters are never affected in multiple neuritis; nor are the automatic acts associated with them.

Dr. H. D. Chapin, of New York, has lately reported some cases observed among children in which the symptoms closely simulated those of infantile paralysis, save that *sensory symptoms coexisted with paralysis and muscular atrophy.* These cases were surmised by him (probably with correctness) to be examples of multiple neuritis. No autopsy has ever, to my knowledge, confirmed such an observation in a child, although the literature of this disease is still somewhat limited.

A disease known as "KAKKE," or "BERI-BERI," has existed among the Chinese for centuries. It is now recognized as an epidemic variety of multiple neuritis, and its bacillus has lately been not only discovered, but cultivated. E. C. Seguin has lately published quite an interesting contribution to this subject, and M. A. Starr gives in some detail the history of this disease in his admirable lectures, to which the reader is referred. It usually begins with a coryza, an inflammation of the palpebral and ocular conjunctiva, and some febrile symptoms. Following these symptoms, those of a typical multiple neuritis are developed more or less rapidly. It has proven to be at times a very fatal type of epidemic.

Diagnosis.—As multiple neuritis occasionally so closely simulates some organic spinal diseases that a diagnosis is rendered extremely difficult, I have deemed it advisable to present its chief symptoms in contrast with those of poliomyelitis anterior, locomotor ataxia, and diffuse myelitis. These three diseases are particularly liable to be confounded with it, and, in the case of the two latter, it is believed that multiple neuritis can occur as a complication. According to the statements of Leyden, who disagrees with many other observers, the existence of diffuse myelitis without a coexisting multiple neuritis is rendered highly improbable. It is certain that the symptoms of these two diseases present many points of similarity which might easily mislead any one in making a diagnosis.

In connection with locomotor ataxia, there seems to be a growing tendency among French neurologists to regard the serious trophic disturbances which are occasionally encountered during the final stages of tabes as dependent upon a complicating multiple neuritis.

The table opposite may aid the reader somewhat in making a differential diagnosis:—

	MULTIPLE NEURITIS.	POLIOMYELITIS ANTERIOR.	LOCOMOTOR ATAXIA.	DIFFUSE MYELITIS.
ONSET	Gradual, preceded and attended by numbness, pain, marked muscular hyperæsthesia, and tenderness along the affected nerves.	Sudden, with no abnormal sensory phenomena.	Characterized by abnormal sensory phenomena, such as the typical "lightning pains," moderate anæsthesia, paræsthesiæ, etc.	Spinal tenderness to heat or pressure is apt to be present. The nerves and muscles are not hyperæsthetic. Is a rare disease.
ATAXIA	Ataxia and loss of muscular sense may occur early.	Ataxia does not develop.	Develops late in the disease, as a rule.	Not present, as a rule.
PARALYSIS	Advances gradually, and tends to deepen rather than to subside. Affects the legs first, as a rule.	Subsides more or less rapidly after its onset. Its distribution depends upon the spinal segments attacked.	True paralysis or paresis does not develop. Incoordination of movement may seriously disturb locomotion, however.	Not confined to the distal portions of the extremities. Develops progressively.
MUSCLES ATTACKED	Grouped according to their nerve-distribution. Legs affected below knees, and arms below elbows.	Are apt to be grouped physiologically, *i.e.*, according to their function.	Legs usually affected first. The arms may become ataxic later.	All the extremities and even the muscles of the trunk may be attacked.
ELECTRICAL TESTS	Reaction of degeneration.	Reaction of degeneration.	No reaction of degeneration.	Reaction of degeneration may be detected.
BLADDER AND SEX'L ORGANS	Not affected.	Not affected, as a rule	Very often affected.	Are generally affected. Cystitis commonly observed.
HISTORY OF PATIENT	Poisoning by alcohol, lead, arsenic, infectious diseases, etc., in most cases.	No toxic history. Cause may often be very obscure.	The cause may be very obscure. Toxic conditions (save syphilis) are not detected.	Toxic causes are usually to be detected.
REFLEXES	Knee-jerk may be abolished, and subsequently return.	The reflexes may be normal.	Knee-jerk is usually abolished early in the disease, and seldom, if ever, returns.	Are apt to be modified or abolished.
TROPHIC DISTURBANCES	Muscular atrophy. No bedsores.	Muscular atrophy. No bedsores.	No muscular atrophy. Bedsores may be developed, Charcot's disease, skin-lesions, etc.	May occur.
CINCTURE-FEELING	Absent.	Generally absent.	Well marked.	Generally present.
RECOVERY	May be complete.	Is seldom complete.	Recovery very rare, if it ever occurs.	Is usually fatal.

Prognosis.—A complete removal of the toxic condition which generally constitutes the underlying cause of multiple neuritis tends, as a rule, to aid in establishing a more or less complete regeneration of the nerves. Time is an essential factor in this step, however; the process of regeneration often extending over a period of from two to sixteen months.

In the majority of non-epidemic cases, a complete recovery may reasonably be anticipated. In epidemics, the proportion of recoveries is less. The evidences of a commencing regeneration are shown first in a stationary condition, followed by an amelioration of the symptoms and an alteration in the galvanic formulæ of the nerves and muscles affected, and a return of faradaic contractility. The cases which offer the most serious prognosis are those which are due to an excessive toxic condition, and, according to Starr, those in which the disease "begins with great suddenness, advances rapidly, and involves the phrenic and pneumogastric nerves."

Complications may occasionally arise in connection with multiple neuritis and materially modify the prognosis. Among those to be anticipated, the following may be mentioned: (1), myelitis; (2), cerebral and gastro-intestinal symptoms (chiefly in alcoholic patients); (3), ataxia; (4), paralysis of the cranial nerves (chiefly in diphtheritic subjects); and (5), where tuberculosis or syphilitic lesions develop in the nerve-centres or viscera.

Treatment.—The pains should be controlled by a judicious use of hot or cold applications, and also by morphine, whenever excessively severe. The tenderness of the limbs often renders the employment of evaporating lotions of real service. The internal administration of the salicylate of soda in large doses is said to exert a specific action upon the disease in its early stages. Starr recommends also the employment of cold douches, lotions of carbolic acid (5 per cent.), enveloping the limbs in cotton and oil-silk, and rubbing of the limbs with cocoanut oil.

The suggestions previously made in relation to the treatment of syphilis (p. 291), and those which are subsequently given relative to the treatment of lead-, arsenic-, and alcohol-poisoning, are applicable to certain types of this disease. In alcoholic patients, every precaution should be taken to prevent the patient from surreptitious indulgence.

In chronic cases, strychnia (in doses of $\frac{1}{60}$ to $\frac{1}{20}$ of a grain), phosphorus (chiefly in some preparation of the hyperphosphites), iron, and Fowler's solution of arsenic may prove of benefit whenever the patient is anæmic or exhibits low vitality. It is also well to stimulate the circulation of the limbs by alternating hot and cold douches, warm baths at night, systematic daily massage, and electrical applications.

Respecting electrical applications, the employment of galvanism or static electricity is often rendered imperative when faradaic contractility

is abolished by the degeneration of the affected nerves. This subject will be discussed more fully in a subsequent chapter. The reader is also referred to the tests for nerve-degeneration given on page 189.

CHRONIC LEAD-POISONING.

(*Plumbism—Saturnine-poisoning.*)

Lead may be taken into the system (1), by means of the skin in certain occupations; (2), by the drinking of water which has remained too long in lead pipes or tanks; or (3), by its inhalation in the form of dust. A case of a peculiar character came under my observation some years since, where a child swallowed a lead toy and was subsequently affected by recurring attacks of lead-poisoning. It eventually proved fatal.

Etiology.—In some of the manufacturing processes, the workmen are constantly exposed to the deleterious effects of lead. Thus, for example, the manufacture of paint, of colored papers, of rubber, of enamel, etc., predisposes the workmen employed to this disease. Many of our foods which are preserved in tin cans may become impregnated with lead. A child of one of my intimate friends was thus fatally poisoned. The drinking-water of suburban residences is particularly liable to become poisonous in case it is allowed to stand during the winter months in lead pipes or in cisterns lined with lead. Some of the mineral waters, as well as ale, beer, etc., are liable to contain lead from being stored in improper receptables.

Morbid Anatomy.—The morbid changes observed in all toxic paralyses are closely allied to those of multiple neuritis. The *nerves* which supply the paralyzed muscles frequently undergo atrophy and degeneration.

In the *spinal cord*, changes have been observed in the cells which compose the anterior horns.

The *extensor muscles* of the upper extremities and the *musculo-spiral nerve* are most frequently affected when lead is the exciting cause.

The *affected muscles* exhibit a marked increase of nuclei of the sarcolemma, a gradual wasting of the muscular fibres, an increase of the interstitial tissue, and occasionally an increase of fatty tissue. According to some authors, the changes in the muscles are primary; those of the nerves and the spinal cord being developed after the muscles. It is maintained by others that the peripheral nerves first exhibit a degenerative form of atrophy.

In some instances, *the brain* has been found to exhibit induration and atrophy; and also marked evidences of inflammation and softening.

Symptoms.—The effects of lead-poisoning may be indicated by the following conditions: (1) Lead-colic; (2) lead-paralysis; (3) brain symptoms; (4) lead-anæsthesia; (5) lead-hyperæsthesia.

Lead-colic.—The presence of pain of a very severe character in the region of the umbilicus, the epigastrium, the hypogastrium, or the entire abdomen, should always give rise to a suspicion of lead-poisoning, in case the abdomen is found to be unusually hard or retracted during the height of the paroxysm. Such attacks are usually accompanied, moreover, by nausea and vomiting and the most marked constipation.

The pain is generally relieved by firm and uniform pressure. The duration of the paroxysm varies from a few minutes to an hour or more. The paroxysms tend to return, and are generally most severe during the night. Patients usually lie upon the abdomen or press firmly upon it with their hands.

The distress of the patient during a paroxysm is intense, and the respiration is somewhat more frequent than normal and often quite irregular. The pulse is seldom accelerated. This form of lead-poisoning is particularly liable to occur in painters.

Lead-paralysis.—This is one of the later symptoms of lead-poisoning. A blue line on the gums and more or less frequent attacks of lead-colic usually precede development. An excessive use of alcohol and the persistent use of the muscles of the forearm are said to predispose to it.

As a rule, *this form of paralysis* develops gradually. In exceptional cases, however, a sudden onset may be observed. The muscles supplied by the musculo-spiral nerve are more often attacked than those of the lower limbs, the back, or those of the chest. It may occur in one arm or in both. The common extensor of the fingers and the extensor to the wrist, little finger, and index finger are usually attacked first. The muscles of the "ball of the thumb" and the first interosseous may be involved later. The triceps usually escapes. In the lower limbs, the muscles of the peroneal group are usually first attacked.

The *onset* of this form of paralysis is usually accompanied by a slight numbness and a tendency to tremor in the muscles affected. The patient notices a progressive loss of strength in one or both hands, and a marked difficulty in extending the wrist and the fingers. The circulation in the affected limbs is imperfect, and the joints may be more or less painful and swollen.

The "*reaction of degeneration*" develops in the paralyzed muscles. Faradaic irritability of the muscles is gradually abolished.

Fibrillary contractions in the affected muscles are often observed.

The *cutaneous and tendon reflexes* are decreased or abolished in those regions where the muscles are affected.

The *peculiar cachexia* can generally be observed. The disease tends to run a chronic course. Occasionally relapses are observed, without any fresh exposure to lead.

Trophic disturbances may occasionally be noted, chiefly in the form of an enlargement of the sheaths of the tendons and of the phalanges.

Brain Symptoms.—These are comprised under the condition known as "*lead-encephalopathy.*"

The symptoms of this condition, if of *a mild character*, include dizziness, headache, an abnormal irritability or depression, an incapability for mental exertion, disturbed digestion, tremors in the hands, and disturbed sleep. The tremulous condition of the hand exists when the limbs are at rest; but it is greatly aggravated whenever the muscles are actively employed.

When this condition is of a *more severe type*, delirium, convulsions, and coma may develop. The convulsive attacks may closely resemble those of genuine epilepsy. Repeated convulsions of this type may occasionally follow each other.

If *coma develops*, the loss of sensibility is only partial.

The *breath* usually has a peculiar odor. As is noticed in all severe forms of lead-poisoning, the complexion of these patients is usually pale, and marked emaciation is present.

Lead-anæsthesia.—This commonly affects the *optic nerve*, and causes more or less complete blindness. It may occasionally be observed both in the skin and muscles of the extremities and the trunk. Its onset is usually rapid. Hammond states that it reaches its height in a few hours.

Lead-hyperæsthesia.—Lead-poisoning may create pains in the limbs, chiefly in the *flexures* of the joints of the leg. The groin and the popliteal space are generally affected when the leg is attacked; and the axilla and bend of the elbow, when the upper extremity is attacked.

These *pains occur in paroxysms* and appear to be excited by movements, exposure to cold, or marked emotional disturbances. They are usually relieved by firm and uniform pressure over the seat of the pain. There is no redness, swelling or heat in the affected region. The character of the pain presents many variations. It may be an ache, a sense of heat, or a sharp twinge of acute pain.

The use of cosmetics may be the cause of lead-poisoning. From such a cause, the muscles of the face may be paralyzed; an occurrence which is extremely rare under other circumstances. The use of certain hair-dyes containing lead is said to result in lead-poisoning in occasional instances.

Diagnosis.—The diagnosis of lead-poisoning is usually not difficult. A careful investigation of the case will show that the patient has been exposed to the poisonous action of lead in some way. The muscles affected by paralysis are so often those which are supplied by the musculo-spiral nerve (with the exception of the supinators) that it can

scarcely be confounded with a local affection involving that nerve; because the supinators would in that case be paralyzed as well as the extensors, and sensory disturbances, moreover, would probably coexist with the motor symptoms.

In peripheral palsy of the musculo-spiral nerve, a local cause can generally be demonstrated, such for example as a traumatism, sleeping upon the arm so as to affect that individual nerve, crutch-injury, etc. It is stated that Russian coachmen frequently get paralysis of this nerve from winding the reins about the arm while they fall asleep upon the box.

The *history of the patient* will usually show that attacks of lead-colic have existed before the paralysis appeared; and the appearance of the gums may also indicate the existence of the poisonous effects of lead in the system. Lead may *almost invariably be detected in the urine.*

Poliomyelitis may in some cases resemble lead paralysis. The distribution of the paralysis is, as a rule, very different from that which is due to lead; and, in the history of the patient, nothing can be detected which points toward a toxic condition. The urine would also be found free from lead-salts.

Acute cerebro-spinal meningitis may be recognized, from the brain symptoms which follow lead-poisoning in some cases, by the fact that there is a marked rise in temperature in the former, while there is no increase in temperature, as a rule, when the symptoms are referable to lead.

Prognosis.—Unless the patient takes precautions to prevent subsequent poisoning by this agent, relapses are very liable to occur. Even without renewed poisoning, relapses are not infrequent. The prognosis depends, in individual cases, upon the extent of the paralysis and the reactions of the affected muscles to electric stimulation. If the muscles shall have entirely lost their excitability to the faradaic current, a complete recovery cannot reasonably be expected inside of several months, if at all.

The *brain symptoms* which sometimes accompany plumbism are the most serious of its clinical manifestations. In these cases death may occur.

Under proper treatment, I have never observed a fatal result from lead-poisoning.

Treatment.—Whenever a patient is known to be exposed to the poisoning influence of lead, it is the duty of the physician to warn him of his danger and of the serious consequences which are apt to follow. His urine should be examined for lead from time to time. If an attack of lead-colic should occur, the patient should at once remove himself from such a danger; even if the attack should yield promptly to treatment.

In the construction of factories where lead is employed too much stress cannot be laid upon the proper protection of the workmen employed. Again, patients who are known to be addicted to the use of cosmetics and hair-dyes should be instructed to discontinue their use in case they are found to contain lead.

The *internal administration* of the *iodide of potash*, in doses of from ten to fifteen grains three times a day, has been shown to set free the lead that is in the system, by causing a decomposition of the albuminates with which that metal becomes united. The iodide of lead is thus formed, and this salt is rapidly excreted from the system, chiefly by the kidneys. Under its use, a disappearance of the blue line around the gums will usually take place rapidly.

When there is great debility the *administration of iron*, *quinine*, or *strychnia*, may often be advisable.

The *hygienic surroundings* of a patient suffering from lead-poisoning should be carefully regarded, and nutritious and easily digested food should be given.

Attacks of lead-colic frequently demand the use of morphine, preferably by the hypodermic syringe, to control the pain. An occasional purgative may also be given with advantage, preferably castor oil.

Warm baths, *particularly sulphur baths*, are said to greatly assist the recovery of the patient: they should be given for from fifteen to thirty minutes daily.

Lead-paralysis is best treated by the use of the faradaic or galvanic currents. The latter current is particularly indicated as long as the former fails to produce muscular contraction. The applications should be made daily for a duration not to exceed five minutes.

When *galvanism* is employed, a labile application of the cathode, with the anode upon the nape of the neck or the sternum, yields the best results.

I have employed the static spark to these muscles with very marked benefit in many cases. The instrument must be of considerable power if employed for this purpose.

Frictions to the part, *systematic massage*, and *passive exercise* are valuable adjuncts to electrical applications.

Mechanical appliances may be employed to the paralyzed limb, with advantage to the patient, in some instances.

ARSENIC-PARALYSIS.

This form of paralysis generally occurs after acute poisoning. It usually develops rapidly, and, like other toxic paralyses, is probably to be attributed to a multiple neuritis which has been induced.

It may be distinguished from lead-paralysis by the fact that the lower limbs are more apt to be affected than the upper, by the rapid

atrophy which occurs in the muscles paralyzed, and by the presence of marked sensory disturbances.

Violent pains usually precede the paralysis and atrophy. Among the sensory disturbances noticed, *numbness*, *analgesia*, and *formication* are prominent.

Symptoms of *incoördination of movement* have been observed to follow arsenical poisoning.

The condition of the reflexes, and the alteration in the electrical excitability of the muscles are similar to those observed in lead-poisoning.

Treatment.—This is similar to that already described in connection with plumbism.

PHOSPHORUS-PARALYSIS.

This form of poisoning may be of the acute or the chronic type. In connection with both forms, monoplegia, paraplegia, and peripheral paralysis have been observed.

In the paralyzed parts, all forms of *subjective sensory disturbances* (paræsthesiæ, see p. 354) and *pain* may be experienced by the patient.

Little is positively known regarding the morbid changes which are induced in the nerves or nerve-centres by arsenic or phosphorus. The evidences of multiple neuritis are generally detected in all forms of toxic paralyses. Experimentation made upon dogs by Vulpian seem to show that changes in the peripheral nerves, as well as in white matter and anterior horns of the spinal cord, take place.

The history of the case will usually suffice for a diagnosis. The treatment of the symptoms is not unlike that employed in lead-poisoning.

ALCOHOLIC POISONING.

(*Alcoholism—Delirium Tremens.*)

The ingestion of a large quantity of alcohol produces effects which are modified by special idiosyncrasies of the patient, as well as by the habits of the patient in respect to alcohol.

ACUTE ALCOHOLISM.

The condition which is here described does not include simple intoxication, which is too often seen not to be recognized, but rather a state which is induced by the poisonous effects of very large quantities taken at once or gradually established after a prolonged and excessive indulgence.

"Delirium tremens," or acute alcoholic poisoning is most commonly observed after a prolonged debauch, or after an excessive indulgence in alcoholic liquors by one who has been an habitual drinker for a sufficient

length of time to seriously undermine the general health. It is claimed that it may also occur in habitual drinkers from the sudden cessation of the habit. This fact is probably attributable (if it really ever occurs) to a condition of system which is so thoroughly under the toxic influence of alcohol that its ingestion is no longer possible, because it is refused by the stomach. If this view be a correct one, the development of alcoholismus is due rather to the depressed systemic condition than to the stoppage of alcohol. It is well known that many habitual drinkers eat sparingly and irregularly, sleep but little, and undergo more or less exposure. These circumstances are probably conducive to a condition of physical weakness, of which this disease is but a manifestation.

Symptoms.—Preceding the actual onset of an attack, it is not uncommon to observe more or less tremor of the hands and tongue, and a marked unsteadiness in the upper limbs when voluntary movements are attempted. The muscles of the lower limbs and trunk are sometimes similarly affected, causing an unsteadiness of gait. As a rule, these phenomena are most marked on arising, and they tend to disappear to a greater or less extent after the patient has taken several drinks to develop an appetite for breakfast. The feebleness of gait in some of these patients and the unsteadiness of the hands and upper limbs are too frequently spoken of by such patients as "nervousness." Respecting this point, Hammond very aptly remarks: "From this feebleness, or paresis, the distance to paralysis is not great." It may indicate the beginning of multiple neuritis.

Prior to the onset of an attack *vomiting* is often a prominent symptom. The patient may also exhibit sudden startings and a peculiar watchfulness and distrust of his surroundings. Morbid fears of various kinds may be developed. The expression may be markedly altered, and the eyes may be peculiarly watery or red. Perspiration is sometimes excessive. The fingers may be observed in some instances to be more or less constantly employed in some aimless pursuit. Finally vertigo, headache, and confusion of ideas may be encountered in such patients.

As the *onset approaches*, these symptoms tend to increase in intensity, and other morbid phenomena appear. The face of the patient gives evidence of more or less alarm. This is frequently due to the fact that they experience illusions, hallucinations, and delusions which are of an alarming type. Many cases of suicide have occurred among this class of patients in their endeavor to escape from some horrible object which they believe is pursuing them. They see demons, snakes, lizards, cats, etc., about the room and menacing their safety. Sleep is no longer possible on account of these visions. The patient is liable to become more or less uncontrollable and to form bitter dislikes toward his friends, relatives, or attendants The temperature is generally elevated, although

the body may be bathed in a profuse perspiration. The pulse is markedly accelerated, small, and weak. The bowels are constipated. The patient talks incessantly concerning various illusions or delusions which disturb him. The pupils are small, and the retina may be congested in the region of the disk. Convulsions sometimes occur, usually of the epileptiform character. When these are developed, they are liable to be frequently repeated. They are a grave symptom, and death may occur in one of these paroxysms.

The *duration* of such an attack varies from three days to a week or more.

When the attack produces dangerous exhaustion, a low muttering delirium takes the place of the loud ravings of the patient. The fingers pick aimlessly at the bed-clothes. Gradually the pulse becomes almost too rapid to count and very weak. A state of coma develops. The patient then either gradually sinks or dies in a convulsive attack.

CHRONIC ALCOHOLISM.

This form of alcoholic poisoning differs from the acute chiefly in the absence of a marked onset and the characteristic phenomena of delirium tremens already described. In cities, it is more often encountered among the idle and wealthy classes than the acute form. These are prone to indulge in alcohol to great excess during the hours spent at the club, the gaming table, and social festivities.

Symptoms.—The manifestations of chronic alcoholism differ in individuals. It may be well to describe them under the following heads: (1) That in which tremor predominates. (2) The anæsthetic type. (3) The convulsive type. (4) The paralytic type. (5) The mental type.

Alcoholic Tremor.—This may precede an attack of delirium tremens in occasional instances. It has been already described, therefore, among the premonitory symptoms which occasionally indicate the approaching onset of the acute variety of alcoholism. It chiefly affects the hands, and is liable to be accompanied by unsteadiness of the legs and trunk—particularly in subjects who are victims to chronic alcoholismus. On account of this tremor, such patients are often prone to drop things from their grasp unless they constantly watch the object. When their eyes are withdrawn, the grasp is unexpectedly relaxed.

The muscles of the legs and trunk suffer from more or less *paresis*, as the condition progresses. I have repeatedly known such patients to lose in time their ability to walk without the aid of an attendant. For a while they get along by the support of a heavy cane.

Vertigo and *dimness of vision* are sometimes experienced as the result of chronic alcoholism. Various disturbances of sensation may be

developed, such as numbness, formication, analgesia, etc. These will be discussed under the next variety.

The paresis and abnormal sensory phenomena are probably to be attributed to morbid changes in the peripheral nerves. In many cases of this type, the pathological evidences of multiple neuritis are unmistakably present.

The Anæsthetic Type.—Occasionally, from the very onset sensibility may be perverted or entirely lost in chronic alcoholism.

The presence of anæsthesia may be limited to one lateral half of the body (hemianæsthesia). Hammond speaks of this condition as peculiarly characteristic of chronic alcoholism,—a view which he states to be in accord with the observations of Magnan and Virenque.

In addition to this loss of sensation, the same observer states that the sight of one eye, the hearing of one ear, and the functions of one nostril and one lateral half of the tongue are often abolished. He mentions one case cited by Magnan, in which the sensibility of the cornea was totally lost in one eye.

As a result of anæsthesia, the patient has imperfect conceptions of the "feel" of objects; and the sense of pain may be totally lost both in the skin and the muscles. These patients may thus be rendered personally unconscious of tests made with a view to determine their appreciation of touch, pain, or temperature.

A more or less marked *loss of motor power* invariably accompanies the abolition of sensation.

The Convulsive Type.—It is not uncommon to observe, in patients who are suffering from chronic alcoholism, convulsive seizures of an epileptiform character. These may occur as early symptoms, or they may follow some of the other symptoms already described. As a rule, they are usually accompanied by marked and permanent derangements of sensibility, and more or less paresis during the intervals between the convulsive attacks.

The *convulsive seizures* may or may not be accompanied by loss of consciousness.

According to Huss, the frequency of convulsive attacks tends to diminish as the condition of chronic alcoholism increases.

The Paralytic Form.—In connection with the previous types of chronic alcoholism, paresis has been mentioned as a symptom which is generally present. In some cases the loss of motility progresses to such an extent as to render locomotion impossible and to constitute true paralysis. One very marked case of this character has come under my observation.

In connection with the paralysis of the limbs, the muscles of the eye are occasionally observed to suffer from paralysis. In this way the

development of strabismus and ptosis is to be explained. Again, the muscles of the organs associated with speech and swallowing may be paralyzed.

The Mental Type.—In connection with chronic alcoholism, the mental condition of the patient is liable to be seriously impaired. Illusions and hallucinations are not uncommon, and the power of intellect and will are generally lessened in a marked degree.

Emotional manifestations, chiefly those of a "sorrowful type," are very frequently observed. A large proportion of these subjects are prone to weep easily over imaginary troubles, and to exhibit a disposition to greatly exaggerate their business cares and perplexities. Those who are naturally inclined to be vicious may be rendered still more so by this form of alcoholic poisoning. It has not been my experience, however, to encounter any exhibitions of mental disturbance in this class of patients that would justly create alarm. They are far more apt to become extremely irritable, peevish, and lachrymose. They are very liable to take offense easily, and to misconstrue unintentional acts of their friends and companions as personal slights and insults.

Suicidal tendencies may occasionally be observed in these patients as the results of melancholia or imaginary fears. One of my patients, who was a very rich man, was constantly haunted by the fear of beggary, and was with difficulty restrained from acts of violence to himself.

The capabilities of the patient for mental application or for accurate reasoning in respect to the ordinary affairs of life is liable to be seriously impaired. Their sense of comprehension is often very much blunted, and their judgment is often seriously in error. Hammond states that the memory is the first faculty which shows impairment, and that the sense of right and justice which the patient may have had in health is often weakened or destroyed. This author lays stress upon the fact that lying, stealing, and even serious outrages without known provocation, may be committed by these patients; and he regards the existence of any motive for such acts as extremely infrequent among this class of sufferers.

Hallucinations and delusions in chronic alcoholism, as in the acute form, tend to assume a peculiarly painful and distressing character. These patients are frequently the victims of fear of personal violence either to themselves or those to whom they are attached. There are exceptional instances, however, where the delusions assume a more pleasant character.

Prognosis.—In the acute form recovery usually occurs, provided the attacks have not been frequent. Convulsions in this form give a grave aspect to the case. A failure of the digestive organs to assimilate food after the symptoms have subsided to a greater or less extent is also an unfavorable sign.

The chronic form may be recovered from when the symptoms are not of a severe type, and when the patient's appetite for liquor can be effectually controlled.

Treatment.—An attack of delirium tremens demands active mediation to induce sleep, and careful attention to the nourishment of the patient. It is my custom to administer thirty grains of the bromide of potassium and ten grains of chloral, combined with ten drops of the tincture digitalis, to a patient suffering from delirium tremens, every hour or two as the circumstances seem to demand, until sleep is induced. The digitalis is added as a preventative of heart-failure, which sometimes occurs from chloral-poisoning. When sleep is not induced by this prescription within five or six hours, a hypodermic injection of a quarter of a grain of morphine may be given and repeated in two hours.

The monobromide of camphor has been recommended in doses of four grains every hour for several hours. I have had no personal experience in its use.

Sometimes the administration of capsicum by the stomach acts charmingly in inducing sleep. A teaspoonful of the tincture may be given at a dose.

During the attack, the patient should be carefully watched by an attendant in order to guard against danger to the patient, as they are very prone to jump from the window, or do themselves some personal violence in their efforts to escape from the imaginary objects which haunt them. It is well to remove from the room all articles of china and other utensils which might be employed by the patient as a weapon. One of my patients committed suicide by breaking the handle from a heavy earthen pitcher and cutting his throat with its ragged edge.

The patient should be nourished at regular intervals with milk, beef-tea, gruels, and other liquid foods. During convalescence, quinine, iron, and strychnia may be indicated.

The *treatment of chronic alcoholism* consists in an entire cessation of alcoholic liquors. If the bowels are deranged by constipation or diarrhœa, they should be regulated.

When insomnia is a prominent symptom, the bromides alone, or a combination similar to that used in delirium tremens, may be employed.

The *oxide of zinc*, in doses of from one to three grains three times a day, has been highly recommended in this affection. Hammond suggests the use of bromide of zinc in doses of two grains three or four times a day. This drug may be given in solution with water or syrup. The dose should be gradually increased (up to four or even six grains at a time) as rapidly as the stomach will permit.

The *infusion of digitalis*, in doses of a tablespoonful three times a

day, is often indicated to increase the power of the heart and indirectly to stimulate the action of the kidneys.

Electrical applications to the affected muscles may be administered daily for from five to ten minutes. The faradaic or galvanic currents may be employed, and also the static current in the form of spark or static insulation. I have great faith in static sparks as a curative agent in alcoholic tremor and paresis. When anæsthesia exists the faradaic current is best applied by means of the wire brush.

Careful *regard to the diet* of these patients should be observed. All highly seasoned food should be countermanded, and large quantities of milk should be taken by the patient. I have great faith in the curative effects of an exclusive milk diet in these cases. Three quarts of milk a day are amply sufficient to nourish an adult without a particle of solid food. The addition of a slight quantity of lime-water or some of the admirably made preparations of pepsin may assist in its digestion in those exceptional cases where milk is not well tolerated by the stomach.

MERCURIAL POISONING.

(*Hydrargysm.*)

Mercury may be taken into the system not only by the stomach and intestines, but also through the skin and lungs.

This drug is often taken to excess in the form of blue pill and calomel. Mercury is used extensively in some of the manufacturing arts, such as the silvering of looking-glasses, the making of artificial flowers, the manufacture of bronzes, some of the photographic processes, certain forms of dental work, etc. Some of the cosmetics for the removal of facial eruptions contain mercury. I have observed several cases of mercurial poisoning from an injudicious use of the vapor of mercury in a bath given for medicinal purposes. This is particularly apt to occur when calomel is employed, or when the vapor is inhaled into the lungs.

Symptoms.—In chronic mercurial poisoning, tremor is peculiarly apt to be developed.

When *salivation* is thus produced, the gums will appear very much swollen, sensitive, liable to bleed easily, and more or less separated from the teeth. The breath is extremely fetid, and the teeth are apt to become loosened from their sockets. The patient complains of a metallic taste in the mouth. In severe cases, the tongue becomes enormously swollen, often to such an extent as to protrude from the mouth. The saliva flows in a stream, so that the patient has frequently to hang the head over a bowl, which catches it as it escapes from the mouth. The countenance may be pale or livid. Severe nose-bleed sometimes occurs. The physical strength is very rapidly lost, and marked mental debility may be exhibited.

In some cases, *caries and necrosis*, especially of the lower jaw, with ulcerations of the soft parts, may develop.

Finally, convulsions of the epileptiform type and paralyses of various parts of the body have been reported.

Diagnosis.—The symptoms of this form of poisoning can hardly be mistaken for those of any other disease.

The tremor, the peculiarly fetid odor to the breath, the characteristic appearance of the gums, the loosening of the teeth, the swelling of the tongue, the enormous increase of saliva, and the bone-complications frequently encountered, are met with in no other condition.

It is claimed that the diagnosis may be rendered certain when the symptoms are not fully developed, by the administration of the iodide of potassium to the patient in large doses for two days, and then subjecting a few drops of the urine on a bright copper plate to a drop of hydrochloric acid. If mercury is present, a bright metallic spot will be found on the copper plate.

The history of the patient will usually reveal the fact that he has been exposed to the poisonous influences of mercury.

Treatment.—During an attack of salivation, tannic acid may be employed as a gargle, with very great relief to the patient. The administration of the iodide of potassium in doses of from 15 to 30 grains three times a day, aids in the elimination of the poison.

When *mercurial tremor* is the prominent symptom in the case, the use of the iodide of potassium is often followed by a very rapid cessation of that symptom.

The administration of iron, quinine, and strychnia in tonic doses may prove valuable as adjuncts in the treatment of this form of poisoning. The patient should be warned against further exposure to mercury.

BROMISM.

The bromides which are chiefly administered as drugs in the treatment of most of the functional nervous diseases comprise those of potassium, sodium, calcium, lithium, ammonium, zinc, and camphor.

The extent to which these drugs are employed probably exceeds to-day that of any known class in the treatment of nervous diseases; hence, I feel it my duty to raise my voice here in protest against the view too commonly held by practitioners, that these bromide preparations are harmless, and that they are indicated in every functional nervous disturbance whose cause may be obscure.

I believe that more harm has been done to the human race by the bromides than good. In epilepsy, for example, the continued administration of very large doses of the various bromide salts unquestionably assists in bringing the mental state of the patient to a deplorable

condition, in depressing the digestive functions, in weakening the general muscular tone, and in seriously delaying (if it does not sometimes prevent) the recovery of the patient in many instances.

I have known of several epilepties where recurring dislocations of the shoulder could, in my opinion, be attributed to a relaxed state of the muscles, which had been induced by excessive and long-continued administration of the bromides. I have seen a very large number of patients precipitated into a condition closely bordering upon imbecility from the same cause.

Personally, I have for some years avoided the use of bromides as far as possible in the treatment of functional nervous diseases; and I believe that by so doing I have greatly added to the comfort and health of my patients. The prevention of convulsive nervous phenomena by the continued use of a drug whose poisonous effects are well-known and generally recognized, cannot be considered in any light as a cure. It is a question to my mind if epileptics, for example, do not improve as much under a well-regulated diet from which nitrogen is eliminated as far as possible, and the use of other drugs than the bromides whose poisonous effects are far less marked if not totally absent, than by the use of bromide-salts, which tend in time to render their mental condition, if not their physical, a deplorable one in many cases.

Symptoms.—The poisonous effects of bromine may be developed in some adults and often in children, from very small quantities of the drug.

The evidences of its poisonous effects may be manifested in a variety of ways. I shall consider some of these effects in detail.

Excessive somnolence is an early symptom. The patient may sleep or remain in a drowsy condition not only during the night, but also during the day.

A *feebleness* in the legs and in the arms, may tend to develop. This is shown by a marked alteration in the gait and a loss of the grasping power of the hand.

The *heart's action may be markedly weakened*, and the pulse proportionately accelerated. Frequently the skin is rendered cold and clammy, and the pupils may be occasionally widely dilated and rendered insensible to the action of strong light.

Pustular eruptions may develop upon the skin—chiefly upon the face, neck, and trunk. Boils and carbuncles have been known to follow the abuse of the bromides.

The *digestive organs* are *very often seriously disturbed.* This is shown by a coating, unnatural dryness, and occasionally an excessive soreness of the tongue. The bowels are usually constipated and the breath has a fetid odor. The mucous membrane of the mouth and fauces may become covered with aphthous patches, and show a markedly con-

gested state. A similar condition is probably induced in the mucous lining of the respiratory passages; as evidenced by accelerated respiration, cough, and the physical signs of bronchitis.

The mucous lining of the pharynx, larynx, and the bladder may lose its normal sensibility. Specialists on the throat employed this agent often for the purpose of rendering the introduction of instruments into the pharynx and larynx possible without exciting nausea or efforts to vomit.

In severe cases of bromism, walking often becomes impossible; the patient lies in a deep stupor; the sphincters are no longer controlled; the heart's action is excessively weakened; lung complications are developed; and death may occur.

Treatment.—When bromism has developed, the administration of the drug should be immediately discontinued. Digitalis may be given to increase the power of the heart and thus to excite the action of the kidneys. The patient should be stimulated judiciously, and nourished at regular intervals. Tonics may be given during convalescence.

TETANUS.

(*Trismus—Lock-jaw.*)

This disease is characterized by an exaggerated excitability of the motor and reflex functions of the spinal cord. It manifests itself by convulsive seizures of a tonic character. Its course is usually a rapid one. Consciousness remains intact throughout the disease.

Etiology.—Tetanus is commonly divided into four varieties, viz., the traumatic, rheumatic, toxic, and idiopathic. To these varieties some authors add the hysterical, inflammatory, intermittent, and that of the newly born (trismus neonatorum).

Lock-jaw, or "*trismus*," is considered by some authorities as a distinct disease. With this view I am not in accord, since spasms of the jaw-muscles are nearly always combined with spasms of the trunk and limbs. I regard typical trismus as but a modified form of true tetanus.

The *traumatic variety* is the most frequently encountered. It seems to occur in some regions more than in others. The atmosphere of some localities apparently renders any wound particularly dangerous on this account. Contused and torn wounds about the hands and feet, especially if they injure nerves or tendons, are peculiarly liable to be followed by tetanus. It is thought by the laity that a wound inflicted by a rusty piece of iron is particularly apt to cause this condition.

Tetanus may follow immediately after a wound, or it may develop after a lapse of several days or weeks.

Among the *predisposing causes* to tetanus may be mentioned certain psychical influences, irritation of the wound, epidemic influences, atmospheric conditions, concussion of the head or spine, pyæmic poisoning, etc.

The exciting wound may be extremely insignificant in some cases. Tetanus has been known to follow the sting of a bee, the pulling of a tooth, the piercing of the ear, ulcerations, etc.

Among other forms of injuries received of a more serious character, which have resulted in tetanus, may be mentioned the wounds of the surgeon's knife, lacerations of the cervix uteri, the exposure of an abraded mucous membrane (as in the puerperal state), and accidental traumatisms of various kinds.

A variety of tetanus is not uncommonly observed between the fifth and twelfth days of life, which is known as "*trismus neonatorum.*" It may be attributed in many cases to disease of the umbilicus, fæcal stasis, and mechanical irritation of the medulla oblongata from strong extension of the head during delivery.

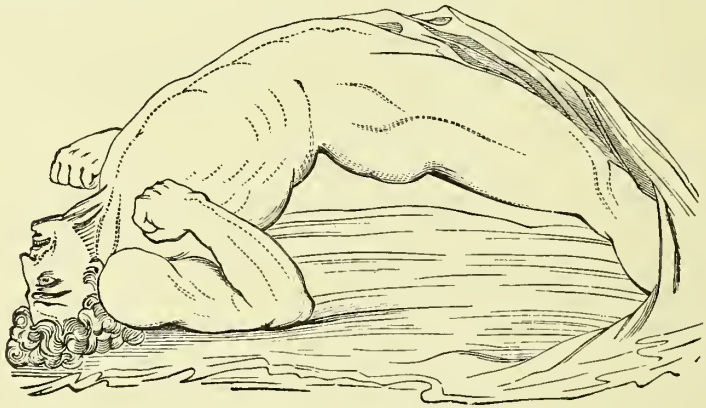


FIG. 127.—A MARKED PAROXYSM OF TETANUS. (After a drawing by Sir Chas. Bell.)

The *rheumatic variety* of tetanus is a rare form of disease. Many authors of to-day express a doubt as to whether genuine tetanus is ever dependent upon exposure to cold or dampness, or an excess of lactic acid in the blood.

The *toxic variety* of tetanus may occur in certain forms of poisoning by drugs, chiefly from an overdose of strychnine or picrotoxine. It is stated by some authors that clonic muscular spasms are more often noted than the tonic in this variety of tetanus.

The *idiopathic variety* of tetanus comprises all cases in which no exciting cause can be discovered. In many such cases the presence of an epidemic, due probably to some atmospheric influences, exists.

Symptoms.—After a wound, the onset of tetanus is usually preceded by more or less pain in the region of the wound, or an unnatural appearance of the injured part. These pains frequently shoot along the course of some adjacent nerve. Local symptoms are often accompanied by a

peculiar restless, excited, or anxious condition of the patient. Insomnia may be a marked symptom.

As the onset of the disease approaches, movements of the jaw are associated with more or less pain and stiffness, as are frequently also movements of the neck and attempts at swallowing. The first signs of muscular spasm generally appear in the face and in the back of the neck. Later, a similar condition develops in the muscles of mastication, in the pharynx, and in the muscles of the back and limbs. In many instances, the extremities are but slightly affected.

The *facial expression* of tetanus is characterized by a peculiar wrinkling of the forehead, an expansion and elevation of the nostrils, and an attitude of the mouth which exposes the teeth, giving to it the expression termed the "risus sardonicus" because it resembles that of laughter. Respecting this peculiar condition of countenance, Eichhorst very aptly says: "The features express the most antagonistic feelings. While the upper half of the face has a cheerful though tired expression (due to diminution in the size of the palpebral fissure), the lower part is sad, and the mouth has the expression of one who is sobbing. On account of the uncovering of the teeth, the expression has been compared to that of laughing."

The ability to open the mouth is partially or completely lost by a tonic state of the masseter muscles. This interferes seriously with the ability of the patient to take food. Infants, when attacked by tetanus are unable to retain the nipple between the jaws. Any attempt to open the mouth is so strongly resisted that a danger of fracture or dislocation of the lower jaw might be created before the spasm could be overcome. The nutrition of the patient is, furthermore, embarrassed by a similar state of the muscles of the pharyx, in some cases. The tortures of hunger and thirst may therefore constitute an important part of the sufferings of the patient.

During the *paroxysm* the head is drawn forcibly backward, and the muscles of the back distort the trunk so that the patient may rest upon his shoulders and hips. In occasional instances, the trunk may be bent toward one side (pleurosthotonus); or the body may be drawn forward toward the thigh (emprosthotonus). Cases have been reported where the trunk-muscles have rendered the spinal column rigid without distortion.

The *respiratory movements* are seriously embarrassed when the diaphragm and the intercostals are affected. Under such circumstances extreme cyanosis may develop, and death may occur from a lack of ability on the part of the patient to breathe.

The *muscles of the limbs* may participate, to a greater or less extent, in the tonic rigidity so commonly observed in the trunk and neck.

Flexion of the upper limbs and extension of the lower are generally observed. The contracted muscles stand out with great prominence beneath the skin, and, in severe cases, the patient may be raised from the bed so that he rests upon his head and heels.

In very exceptional instances the third cranial nerve, in addition to the seventh and ninth, may be affected and cause a strabismus. Eye-symptoms in tetanus are justly regarded as of evil import.

The *frequency and duration* of tetanic spasms vary. During sleep, they generally tend to subside or entirely disappear. In some cases, distinct paroxysms are observed; beginning, as a rule, with clonic contractions, which gradually tend to assume the tonic type. In other cases, the tonic contraction of muscles is persistent. Such a condition may continue for days without intermission.

The reflex excitability of the spinal cord is very markedly exaggerated in some cases. The slightest noise or disturbance of the patient in any way may excite a paroxysm.

The force of the tonic contractions of muscles has been known to break off the teeth, and to fracture and dislocate the long bones. During a paroxysm, the development of fibrillary twitchings in the rigid muscles may occasionally be observed.

The onset of a paroxysm is looked forward to with great terror by the patient on account of the extreme pain which is generally experienced while the muscles are thrown into rigidity. In severe cases, the patient is unable to obtain sleep or to quench his thirst or hunger. Occasionally, delirium sets in late in the disease; but, as a rule, the mind of the patient is perfectly clear. Profuse sweating is apt to occur during the paroxysms. The sensibility of the skin is unaffected, as a rule, and little, if any, febrile excitement is usually observed.

Diagnosis.—The only disease which could be confounded with tetanus is spinal meningitis. The character of the spasms, the stiffness or complete fixation of the jaws, the facial expression observed in tetanus, and the difficulty in swallowing produced by spasms in the muscles of the pharynx are sufficient to distinguish tetanus from an inflammatory affection of the spinal meninges.

Prognosis.—The duration of tetanus, as well as the character of its symptoms, vary in individual cases. A case is reported where death occurred in a negro fifteen minutes after the accident; while, on the other hand, the duration may extend over a period of weeks or months. Paralysis and paresis have been known to occur as sequelæ of tetanus.

The mortality in infants is enormously large. In adults, the severity of the symptoms and the exciting cause of the attack materially modify the prognosis. In a large percentage of cases it is unfavorable. A long duration of the disease without rise in temperature or marked insomnia

indicates a favorable termination. The idiopathic variety is more often recovered from than the traumatic. The prognosis is also more favorable, in case the attack be of the traumatic type, when a long interval elapses between the exciting injury and the development of tetanic phenomena.

Treatment.—The indications for treatment are modified by the exciting cause and the symptoms. When an open wound exists, perfect cleanliness and antiseptic dressings should be employed. The patient should be placed in a darkened and quiet room; and every precaution should be taken to avoid a recurrence of the paroxysms.

The patient may often be nourished by means of a tube passed behind the last molar tooth and through the pharynx into the stomach. When this is impossible, the patient may be put under the influence of ether or chloroform and a tube introduced between the teeth; or, when that is impossible, through the nostril into the stomach.

Among the drugs which have been highly recommended, the following may be mentioned: Calabar bean, cannabis indica, conium, anæsthetics, chloral and bromide of potassium in large doses, gelsemium in doses of from ten to forty drops every two hours, paraldehyd in doses of from thirty grains to two drachms during the twenty-four hours, belladonna, and aconite.

Ice-bags and counter-irritants may be applied to the spine. Ether spray and leeches have also been employed over this region.

Certain surgical procedures have been recommended. Among these, stretching of the nerve leading to the wound may be mentioned as having given satisfactory results in a few cases. Accumulations of pus may demand an incision in the region of the wound, and care should be taken to free the wound from the presence of any foreign substances which may have entered at the time of the accident.

Hammond has carefully analyzed the results of treatment in reported cases which were obtained by most of the drugs enumerated; and by surgical procedures in typical cases of tetanus. The conclusions of this author go to show that some cases apparently recover rather in spite of drugs than by their direct aid. Perhaps it may be said that large doses of chloral, combined with large doses of the bromide of potassium, give as much relief to the patient as any known treatment. The application of ice-bags to the spine, combined with the internal administration of half a grain of the extract of cannabis indica every two hours, may be attended with good results.

I have been awaiting for some time an opportunity to test the effects of *strong static sparks* upon a patient in a paroxysm of tetanus, on account of the remarkable effect which such an electrical application seems to exert upon muscular contracture. Thus far I have been unsuccessful in obtaining an opportunity of this kind.

TREMOR
AND
PARALYSIS AGITANS.

Intermittent clonic spasms of the muscles, of a persistent type, are not infrequently observed in connection with any condition which increases the susceptibility of the patient to central or peripheral irritation, or renders the action of the nerve-centres more or less imperfect.

Etiology.—In the human race, the presence of persistent tremor usually indicates a condition of irritation in the nerve-centres.

This condition may be dependent upon a morbid lesion; such as inflammatory processes, tumors, sclerosis, changes in the vessels, etc. It may also be apparently due to some excessive mental strain or excitement in subjects predisposed to nervous disturbances; hence, it has been known to follow sorrow, fright, prolonged anxiety, protracted mental application, religious excitement, etc. Again, degenerations of the cord or brain, which may or may not follow or accompany an attack of paresis or paralysis, may cause persistent clonic spasms in the limbs or trunk. Finally, nervous and easily-excited individuals, who inherit the so-called "neuropathic predisposition," may develop tremor in some of its forms simply as the result of peripheral irritation. In this class of patients, my experience leads me to believe that eye-strain exists more frequently as an exciting cause than is generally suspected. (See my remarks respecting chorea and other convulsive diseases.)

It has been noted, in connection with some of the toxic neuroses, that tremor is often induced by the introduction of poisons into the general system. Under this class of causes, chronic alcoholism, the opium habit, excessive use of tobacco, and mercury or lead-poisoning may be prominently mentioned. In such cases, the morbid evidences of a multiple neuritis can often be detected.

Finally, the weakness of old age is often manifested by the development of persistent tremor.

PARALYSIS AGITANS.
(Parkinson's Disease—Shaking Palsy.)

This form of tremor was first carefully observed and described by Parkinson in 1817. It is characterized by a tendency to steadily progress both in extent and severity, and to be accompanied by evidences of enfeebled motor power or general paralysis.

Morbid Anatomy.—The anatomical changes which occur in this disease are unknown. By some observers, morbid changes (pigmentation, hypertrophy, etc.) have been detected in the ganglionic cells of the

brain and spinal cord, chiefly in the pons, medulla, and Ammon's horn. Our knowledge of these changes is, however, so imperfect that we are as yet forced to consider this disease as a purely functional one.

Etiology.—Among the causes of this affection which have been mentioned by authors of note, the following conditions seem to predispose to it: Exposure to cold, injuries, excessive fright, prolonged mental excitement, excessive venery, certain infectious diseases, heredity, excesses in alcohol, gout, etc.

In many of these subjects, an inquiry into the health of the patient's blood-relations will often show that corea, epilepsy, hysteria, insanity, neuralgia, or phthisis has existed in some branches of the family. Personally, I regard the question of heredity as one which should always be thoroughly investigated. It tends to shed much light upon the possible factors which aid in creating diseased conditions.

Among the traumatic cases, injuries to nerve-trunks are more frequently followed by this condition than other forms of accident. This disease has, however, been known to follow the opening of a carbuncle, a severe burn, and other peripheral injuries.

Symptoms.—Shaking palsy is very rare before the age of twenty years. It usually occurs in advanced age, chiefly between forty and sixty years of age. It seems to affect both sexes equally.

The onset of this disease is usually so gradual (except in cases where it originated after a severe fright) that the patient cannot accurately locate when the first symptoms were observed.

Prior to the development of the onset, the patient may have suffered from marked insomnia, unnatural irritability, temporary weakness of the limbs, vertigo, neuralgic pains, and paræsthesiæ.

The disease first manifests itself by slight trembling in the muscles of the fingers and hands; later in the muscles of the arms; and still later in the legs. It is claimed that the right arm and the corresponding leg are markedly affected before those of the left side. While this may be generally true, it is not in my experience an universal rule. Sometimes the muscles in the head and face may be attacked with tremor; in which case the tongue is also liable to be involved.

The *trembling of the muscles* is present during repose; and does not seem to be affected by voluntary movements. They are intensified, however, by any form of mental excitement. Although at first the patient may partially control them by an act of will, the tremulous movements soon become uncontrollable, and persist even during sleep. In the latter stages of the disease, the tremors tend to become excessively violent. Cases have been reported where the floor as well as the bed have been set in vibration by the shakings of the patient. It is needless, perhaps, to state that the ability to perform delicate movements

of the fingers is rapidly lost; and that the patient sooner or later may become unable even to dress or feed himself.

The development of *paresis* and a *rigidity* of certain muscles generally accompany or follow the onset of tremor. In exceptional cases, paresis may precede the development of tremor.

An *abnormal attitude of the fingers* is very frequently observed in connection with this disease. Sometimes the fingers assume the attitude commonly employed in holding the pen; again, the constant movement of the thumb against the extended fingers gives to the patient the appearance of rolling a ball in each hand between the thumb and forefinger; finally, the fingers may assume an attitude characterized by flexion of the first phalanges, and over-extension of the second phalanges, and a flexion of the third phalanges. The latter deformity very closely resembles that observed in connection with arthritis deformans.

As a rule, the upper extremities are flexed also at the elbow, and the elbow is carried away from the chest. The admirable drawing made by Charcot (Fig. 128) illustrates this point.

The *gait of these patients* is very peculiar. It has been described on a preceding page. As they trot along, the knees are apt to rub against each other and the feet to become crossed. In some reported cases the friction made by the rubbing of the knees has given rise to eruptions, ulcerations, and even gangrene. Deformities of the toes and pseudo-talipes may be detected in these patients. The head is thrown excessively forward, causing a prominence of the seventh cervical spine; and the body is also inclined very markedly forward. A prominent author speaks of this attitude as one which indicates to the observer a danger to the patient of tumbling head over heels. When these patients attempt to walk, if it can be called walking and not running, they experience great difficulty in suddenly stopping or in turning around suddenly to either side (*propulsion*). I have known them to catch hold of objects in order to stop when called upon to do so. Occasionally, when a patient is requested to walk backward, he continues to do so more and more rapidly, until he falls or is stopped by an attendant. This condition is known as that of "*retropulsion.*"

There are *no evidences of atrophy* or *trophic changes* in the muscles of the limbs, even in the advanced stages of the disease. The sensory functions are seldom affected, nor is the electrical irritability of the muscles materially altered. In some cases an increase of the deep reflexes has been observed.

A *peculiarity of facial expression* is commonly observed in victims of paralysis agitans. It is described as comparable to a mask, because there is so little play of the features. Very often the mouth is kept wide open and the saliva drools constantly from the lips. Articulation

and deglutition may be embarrassed. The voice is generally high pitched and tremulous. The speech is liable to be slow and monotonous. Disturbances of vision are observed in many of these patients, which are exhibited by a difficulty in following the lines on a printed page.

The *bladder and rectum* are not usually impaired. In many cases, constipation exists.

Diagnosis.—The tremor of this disease differs from that of *multiple sclerosis* in the fact that it is not excited by voluntary movements; that

Fig. 128.—Paralysis Agitans, or Shaking Palsy. (Charcot.)

it is not accompanied by symptoms of oscillation of the eyeball; that sensory disturbances are wanting; and that it begins usually in the upper limbs. The characteristic gait is also a prominent feature in paralysis agitans.

From *chorea*, it can be told by the marked regularity of the spasmodic movements; the persistence of the tremor, even during sleep; the violence of the shaking; and the fact that voluntary movements do not increase the tremulous condition of the limbs.

From the *tremor of alcohol*, *mercury* and *lead*, the history of the patient would render the diagnosis easy, and the characteristic gait would confirm it.

Prognosis.—No immediate danger to life is created by this disease. From the period of its onset, many years may elapse before death occurs from exhaustion. In some cases the tremor may exhibit intermissions; but, as a rule, the disease tends to steadily advance. Whenever the tremor has not become general, or when the spasmodic movements are comparatively slight, recovery has been known to occur in exceptional cases.

Treatment.—The preponderance of medical testimony goes to show that this disease is seldom permanently benefited by treatment. Hammond states, however, that he has succeeded in curing eight out of twenty-five cases, and partially curing five others. Such a remarkable result might lead to the suspicion that all of these cases were not of a typical character. The treatment which this author adopted comprised the employment of the primary current to the spinal cord, sympathetic nerve, and the affected muscles, together with the internal administration of strychnia and phosphorus. Four out of the eight cases, which were entirely cured by this treatment according to the observations of the author cited, recovered within two months.

The first point in the treatment of all cases is to remove the exciting cause, if possible, provided it can be ascertained. The value of the constant current in this disease is not regarded by all authors as highly as by Hammond. Static electricity has yielded satisfactory results in a few reported cases.

Internal administration of Fowler's solution of arsenic yielded favorable results in the experience of Eulenberg.

Hyoscyamine, in doses of one-twentieth of a grain three times a day, is stated to have produced a very marked improvement in some cases reported by Oulmont. The phosphide of zinc, in doses of one-twelfth of a grain, prepared in pill form and administered after eating, has also been highly recommended.

As a rule it is safe to consider most cases of this disease, that have become well established, as practically incurable. The tremulous condition may be improved by the various remedies suggested in many cases; but, after the cessation of treatment, the patient is apt to relapse to his former condition.

Due regard should be paid to the avoidance of all mental excitement and excessive fatigue of the muscles. The diet of the patient should be as nutritious as the digestive powers of the patient will allow of.

The persistent insomnia which these patients are apt to suffer from may be controlled by the use of a combination of chloral and the bromide of potassium.

POSTERO-LATERAL SPINAL SCLEROSIS (GENERIC ORIGIN).

(Friedreich's Disease—Hereditary Ataxia—Generic Ataxia.)

The term that I have selected by which to designate this peculiar form of ataxia that shows a marked tendency to affect family groups has been lately suggested and strongly advocated by Dr. W. Everett Smith, in an admirable essay on its nomenclature.*

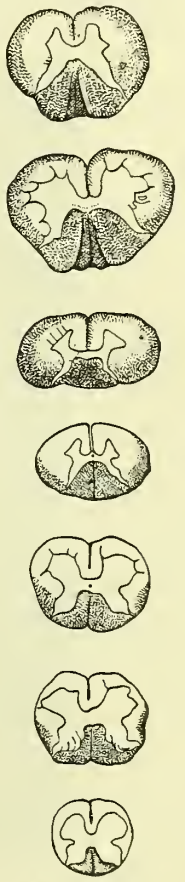


FIG. 129.—MORBID APPEARANCES PRESENTED IN HEREDITARY ATAXIA. (After Friedreich.)

Friedreich was the first, in 1861, to describe certain cases which he had encountered as those of a type of ataxia which seemed to select its victims from lines of direct consanguinity. He believed that they presented special points of distinction from the clinical history of ordinary locomotor ataxia. Since his first description of these cases, others have published a sufficient number of similar cases, accompanied by the results of an autopsy, to warrant our acceptance of this condition as a distinct disease.

Among those who have contributed to the literature of the subject may be mentioned Friedreich, Carré, Topinard, Immermann, Rütimeyer, Carpenter, Kellogg, Power, Hammond, and Smith. Respecting some of the cases reported, however, there is apparently ground for doubt as to whether they are to be regarded as typical representatives of generic ataxia.

This form of disease was observed by Friedreich to affect nine persons in three families; Carré, of Paris, reported seven cases in one family; eleven cases in two families have been described by Immermann and Rütimeyer (as quoted by Eichhorst); and six in one family have been observed by Dr. W. E. Smith. I have in my possession some beautiful sections of the spinal cord taken from a patient who was so affected (in common with several members of his family) by Dr. W. Everett Smith (late of Framingham, Mass., but now of Boston), and kindly presented to me by him.

Morbid Anatomy.—In this disease, a degeneration (more or less systematic) not only of the posterior columns of the spinal cord and

* *Boston Med. and Surg. Jour.*, March 1, 1888.

the posterior nerve-roots, but also of the lateral and anterior columns, has been observed. Our knowledge, however, rests upon only eleven autopsies. In the spinal sections which I possess, the crossed pyramidal and the direct pyramidal fibres of both sides are somewhat affected; although not to so great an extent as the columns of Goll and Burdach. Unlike in true locomotor ataxia, females seem to be somewhat more frequently affected than males. Although it seems

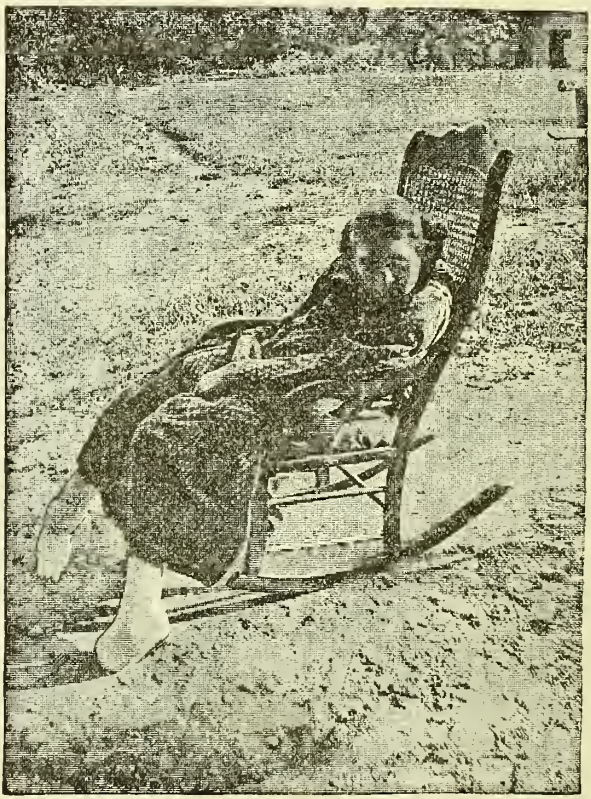


FIG. 130.—PHOTOGRAPH OF CASE. (Reported by W. E. Smith.)

to be a systematic disease of both the motor and sensory columns of the spinal cord, the question is still unsettled regarding the proper classification of this affection.

Symptoms.—These may begin during childhood, usually between the fourth and seventh years, or at the period of puberty. The lancinating pains (which are typical of true ataxia) may be wanting; or, in some cases, they may develop late in the disease. From the very onset, marked incoördination of movement or a sense of weakness is developed

in both the upper and lower extremities. In some cases reported, the ataxic symptoms have been confined to the upper limbs. The disturbance of coördination is very rapidly developed. It may affect the movements of the head, the muscles of speech, and possibly those of the eyeball. Changes in the pupil and defects of vision are not commonly observed,—a fact which is a marked contrast to the course commonly observed in tabes dorsalis.

Fig. 131.—Photograph of Case. (Reported by W. E. Smith.)

According to most observers, the tendon reflexes are usually abolished. The sensory functions are less disturbed than in ataxia of adults; although tactile anæsthesia is generally developed. The muscular sense is usually retained to a greater or less extent. The plantar reflex may often be retained, in spite of the complete abolition of the knee-jerk.

Some time after the symptoms of incoördination appear, paralysis and contracture tend to develop in some cases, these symptoms are usually more marked in the lower limbs than in the upper. The patients

cannot walk, as a rule; and free movements of the hands or arms may become impossible.

Bed-sores do not occur in this disease. The sphincters are not affected. The mental faculties seldom exhibit any marked impairment. The speech, however, tends to become of a drawling kind, and often more or less unintelligible.

Vaso-motor disturbances may be observed; chiefly in the form of polyuria, salivation, and excessive sweating.

The two foregoing figures (Figs. 130 and 131, taken from photographs made by Dr. Smith), together with the two sections of the cord of one of these patients (Figs. 132 and 133), which have been very accurately drawn by Dr. H. P. Quincy, present some very interesting points in relation to this rare affection.

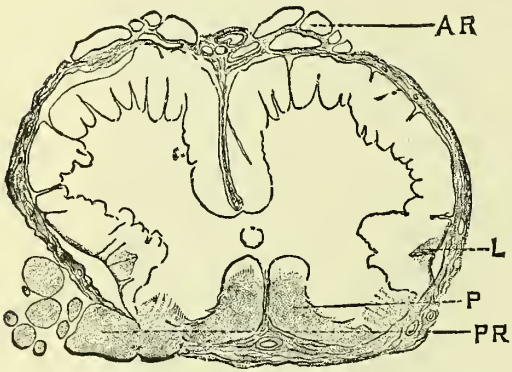


Fig. 132.—Section of Dorsal Region. (After Smith and Quincy.) AR, Anterior nerve-roots (healthy). L, Lateral column (diseased). PR, Posterior nerve-roots (diseased). P, Posterior columns (diseased). C, Central canal.

The *involvement of the posterior spinal nerve-roots* was very marked in this case. The anterior nerve-roots were found to be perfectly healthy. The clubbed feet shown in the photographs of two members of this family constituted a very marked deformity.

In Dr. Smith's six cases, a sense of *weakness* and *uncertainty of movement of the limbs preceded by the development of ataxia and girdle-pains; and likewise abnormal sensory phenomena* in five of the subjects. Incoördination of movement was an early symptom.

In two cases "wrist-drop" developed. In the two fatal cases reported, death was preceded by symptoms of spinal meningitis and myelitis.

The *movements of the head* were attended with "irregular oscillations," which were aggravated by voluntary attempts at movement of any part of the body.

The *limbs became greatly distorted* by progressive atrophy and contractures. Attacks of extreme dyspnœa and impending collapse were frequently noted.

In the case shown in the photographs, epileptic attacks had occurred frequently for fifteen years prior to the published notes upon her condition. She had developed right lateral curvature with kyphosis; also a

talipes equino-varus; and extreme flexion of the hands, wrist, forearm, and head. She could neither pick up objects from her lap nor hold them when placed in her grasp.

Differential Diagnosis.—This disease might possibly be mistaken for lead-poisoning, cerebellar disease, cerebro-spinal sclerosis, lateral spinal sclerosis, amyotrophic lateral spinal sclerosis, locomotor ataxia, chronic myelitis, and progressive muscular atrophy.

Many of the tables given in connection with these diseases will aid the reader in making the necessary discriminations.

I take the liberty of quoting, however, in this connection, several paragraphs from a late brochure of W. E. Smith, respecting the clinical distinctions which seem to be regarded by him as justified by the somewhat scanty literature of this subject. He says:—

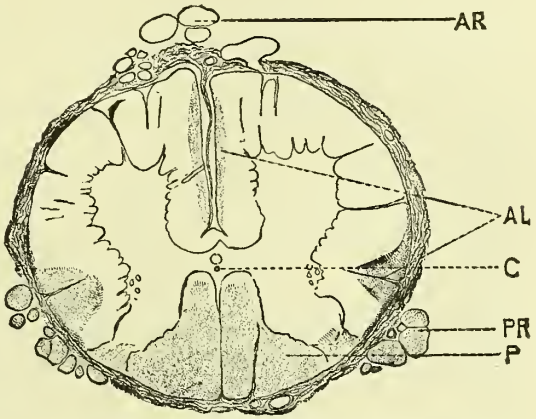


Fig. 133.—Section of Dorsal Region. (After Smith and Quincy.) AR, Anterior nerve-roots (healthy). AL, Antero-lateral column (diseased). PR, Posterior nerve-roots (diseased). P, Posterior columns (diseased). C, Central canal.

"Now, although it is true, as Ormerod has said, that there have been cases of Friedreich's disease, so-called, where paralysis of the lower limbs has been reported, 'in no case has it been observed until the disease had existed for a period of years, and in some very carefully observed cases there has been no paresis at all, but simply ataxia. Again, although in some cases diminution of sensibility has been noted, it is usually for the earlier periods in quite an insignificant degree, and in many cases every abnormality of sensation has been thoroughly excluded. We have, therefore, in this disease an illustration of ataxia pure and simple, apart from motor or sensory paralysis.'

"Clinically speaking, the difference between the classic locomotor ataxia and the so-called hereditary ataxia may be distinctively arranged into three general groups. In the first place, ordinary tabes does not run in family groups, while Friedreich's disease clearly does. Yet the cases are few where, even in the latter disease, a strict heredity can be claimed. It attacks, as a rule, members of the same generation in the same family, although occasionally successive, as well as simultaneous, outbreaks have been observed in the same family, as, for example, in

Carré's cases, where the mother of the seven ataxic patients, her brothers and sisters to the number of eight, and her mother were all ataxic; in Brousse's case, where the mother was ataxic; in one of Rütimeyer's cases, where a male ancestor five generations back was ataxic; and in my series of cases,* where the father of the five affected girls clearly developed the disease late in life. More often, however, a remarkable proclivity to other forms of disease has been observed in these family forms of ataxia: 'on the side of the nervous system, to chorea, paralysis, hysteria, mental affections, intemperance; on the side of general diseases, to phthisis.'

"Secondly, ordinary tabes is a disease of middle age or of later life, while the family form of ataxia develops usually in early childhood. Friedreich held that its development was connected with the changes of puberty, and that females were particularly prone to it; while Ormerod believes that the onset of an acute disease may have a possible influence in its production. The disease is unquestionably due to a lack of proper development of the nervous elements of the cord and their consequent atrophy, and it is worthy of notice that the ataxic families are generally large; so that it may be that the rapid production of children may have caused an imperfect development of some of them. At any rate, enough cases have now been reported to show that sex has nothing to do with either the development or the occurrence of the disease.

"Nor do I believe that puberty is a potent or a common cause.† The majority of cases thus far reported began to show an evident lack of development at as early an age as five or seven years, and I very much suspect that if these cases had been more carefully watched, they would be found never to have been as steady in their movements as were other children. At the age, however, when they would naturally begin to romp and run, the parents may possibly observe that they cannot carry themselves as well as their playmates can, but think little of it perhaps, until the onset of an acute disease, or the extra demands which puberty makes upon the system has allowed the incoördination to develop to such an extent that it can no longer be unnoticed.

"Thirdly, the absence of sensory derangements in the hereditary forms of ataxia is of great diagnostic value. In typical tabes, as is well known, the lancinating pains are usually a prominent symptom, especially in the early stages, and are rarely absent throughout the entire history of the disease, while other sensory disturbances, such as numbness and local anæsthesia, frequently accompany the incoördination of movement. But in the majority of cases of family or 'hereditary' ataxia pain is conspicuous by its absence, until at least the disease is very far advanced; although it is true that cases have been observed, such as those

* *Boston Med. and Surg. Journal*, October 15, 1885, p. 361.

† *Ibid.*, February 16, 1888, p. 175.

of Carré and Dreshfeld, where it occurred as an initial symptom. The absence of the knee-jerk is, indeed, common to both of the forms of ataxia which we are discussing, but the curvature of the spinal column, which occurs so early and so markedly in the family form of the disease, does not belong to the history of locomotor ataxia. Another critical diagnostic symptom is the affection of the speech in the hereditary form of ataxia. 'From a mere drawl, stammer, undue confluence or undue separation of syllables, the affection may advance till speech becomes wholly unintelligible.'

"The highest level to which the hereditary disease extends is marked in its clinical aspect by the symptom of nystagmus. Bilateral and transverse in its action, it is observed only when the patient looks at or follows an object intently. Being a very late symptom, its absence in a given case need not tell necessarily against the diagnosis of the family form of the disease. Bed-sores and visceral disturbances never occur, as they so commonly do in locomotor ataxia.

"Seeing, then, how marked the contrast is between the two diseases, it is not in the least surprising that some authors should have denied altogether the existence of relationship between them. Thus, on the one hand, Hammond maintains that Friedreich's cases are not examples of primary disease of the cord at all, but of disease of the medulla or cerebellum, extending secondarily to the cord; while, on the other hand, Vulpian, Charcot, and Bourneville regard them to be simply a variety of disseminated sclerosis. Although the affection of speech and the nystagmus would seem to lend some plausibility to the latter theory, the morbid anatomy of the cases, as revealed by autopsies, must be our final test, and by this we find conclusively that the disease is spinal, and not cerebral, in its origin; that whatever cerebral degeneration there is is late and entirely secondary in its development; and that, moreover, the spinal sclerosis is systematic, and not disseminated, in its character.

"Nor does the suggestion of Erb that cases of tabes may be grouped under two types, the classical type and the type described by Friedreich, seem entirely appropriate when we come to look at the pathology, although the occurrence of forms apparently transitional between the two may give some reasonable support to the idea. Such cases are those reported by Carré, where there was a well-marked affection of the speech and a distinct heredity, yet where the disease began with pains and numbness in the legs and feet; and those reported by Dreshfeld, where there were pains from the onset, and no affection of the speech reported. In three of Friedreich's own cases, also, these early pains were noticed, and in Powers' case there appear to have been vomitings at an early period. Other aberrant forms are the two cases of Seeligmüller which Friedreich refused to recognize as of the same type as his

own cases, chiefly because of some mental peculiarities and the persistence of the knee-jerk."

Treatment.—The remarks already made when the treatment of locomotor ataxia was considered are equally applicable to this disease.

The only unexplored field that suggests itself to my mind relates to a question of its possible causation. The accurate determination of the presence or absence of some marked abnormality in the visual apparatus (the eyes themselves or the ocular muscles) of the patients so afflicted, and also of all of the members of the family who have escaped, might possibly shed some light upon a factor which, if present as a family defect, might act as a cause of serious impairment of the nervous energies of those who had thus far escaped any of the serious manifestations of organic disease.

The proclivity of other members of the families, into which this disease has entered, to functional nervous diseases,—such, for example, as chorea, hysteria, intemperance, mental disturbances, etc.,—would certainly justify us in investigating this point very carefully.

EXOPHTHALMIC GOITRE.

(*Graves' Disease—Basedow's Disease.*)

A peculiar combination of an abnormal irritability of the heart, a protrusion of the eyes from their sockets, and the development of an enlargement of the thyroid gland (goitre), has been considered as a distinct disease since the original publications of Graves and Basedow. The term "exophthalmic goitre" is now commonly used in speaking of this affection.

Morbid Anatomy.—The pathology of this disease is to-day, as it always has been, a subject upon which authors of note differ. Various morbid conditions in the brain, spinal cord, and sympathetic nervous system have been observed in connection with this train of symptoms. In some instances, however, no marked changes of a morbid character have been found after death. Many of the symptoms observed during life seem to point to some form of disturbance of the centres situated within the medulla, a view which is sustained by Pamas, Hammond, and others. Basedow considered this disease to be a manifestation of an abnormal state of the blood analogous to chlorosis. Piorry, Bouillaud, and others have attributed many of the symptoms to a compression made by the enlarged glands upon the vessels and sympathetic nerves of the neck. The hypothesis advanced by Stokes, that the symptoms depend primarily upon hypertrophy of the heart has to-day few, if any, supporters.

The symptoms of this disease are believed by many authors to depend primarily upon the morbid state of the sympathetic system. On the other hand, the opponents of this view bring forward many justifiable

objections to this conjecture, and urge that such a conclusion is not supported by pathological investigation in many cases.

The view seems to be gaining ground that we are justified in regarding this form of disease as the result of a disturbed action of the medulla oblongata, and possibly of some of the higher centres of the brain.

The prominence of the eyeballs is probably attributable to several factors. Among these may be mentioned an unnatural turgescence of the vessels of the orbit, an increase of the fatty tissues of the orbit back of the eyeball, and a degeneration or lack of power of the ocular muscles which allow the eyeball to bulge forward.

Etiology.—Among the factors of causation of this disease, undue excitement of mind and physical or mental over-exertion may be prominently mentioned. It has been known to follow blows upon the head. It may occur also in connection with any condition which tends to impair the quantity or quality of the blood; and it is not infrequently associated with evidences of hysteria and neurasthenia. A large proportion of such cases seems to occur among women, a fact which is explained by some authors as a result of the frequency of debilitating diseases, hemorrhages, and nervous depression in the female sex. It occurs most frequently between the twentieth and fortieth years of life.

Symptoms.—The first symptoms of this disease are usually noticed as a disturbed action of the heart, and an acceleration of the pulse of a very marked character (120 to 160 beats per minute). At first the heart's action is apt to be irregular only when the patient is excited or fatigued, but, later on, this is observed to occur during repose and independent of any known exciting cause. There are no physical signs of organic disease, but, in many cases, carotid pulsation and a "bruit" in the vessels of the neck may be detected. Occasionally, a systolic murmur and evidences of cardiac hypertrophy may be observed after the disease has existed for some time.

After a lapse of several weeks or months, the patient notices that the thyroid gland is gradually becoming enlarged. This enlargement, as a rule, is at first apparent on one side only. The arteries which supply the gland may often be seen to be more or less enlarged and tortuous, and a blowing murmur may be sometimes detected over the gland. The gland tends to gradually acquire an abnormal hardness. It may be seen later on to rise and fall in rhythm with the pulsations of the carotids.

Simultaneously with or following the enlargement of the thyroid gland, the eyes of the patient begin to be more or less prominent and to assume a peculiar fixed look that has been compared to the expression seen in the eyes of a bull. The size of the palpebral fissure is more or less increased by the protrusion of the eye, and, later in the disease, it often becomes difficult or impossible for the patient to close the lids over

the eyeball. The power of divergence of the eyes is generally more impaired than that of convergence. The upper lid participates imperfectly in the vertical movements of the eyeball. The cornea may lose its sensibility and appear more or less glazed and opaque. Ulcerations of the eyeball have been observed. The secretion of tears may be rendered excessive. An unnatural redness and swelling of the conjunctiva is at times observed. The power of vision and of accommodation is not generally disturbed.

In consequence of the enlargement of the thyroid gland, the voice may be altered and respiration may be somewhat embarrassed.

The general system is apt to be more or less deranged. A loss of appetite, dyspeptic symptoms, diarrhœa, marked emaciation, and chlorosis are not infrequently present in these cases. It is not uncommon to observe the development of dysmenorrhœa or a complete cessation of the catamenia. Unilateral sweating has been recorded in quite a large number of cases.

The course of this disease is an exceedingly chronic one. It may last for many years.

Diagnosis.—In those cases where the chief phenomena are not very pronounced, some difficulty may be experienced in forming a definite opinion respecting the character of the disease. Whenever the eyeballs are at all prominent on both sides, if the thyroid gland transmits to the hand of the observer when placed upon it a peculiar thrill, and the heart's action is accelerated and somewhat irregular, we are justified in suspecting the existence of this affection even in cases where none of these symptoms alone are very well pronounced.

Prognosis.—Cases of this character seldom prove fatal. Some of the later publications upon this subject seem to justify the statement that under proper treatment quite a large proportion of patients so afflicted may be cured. In almost every case, there is reasonable ground to anticipate marked amelioration of the symptoms.

Treatment.—The internal medication of these patients should be directed toward the improvement of the blood by iron; the quieting of the nervous symptoms by a judicious use of the bromides; the improvement of the heart's action by digitalis; and the contraction of the blood-vessels by ergot.

All of these ingredients may be compounded in one prescription, such as that recommended by Hammond:—

℞. Ferri pyrophosphatis, zinci bromidi . . . . . āā ʒj.
Digitalis tincture . . . . . ʒv.
Ergotæ ext. . . . . fʒiv.
M. Ft. Mist.
Dose, a teaspoonful three times a day.

In connection with this prescription, it is well to instruct the

patient to eat plentifully of animal food, and to indulge in moderation in malt liquors.

Hammond suggests that after this prescription has been taken for several weeks, it be changed for a combination of strychnia and phosphorus, or of the extract of nux vomica and the phosphide of zinc.

The *electrical treatment* of these patients is, perhaps, more important than the medicinal. A constant-current battery should be employed. The positive poles should be applied at the nape of the neck, and the negative poles connected with a medium-sized electrode should be placed in contact with the thyroid gland for about five minutes, and afterward stroked up and down the neck so as to influence the sympathetic cords and the pneumogastric nerves of either side. The latter application should not exceed five minutes. The strength of the current should be as great as the patient can comfortably bear during the application to the thyroid, and somewhat less intense during the labile applications to the sides of the neck.

Cases of cure by means of the galvanic current have been reported by Rockwell, Bartholow, and others.

Deep *injections of ergot and alcohol* have been employed in treating the enlargement of the thyroid gland, with results which seem to have been markedly beneficial. Hammond reports multiple injections into the substance of the gland of from twenty to thirty minims of the fluid extract of ergot daily. I have had no personal experience with this form of treatment.

MYXŒDEMA.

(*Cachexic Pachydermique.*)

This disease seems to consist of an abnormal condition of the skin, which is associated with a deposit of a mucoid substance and a degeneration and proliferation of the connective tissue.

Morbid Anatomy.—The changes observed in this disease are probably confined at first to the skin and the connective tissue which binds it to the muscles. The changes in the skin are most frequently observed in the face; prominently in the forehead, eyelids, cheeks, nose, and lips. Moreover, the fingers and toes, and occasionally the limbs and trunk, may also be affected. The mucoid deposit has been observed not only in the skin, but also in the central nervous system. By some authors it is believed that the primary changes are probably confined to the cells in the gray masses of the brain and cord, although the exact character of such changes is largely a matter of conjecture.

Etiology.—This condition occurs chiefly among women. It seldom develops prior to the period of puberty or after the fiftieth year. It is thought to be dependent upon pregnancy, lactation, parturition, exposure

to cold, anxiety, mental shock, atrophy of the thymus gland, sexual excitement, and the "neuropathic tendency."

Symptoms.—A peculiar swelling, which closely resembles œdema, is first noticed in the face. It becomes difficult to close the lids on account of their extreme thickening. The expression of the face tends to assume a coarse and animal appearance. The features become more or less stolid, and fail to express the emotions of the patient. The tears and saliva are secreted in excess in some cases. The mucous membranes which line the mouth, pharynx, digestive track, and larynx may also be markedly thickened. The voice tends to become more or less hoarse and nasal, and the articulation indistinct and monotonous.

When the upper extremities are affected, the hands are usually more or less enlarged and distorted. The fingers are rendered bulbous at their tips, and clumsy in their movements. The ability of the patient to write or sew is materially interfered with, and often entirely lost.

The feet may be affected in the same way as the hands, and the swelling of the skin may, in some cases, extend up the leg and even involve the trunk. The gait of these patients is rendered slow and laborious when the lower limbs are seriously involved.

The swelling which affects the limbs and the trunk differs from that of œdema (for which it might easily be mistaken) by the fact that pressure made by the fingers does not leave a pit after the pressure is removed, as in the case of œdema. The reason why "pitting" is absent is that the skin and the cellular tissue beneath it have a semi-solid consistence from the deposit of musin. The color of the skin has been compared to that of alabaster, or of a yellowish wax. The bodily temperature is not elevated. In many cases reported it has been somewhat lower than normal.

The secretion of perspiration and sebacious material is often markedly decreased, and the skin may appear to be unnaturally dry and more or less wrinkled. Occasionally the hair is lost. The nails may become furrowed and extremely brittle.

These patients are apt to suffer from a sense of coldness in the affected parts. Other forms of subjective sensations, such as numbness, the creeping of ants, the feeling as if the parts were asleep, tingling, etc. These are less common than the sensation of coldness. Occasionally the skin may present a livid color or isolated reddened patches.

The *mental condition* of these patients is more or less affected as the disease progresses. They may exhibit somnolence, apathy, a loss of memory, and inability to converse as intelligently as before the attack, or to solve the simplest mathematical computations. Delirium and hallucinations may also develop. An indifference to the surroundings and a peculiar slowness of speech in answer to questions propounded to

the patient are frequently observed. These symptoms may be attributed in many cases to the fact that the brain and spinal cord participate in the changes observed in the skin.

The *digestive functions* of these patients are usually disturbed to a greater or less extent. Especially in the latter stages of the affection, we are apt to encounter constipation and dyspeptic symptoms.

The course of this disease is progressive, and essentially chronic. Its average duration is said to be about sixteen years.

The *special senses* are not infrequently impaired. The patient may state that objects are seen with a blurred outline, or as if surrounded with a halo. In some cases, double vision has been observed, together with a slowness of movement of the pupils to the effects of light. The sense of hearing may be diminished, and the smell and taste may be very seriously affected. These symptoms may be explained by alterations in the mucous membranes of the nose, throat, and mouth.

Diagnosis—This disease might possibly be mistaken for general œdema. The presence of aluminuria (which sometimes exists in connection with myxœdema) might tend to further mislead the physician regarding the diagnosis of this affection. The absence of pitting upon pressure over the swollen parts, the clubbing of the fingers, and the peculiar distortion of the features, are so diagnostic of this disease that it can hardly be confounded with the œdema which is observed in connection with kidney disease, arsenical poisoning, or diseases of the right heart that seriously interfere with the return of blood from the veins.

A condition of body, known as "*scleroderma*," in which there is an actual hypertrophy of the skin, might be mistaken for this affection. In scleroderma, however, the surface of the skin is hard, and a peculiar "sense of tightness" exists in the affected parts. We do not encounter, moreover, in scleroderma abnormal mental conditions, nor any permanent reduction in the temperature of the body. Besides, it is encountered at a much earlier age than is myxœdema, cases being seldom observed after the thirty-fifth year, according to Hammond.

Prognosis.—Myxœdema may terminate fatally, although in most cases its progressive course covers so many years that death is apt to supervene from some intercurrent affection. There is little hope in any case of arresting the disease.

Treatment.—It is well in these cases to support the general health by the use of mineral and vegetable tonics, and the judicious employment of electricity, massage, and hot-air baths. In spite of the fact that medication seems to exert little if any influence upon the actual symptoms of the disease, the preservation of the strength of the patient and the regulation of the digestive functions may possibly postpone the development of the more serious manifestations which are apt to occur late in the disease.

SECTION VII.

ELECTRICITY IN MEDICINE.

SECTION VII.

ELECTRICITY IN MEDICINE.*

In the diagnosis and treatment of nervous diseases, no agent is more generally applicable than electricity. Its brilliant and often instantaneous effects and the prevalent belief among the laity that electricity is practically identical with the vital forces of the human body have conduced largely to the general use and *frequent abuse* of this important agent.

Thousands of electric batteries are sold yearly by the various manufacturers to persons both in and out of the medical profession. Many who buy them are utterly ignorant of the principles of their construction, and equally so of the indications for their use. A very large majority of the medical profession possess only a faradaic battery or a magneto-electric machine. They employ such a battery upon every case which to their mind requires electricity. A few, in our larger cities, own a galvanic battery; but, as a rule, those who do so are unable to repair it themselves when the connections become oxidized or when it fails to act from a multitude of other causes. In my experience, it is very uncommon to meet a medical practitioner (outside of those who are specially interested in neurology) who thoroughly understands electro-physics and many important facts relating to the uses for which special forms of batteries are best adapted. I have deemed it wise, therefore, to include in this section a terse and practical statement of the more important facts which should be mastered before the treatment of disease by electricity is attempted, and to shed some light upon the forms of current which are indicated in the treatment of many of the nervous diseases commonly encountered. I shall include in these remarks some practical suggestions respecting the selection of batteries and the care of them. The uses of electricity in diagnosis, as well as its therapeutical properties in the treatment of nervous diseases, will be also presented in as concise a form as is comportable with clearness of statement.

PART I.

ELECTRO-PHYSICS.

Under this heading we shall first discuss the varieties of electric currents which may be produced (the faradaic, galvanic, magneto-electric, and static). We shall also consider the construction of a galvanic

* A part of this section has been issued as a separate volume by D. Appleton & Co., New York, 1886.

cell and its many modifications. It is important that physicians know the principles of construction of the various galvanic cells offered to the profession for medical uses, as well as the advantages and disadvantages of each as a part of a medical outfit. In the third place, the reader should be made familiar with many new terms which are commonly used to-day in electrical literature, and also the application of Ohm's law to electrical problems. Finally, he should acquire a familiarity with the many attachments to a battery. These are essential to its proper use, and their purposes should be well understood. Under this heading I shall give some practical hints respecting the selection, care, and repair of an electrical outfit for medical purposes.

VARIETIES OF ELECTRIC CURRENTS.

A few of the more important facts relating to this agent (which we are constantly called upon to employ in the treatment of various types of disease) should be thoroughly understood by all who intend to use it. Time will not permit of a detailed description of the different properties of electric currents. These can be acquired from any of the standard works upon physics. It is necessary, moreover, that such points as are presented here should be briefly and simply stated.

The GALVANIC CURRENT (called also "*voltaism*," the "*battery current*," and the "*constant current*") is one which is derived by chemical decomposition or heat from one or more pairs of elements directly. When the body is placed between two electrodes connected with such a battery in action, the current traverses the part of the body embraced between the electrodes before it returns to the battery—starting at the positive pole (the *anode*), circulating through animal tissue, and returning to the negative pole (the *cathode*). The polarity remains unchanged under all circumstances.

Muscular contractions are produced only when the current is closed or broken, or when its intensity is increased. A very weak current fails to produce muscular contractions.

In connection with the description of the tests employed in the diagnosis of nervous diseases, suggestions have been made by me which may be reviewed in this connection with advantage. (See Section II of this volume.)

By peculiar arrangements of the elements of a battery, the galvanic current can be modified as follows: (1) To produce heat (cautery battery); (2) to insure chemical changes in living tissues (electrolysis); and (3) to aid in many of the mechanical arts, such as electro-plating, electric lighting, telegraphy, etc.

The FARADAIC CURRENT (called also the "*induced*" or "*interrupted current*") differs from the galvanic in that it is an induced current of

high tension, which is *produced by the magnetizing and demagnetizing of a bar of soft iron or a bundle of soft-iron wires by means of a galvanic current.*

The circuit of the generating cell is made to pass through a coil of insulated wire, known as the "*helix*," which surrounds the iron to be magnetized, *but it does not itself pass to the electrodes and thus to the patient.**

When the current of the generating cell passes through the helix, the soft iron is magnetized and draws the interrupter in contact with it.† This breaks the circuit and demagnetizes the iron. The interrupter is then returned to its former place by a spring. This step reconnects the generating cell with the helix, and again allows the iron to be magnetized. The interrupter is again drawn in contact with it. Thus the current is constantly broken and restored by a simple device known as the "interrupter," or "automatic circuit-breaker." An *induced current within the iron core* of the helix is thus produced. This is the current which passes through the electrodes to the patient.

Much ingenuity has been shown in the construction of the "interrupter" of a faradaic machine. It is very desirable that *slow and rapid interruptions* may be produced at the will of the operator. If a machine only insures rapid interruptions, the slow interruptions can be effected by the use of an "interrupting electrode."

The power of producing electrolysis, and some other chemical properties peculiar to the galvanic current, are wanting in the faradaic.

Never attempt to combine a galvanic and a faradaic battery. Separate cells should be employed for each, as the faradaic battery requires a cell

* This is a point which cannot be too strongly impressed upon the minds of the profession. Its accuracy can be readily proved. If the binding-posts of the primary coil of a faradaic machine be united by means of a large copper wire, the current generated in the galvanic cell which runs the interrupter will pass through the wire rather than through the helix which surrounds the iron core (because the wire affords less resistance). The interrupter will then remain stationary, as the iron core is no longer magnetized. Again, the interrupted or faradaic current has no chemical properties. This would not be the case if the current of the generating cell passed to the binding-posts of the faradaic machine. Finally, the external resistance of the human body is far in excess of that afforded by the helix, and this alone would prevent the galvanic current of the generating cell from traversing the animal tissues (the circuit of the greatest resistance).

It is not uncommon for agents of the various manufacturing companies to show a prospective purchaser of a faradaic machine the galvanic cell which works the "interrupter," and to endeavor thus to leave the impression that a galvanic current (as well as the primary and secondary faradaic currents) can be conveyed by a faradaic machine to a patient. Such a statement is untrue, and, if made, indicates either ignorance or dishonesty. Subsequent diagrams will render the mechanism of a faradaic machine intelligible to the reader.

† The expression "in contact" is not strictly correct. The interrupter never actually touches the iron core, because its magnetic action ceases before it reaches it.

of greater capacity than those used in portable galvanic machines. They may be placed in the same case, but each should be perfectly independent of the other. If a battery is designed for transportation, it is best to have one of each rather than two combined in one case.

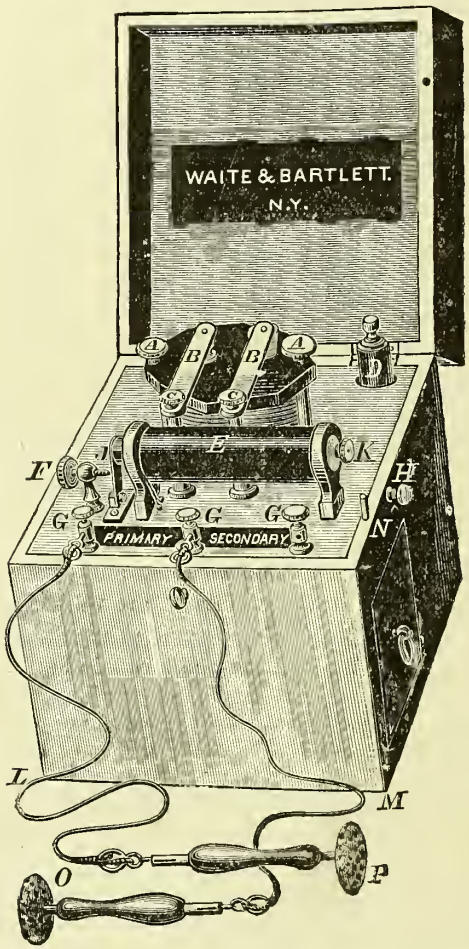


FIG. 134.—ONE OF THE MANY FORMS OF FARADAIC MACHINE.—*BB*, connecting rods attached to the elements of the exciting cell; *D*, a drip-cup, in which the zinc element is placed when not in use (it should contain mercury); *E*, primary and secondary coils; *F*, adjusting screw for the interrupter (*J*); *G*, binding-posts; *K*, plunger; *L*, *M*, rheophores; *O*, *P*, electrodes. The next figure will explain the action of the different parts. The faradaic instruments of different manufacturers vary more or less in their mechanical devices and perfection of workmanship, but the principle of all is the same.

The *faradaic current is an alternating current*,—*i.e.*, one which goes in opposite directions at each make and break of the circuit. It is strongest when the current is broken. These facts are not generally recognized by the profession. The polarity changes with each interrup-

tion. The so-called "cathode" of a faradaic battery is felt the strongest by the patient.

The helix of a faradaic machine is usually surrounded by a secondary coil of wire, known as the "*secondary helix.*"

This coil has no *connection with the elements of the generating cell.* The current produced within it is induced by the passage of the current (formed within the generating cell) through the "primary helix," which magnetizes and demagnetizes the iron core. It is therefore called the "*secondary current.*" It has high tension, is alternating, and is employed in telephonic lines, chiefly on account of its high intensity. It

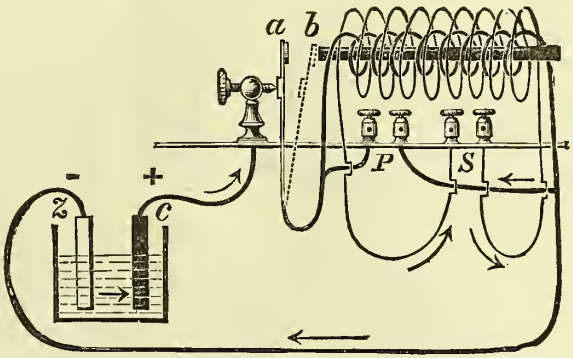


FIG. 135.—A DIAGRAM DESIGNED BY THE AUTHOR TO ILLUSTRATE THE CONSTRUCTION AND ACTION OF A FARADAIC MACHINE.—*Z*, zinc element; *C*, carbon element; *P*, binding-posts for the primary coil; *S*, binding-posts of the secondary coil; *a*, the interrupter when the circuit is passing to the helix; *b*, the interrupter when the circuit is broken. The screw (shown in contact with *a*) allows of the adjustment of the interrupter to the bundle of soft-iron wires within the primary helix, thus making the interruptions fast or slow at the will of the operator. The patient is connected with the battery in action by means of cords attached to the binding-posts at *P* or *S*. These cords are not shown in the diagram, but are shown in Fig. 134. The arrows show the direction of the currents. The zinc is marked as the negative element (—) and the carbon as the positive (+) element of the battery. Note that the wire of the primary coil is represented as coarser than that of the secondary; that the secondary coil has no connection with the elements of the cell; that the current going to the primary binding-posts is generated by the iron core, and is not that which originates in the galvanic cell; and that the interrupter has a small piece of platinum soldered upon it where it comes in contact with the screw, so as to prevent oxidation at that point. Patients feel the current made by the "break" more than that from the "make" of the circuit; hence, one electrode apparently gives a stronger current.

is modified in strength by regulating the amount of the secondary coil which overlaps the primary—the smaller the extent of the overlap, the weaker the current. A sliding tube of metal is sometimes made to pass over the primary coil, or between it and the primary coil. This accomplishes the same results as if the helix was movable.

The primary and secondary coils of a faradaic machine are made of wire of different thickness and length. Many of those sold are poorly constructed. They are the most important features of the instrument, and should be made with the greatest care and of the best materials. A fine finish of brass mountings and varnish does not always indicate good workmanship in the coils themselves.

Induced currents develop in the individual coils of the wire forming the primary spiral (as well as in the iron core which it invests) and also in the secondary helix. These currents are of no therapeutical value without the iron core, as they lack sufficient intensity.

If the secondary helix is composed of very fine wire, the current induced within it is extremely painful. The number of coils and the thickness of the wire selected for the primary and secondary helix should be graduated to a proper relation to each other and the electromotive force of the generating cell employed.

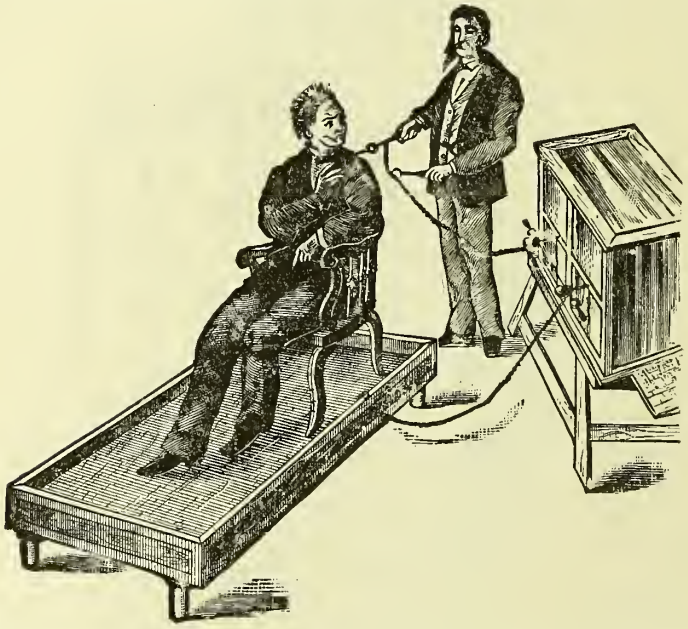


FIG. 136.—A STATIC MACHINE IN USE.—The "direct spark" is here represented as being drawn, *i.e.*, the patient being charged positively and the electrode being connected with the negative pole of the machine. No Leyden jars are employed in this form of administration of static electricity. The "indirect spark" is more commonly employed than the direct, one pole being attached, in this case, to the insulated platform, the other being grounded by a brass chain, and the electrode being grounded by a chain attached to a gas-pipe, a water-faucet, or the like.

The term "*primary current*" is often used as synonymous with the galvanic. It is incorrectly applied by some authors to that faradaic current which is induced by the magnetizing and demagnetizing of the soft-iron core of the helix.

STATIC ELECTRICITY is derived from friction. A revolving plate, or by preference several plates, of glass may be employed as a generator. Static electricity is sometimes called *franklinism.*

This form of current has high tension, but it possesses few, if any, chemical properties. When a patient is charged with it, it is necessary

that the *chair upon which he sits should be insulated* by glass or rubber under the legs. When highly charged, sparks may be elicited from the body of the patient through the clothing.

Static electricity will be quite fully discussed, as to its physics as well as a therapeutical agent, in subsequent pages.

Experimentation has shown that this form of electricity is accumulated upon the periphery of the object charged (as explained in all works upon physics). It apparently does not permeate very deeply below the surface.

The MAGNETO-CURRENT (called also the "*dynamic current*") is derived from a *permanent* or *electro-magnet, in front of which an armature is made to revolve.* The armatures are composed of a core of soft iron wound with insulated wire, and the currents produced are formed within them by *breaking the lines of magnetic force.*

The stronger the magnet, the more rapid the breaks made in the current by the revolving armatures, and the greater the number of turns in the spiral wire of the armatures, the more intense is the current.

This form of current possesses great electro-motive force or intensity. Currents of this kind are of an alternating character. By means of an automatic commutator (polarity changer), they may be carried, however, in one direction, and, when so, they assume properties similar to those of galvanic currents. Electric lighting, electro-plating, and many other similar applications to the mechanical arts, are to-day accomplished by means of dynamo machines at a minimum cost as compared with battery currents. They are practically obsolete as a machine for medical purposes, as the current is unsteady when hand-power is employed.

"Magneto-current" machines (which are turned by a crank when in use) are often sold to physicians. They are of little value. They cost as much as a good faradaic instrument, and are not to be compared with the latter. The current generated in both is practically the same, but it is irregular in point of strength in the magneto-current machine, and uniform in the faradaic instrument.

THE GALVANIC CELL—ITS VARIETIES AND THE GENERAL PRINCIPLES OF ITS CONSTRUCTION.

All substances have an electrical condition which is inherent or capable of being developed. This condition is known as the "POTENTIAL" of a body. The electrical condition of the earth (which may be regarded as fixed and as a reservoir without limit) is used as a standard of comparison of the "potential" of any given substance.

Those bodies from which electricity tends to flow toward the earth are known as "*positive bodies*" or bodies of "*high potential.*" They are designated by the plus sign (+). Those which tend to draw electricity

from the earth are called "*negative bodies*" or bodies of "*low potential*," and are designated with the negative sign (—). Almost every known substance may, therefore, be classified either as positive or negative under certain circumstances. Subsequent explanations will make this more apparent.

When we speak of the "RELATIVE POTENTIAL" of two bodies, we mean the *difference in degree* of the potential of each.

The bodies thus compared must both be positive or negative. One metal, for example, may have a potential seventy times that of the earth, and another one hundred times that of the earth. Both may be positive, yet one is negative as compared with the other. Two such metals have been happily compared to reservoirs at different levels (De Watteville). The tendency between two bodies of different

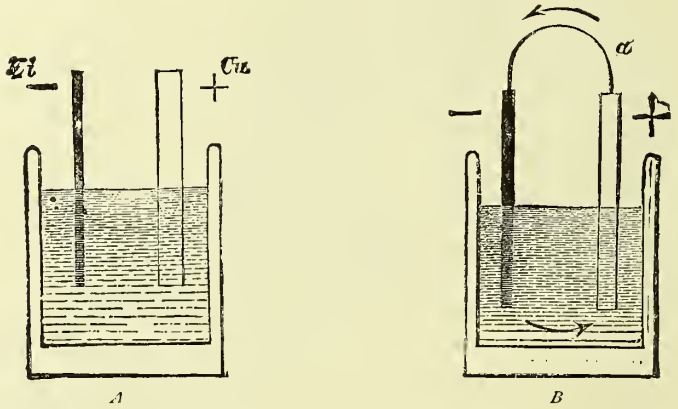


FIG. 137.—A SIMPLE GALVANIC ELEMENT. (After Erb.) *Zi*, zinc element; *Cu*, carbon or copper element. The fluid is composed of diluted sulphuric acid or a solution of some of the salts. In *A* the circuit is open, in *B* it is closed by a wire connecting the elements. The arrow shows the direction of the current outside of and within the cell.

relative potentials is for the current to flow from the body having the highest potential to that possessing the lowest potential, thus tending to establish an equilibrium between them. In a galvanic cell, the element most corroded by the fluid of the cell has the highest potential (positive element).

The difference in equilibrium between the "potentials" of two bodies regulates the intensity of what is known as the "ELECTRO-MOTIVE FORCE" of the bodies selected; because the want of equilibrium is the force which starts the flow of an electric current in all cases. The size of the elements has nothing to do with it.

The simplest form of a GALVANIC CELL consists of two bodies (whose potentials differ widely) immersed in a fluid which tends to excite chemical decomposition of one of the elements. Zinc and carbon are

commonly selected for the elements and dilute sulphuric acid for the exciting agent. The zinc is strongly acted upon by the fluid, while the carbon is not; hence, the zinc becomes the positive element and the carbon the negative. In most batteries of this type the zinc is covered with mercury (amalgamated), to render the action of the cell more uniform and to prevent local action upon the zinc. It also tends to preserve the zinc.

An apparently discordant fact should be remembered, *i.e.*, that the wire connected with the carbon of such a cell (the negative element of the cell) is the *positive pole* of the battery. This is because the electric current passes through the liquid from the zinc to the carbon, and back through the external circuit from the carbon to the zinc. (Fig. 137, *B*.)

When the elements of a cell are connected externally by a wire, a current of electricity flows continuously from the cell-elements through both the wire and fluid. This is known as a "*complete*" or "*closed circuit.*"

The RESISTANCE offered to the passage of the current from the *carbon to the zinc* is the "external resistance"; that between the *zinc and the carbon* is known as the "internal resistance" of the cell.

The INTERNAL RESISTANCE of a galvanic cell may be modified as follows: (1) by the *distance* between the elements; (2) by the *size of the elements;* (3) by the *intervention of some foreign body* (such as a porous cup) between the elements; and (4) by the *character of the fluid* in which the elements are immersed. The nearer the elements are placed, the larger their size, the more direct the passage of the current, and the better the conducting power of the fluid used, the less the internal resistance of the cell, and *vice versâ*. The internal resistance of a cell may vary between a fraction of an ohm and one hundred ohms, according to its construction and its excitants.

The EXTERNAL RESISTANCE is modified by the *length*, the *diameter*, and the *character of the conductor* employed. When any substance (such as the human body, for example) is placed between the electrodes, the resistance offered to the passage of a current by the interpolated substance must be added to that afforded by the conductors themselves. The resistance of the human body varies from 600 to 18,000 ohms. It is extremely low in subjects afflicted with general anasarca—probably because an excess of fluid renders the body a good conductor. The average resistance of the human body is not much above 2500 to 3500 ohms. The resistance afforded by the body is modified (1) by the saturation of the electrodes; (2) by the moisture of the surface of the body; (3) by the tissues through which the current is directed; (4) by pressure made upon the electrode; and (5) by many other factors which will be mentioned hereafter, among which the addition of salt to the

water in which the electrodes are moistened is a very important one.* In cautery batteries the external resistance is increased about $\frac{1}{2}\frac{1}{7}\frac{1}{3}$ for every degree centigrade when the temperature of the platinum wire is raised (De Watteville). Thus heat may be a factor in modifying the external resistance to be overcome.

The relative resistance of living tissues is represented by the following figures (100 being taken as the maximum): The eye, 4; muscle, 6; nerve, 10; cartilage, 20; tendon, 20; fat, 75; bone, 100; skin, 100. Thus, the eye offers only $\frac{1}{2}\frac{1}{5}$ the resistance afforded by skin and bone; muscle, $\frac{1}{1}\frac{1}{6}$; nerves, $\frac{1}{1}\frac{1}{0}$; cartilage and tendon, $\frac{1}{5}$; and fat, $\frac{3}{4}$. The epidermis, when dry, is practically a non-conductor of electrical currents.

Respecting this point, De Watteville happily remarks that "the human body may be compared to a vessel bound with a poorly-conducting material (the skin), unequally packed with non-conducting solid particles, the interstices being filled up with a saline fluid of fair conductive power. The parts most densely packed with solid particles are represented by the bones; those where liquid predominates, by the muscles. Between the two are found the nerves, viscera, etc."

Before we leave the discussion of the various forms of electric currents employed in medicine, it may be well to impress upon your minds some of the more important facts relating to faradaism and galvanism by means of a table in which the two are contrasted with each other. Such a table is not, to my knowledge, to be found in any work upon electricity. It may prove of service in many ways:—

THE FARADAIC CURRENT	THE GALVANIC CURRENT
Is an "induced current." Is produced by the *magnetizing and demagnetizing* of a core of soft iron.	Is due to *chemical decomposition* of one or more of the elements of a galvanic cell.
Its polarity changes with each "make" and "break" of the circuit.	Its polarity is constant. The negative element of the cell becomes the positive pole of the battery.
The current is an interrupted one.	The current is a continuous one.
It produces muscular contractions of an apparently continuous character, provided the interruptions are very rapid.	It does not produce muscular contractions, except when the intensity is increased or when the circuit is made or broken.
The polarity is inconstant, because the currents constantly alternate in direction.	Each pole has a special therapeutical action peculiar to it under all circumstances.

*In testing this point lately by means of a Brenner's rheostat, I found the resistance from right palm to left palm, in a boy of thirteen years of age, to be 17,500 ohms, when pure water was used and the electrodes pressed firmly against the skin of both palms. Adding a teaspoonful of salt to the water and again soaking the sponges reduced the resistance to 7500 ohms. This illustrates well the necessity for so simple a precaution when employing electric currents upon animal tissues. When very high currents are being used, especially in gynecological practice, salt should not be employed because it is apt to be decomposed by the current.

The Faradaic Current (*continued*)	The Galvanic Current (*continued*)
Is seldom administered by the so-called "polar method."	Is administered chiefly by the "polar method."
Wide separation of the poles intensifies the pain.	Separation of the poles does not materially intensify the pain.
The "secondary current" has greater penetrating power than the "primary current." Neither equals the galvanic current in this respect.	Has a remarkable power of penetrating animal tissues placed "in circuit."
It has no chemical properties. It may be modified by an automatic commutator, so as to throw its currents constantly in the same direction, as in a dynamo machine. In this case it possesses chemical attributes.	Possesses inherent chemical properties; hence its power of producing electrolysis, and its use in electro-plating, electric lighting, etc.
A galvanometer will show only one deflection, *i.e.*, the difference in strength of the "make" and "break" currents. This deflection is the same under all circumstances when the machine is in use. It does not, therefore, indicate the strength of the current conveyed to the patient.	Produces galvanometer deflections which are proportionate to the strength of the current employed.
There is no difference in the action of the poles.	The anode is the sedative pole; the cathode is the stimulating pole.
The faradaic instrument makes a "buzzing noise" when in action.	A galvanic instrument gives no external manifestation of activity, because it has no interrupter.

Electrical Units.—Before the construction of an electric battery and the modifications in such an apparatus necessary to produce special effects are considered, it is important that you familiarize yourselves with the various units of measurement employed in electricity, and their symbols. These are as follow:—

THING MEASURED.	SYMBOL.	NAME EMPLOYED FOR UNIT.
Quantity.	Q.	*Coulumb.*
Current.	C.	*Ampère* or *Weber.*
Electro-motive Force.	E. M. F. or E.	*Volt.* (contraction of Volta).
Resistance.	R.	*Ohm.*
Capacity.	K.	*Farad.* (contraction of Faraday).
Work or Energy.	W.	*Joule.*
Power.	P.	*Watt.*

A coulumb is the quantity that passes in one second of time against one ohm of resistance under an electro-motive force of one volt. We use this term as we do "pints" or "quarts" in speaking of fluids. One coulumb will decompose 92 microgrammes of water, and thus evolve 10.4 microgrammes of hydrogen.

An AMPÈRE is the current produced by one volt against one ohm of resistance. In medical practice, the milliampère is generally accepted as the unit of current-strength. An ampère will decompose .00142 of a grain of water.

A VOLT is the electro-motive force necessary to produce a current of one ampère against an ohm of resistance. It practically equals the electro-motive force of one Daniell's cell. We speak of a battery as of so many volts just as we designate an engine as of so many horse-power.

An OHM is the resistance necessary to allow of one ampère of current under an electro-motive force of one volt. It is equivalent to a piece of telegraph-wire one hundred metres in length and of a certain definite sectional area, or a column of mercury one square millimeter in diameter and 1.05 metre in height.

A FARAD is the capacity of a condenser which would contain a charge of one coulumb under an electro-motive force of one volt.

A JOULE is the amount of electric energy absorbed when a coulumb falls one volt. It is equivalent to about $\frac{1}{4}$ of the heat required to raise one gramme of water at 0.° C. one degree, or .7373 foot-pounds.

A WATT is the power developed by one ampère falling one volt. It is equivalent to $\frac{1}{7\,4\,6}$ of a horse-power.

The prefixes "*meg*" and "*micro*" denote million and millionth. For example, a megohm is one million ohms; and a microhm is a millionth of one ohm.

The names selected for the various units of measurement are taken from those of prominent electro-scientists (Ohm, Volta, Faraday, Ampère, and others).

OHM'S LAW.—We are now prepared to consider the law of electric currents discovered by Ohm, by which the intensity of a current that will result from any combination of cells may be mathematically computed, and many other electrical problems solved. It may be thus stated:—

$$\text{INTENSITY OF CURRENT} = \frac{\text{ELECTRO-MOTIVE FORCE}}{\text{RESISTANCE}\ (Internal + External)};$$

or, if expressed in symbols, $C \text{ or } I = \frac{E}{Ir + Er}$.

Now, in constructing a battery, the *object to be attained* must be first considered. A battery designed to produce heat (the cautery battery), for example, is not built upon the same plan as one designed for ordinary medical purposes.

Again, different cells (such as those devised by Daniell, Grove, Leclanché, Grenet, Bunsen, Smee, Hill, and others) possess special

advantages and disadvantages which have to be considered carefully before a decision is made respecting the one which should be employed.

Finally, the *number of cells*, the *arrangement of the elements*, and the *size of the elements* are problems to be determined with special reference to the purpose for which the battery is designed. These points will be touched upon hereafter.

It is important that a few facts be stated in the beginning respecting the more common methods of connecting and grouping galvanic cells. Subsequently, the different forms of cells employed by well-known manufacturers of electrical apparatus may be tersely described with advantage. Finally, the various attachments to an electric battery designed for medical purposes should be mentioned, and the uses of each briefly outlined.

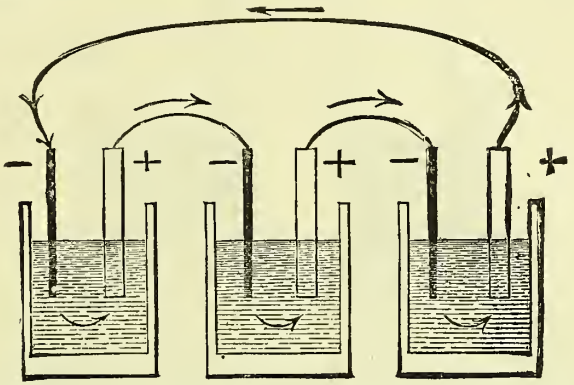


FIG. 138.—A COMPOUND CHAIN. (After Erb.) Three sets of elements are here connected "behind one another," or "in series." The direction of the current is shown by the arrows. The circuit of closure is effected by a wire, as in Fig. 137, *B*.

Let us suppose, for the purpose of illustration, that we have decided to use a certain number of cells (one of the numerous forms subsequently mentioned) in preparing a battery for medical use. How shall we connect them so as to best accomplish our purpose?

If we join the carbon and zinc elements together (using that form of cell for example), and continue to do so throughout the entire series of cells (Fig. 138), we have formed what is known as a "*compound circuit*" or an arrangement "in series." If we join all the negative or carbon elements together, and then the positive or zinc elements in a similar way, we have what is known as a "*simple circuit*" (Fig. 141). Finally, we may *divide the cells into groups;* then join those of each group in simple circuit; and afterward unite these groups as if they were single cells.

Now, what will the effect of each of these methods of combination have on the intensity of the current? Ohm's law comes into play in deciding such a problem.

We must first ascertain the internal resistance of the form of cell which we have selected for our battery.* We must know also the external resistance which we shall have to overcome in our proposed use of it. Finally, we must ascertain the electro-motive force of the elements of each cell.

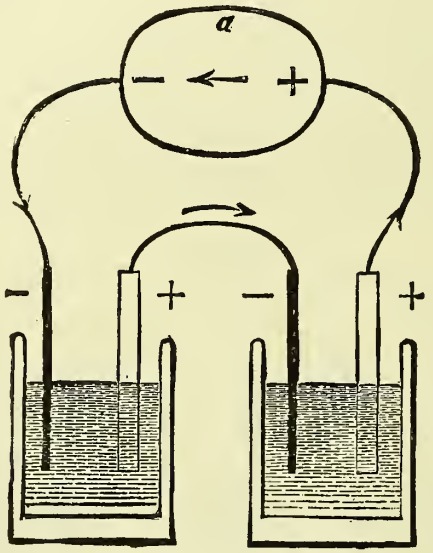


Fig. 139.—A Schematic Representation of the Introduction of a Human Body (*a*) into the Circuit of Closure of a Galvanic Chain. (After Erb.) + = the anode; − = the cathode.

Suppose, for example, that E = 1, Ir = 20, Er = 10. The current of each cell would then be expressed as follows:—

$$C = \frac{1}{20+10} = \frac{1}{30} = .033+.$$

* To compute the internal resistance of a cell or battery requires apparatus not generally owned by medical practitioners, *i.e.*, a coil rheostat, which may be confidently regarded as accurate, and a carefully calibrated galvanometer, by a standard maker. The rule given by De Watteville, and copied from him, apparently, by Amidon, would be simple if it were true. I have tested it again and again, and have personally discarded it as unworthy of credence. I have also had a professional electrician test it. He arrived at the same unsatisfactory results. The rule of De Watteville, to which I refer, is as follows: First note the needle-deflection of the cell or battery to be tested under a given resistance, then introduce sufficient additional resistance to reduce the recorded needle-deflection exactly one-half. The added resistance will equal the internal resistance of the cell or battery tested. The internal resistance of any cell can be computed with accuracy; but by a more complicated method, described in most of the standard works upon electricity. Most manufacturers can give the requisite information respecting the internal resistance of any cell used by them, and that resistance, multiplied by the number of cells employed, will equal the total resistance of a battery (the cells being united "in series," as shown in Fig. 138).

Now, if twenty cells of this kind be joined in "simple circuit," the elements have each been practically increased twenty times, and the internal resistance has therefore been decreased twenty times. The external resistance remains the same. We therefore have

$$C = \frac{1}{\frac{20}{20}+10} = \frac{1}{11} = .0909+.$$

If these cells be now arranged in "compound circuit," the electro-

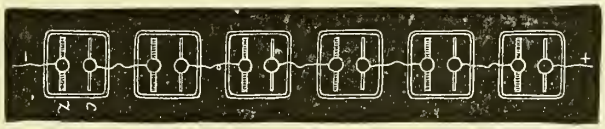


Fig. 140.—Six Cells Connected for Intensity. (After De Watteville.) *z*, zinc elements; *c*, carbon or platinum elements. This arrangement is known as "in series" or "compound circuit." It increases the "electro-motive force" of the battery.

motive force and the internal resistance will be increased twenty times. We should thus have the following formula:—

$$C = \frac{1 \times 20}{20 \times 20 + 10} = \frac{2}{41} = .048+.$$

Finally, if the cells were arranged in four groups of five each in simple circuit, we should have practically four cells with elements five times as large; hence, the internal resistance would be only one-fifth that

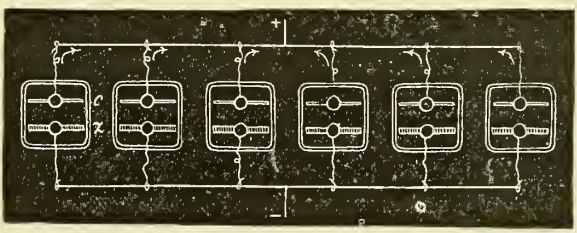


Fig. 141.—Six Cells Connected for Quantity, *i.e.*, "in surface," or in "simple circuit." (After De Watteville.) *z*, zinc elements; *c*, carbon or platinum elements. This arrangement does not affect the "electro-motive force" of the battery.

of a single cell and the electro-motive force four times as great. We should then have the following formula:—

$$C = \frac{1 \times 4}{\frac{20}{5}+10} = \frac{4}{14} = \frac{2}{7} = .285+.$$

Remember that the *electro-motive force means the difference in potential of the cell elements. It is therefore unchanged by their size.* A cup of water elevated one hundred feet will produce as much pressure through a pipe connected with it (provided that the cup be kept

constantly filled) as would a lake ten miles in eireumference, at the same elevation and similarly connected with the pipe. So it is with electro-motive force. The size of the elements will alter the *quantity* of electricity generated; but the electro-motive force of a cell or battery will remain undisturbed by increasing or diminishing the size of the elements. I frequently hear this remark made: "The cells are too small for medical purposes, are they not?" To this question I would reply that intensity of current and moderate quantity are to be aimed at in constructing a a medical battery.

The few illustrations which have been given show that the current-strength has been modified in each instance by the changes made in the arrangement of the cell-elements.

If, however, we took a higher external resistance (as would be required in a medical battery—say about 2500 ohms), we should find that the simple circuit arrangement made but little difference in the power of the current, while the compound circuit materially increased the current-strength. It is important to remember, therefore, that the *external resistance is an important factor in modifying the strength of the current*, and that all combinations of cells are not equally efficient.

The most useful battery for medical purposes is one which is planned with a view of making the internal and external resistances as nearly equal as possible.

When we wish to construct a battery for *ordinary galvanic treatment*, it is best to overcome the high resistances encountered by using a *large number of small cells, with a high electro-motive force, coupled in compound circuit,—i.e.*, "in series." The aggregate internal resistances of the cells never will exceed the external resistance furnished by the living tissues.

In devising a battery for *electrolysis*, the arrangement should be such as will secure simple intensity. The resistance to be overcome by the current in passing through small portions of the body seldom exceeds 100 to 500 ohms. A *small number of cells of medium size* (16 to 24 of Grenet's cells), coupled in compound circuit, will give us the desired ends and accomplish the best results.

A *cautery battery* requires *very large plates, placed closely together.* In the "Piffard battery" the zinc plates are perforated, and the elements are so arranged as to be mechanically shaken in the fluid while the battery is in action. I regard this as the best instrument of its kind. Its action is continuous, and the heat generated may be maintained at any desired temperature by one familiar with its management.

The Various Forms of Cells.—Human ingenuity has been strained to its utmost for nearly a century to devise an absolutely perfect galvanic cell. Space will only allow here of a brief statement of the

varieties now in common use. The construction of each and the peculiar advantages and disadvantages of each will be also tersely mentioned.

SPECIAL FORMS OF THE GALVANIC CELL.

All forms of galvanic cells may be classed under one of three groups, as follows: (1) one-fluid cells, with no depolarizer; (2) one-fluid cells, with a solid or liquid depolarizer; (3) two-fluid cells.

Each of these three varieties has many modifications, which are commonly named after the inventor. A few of each only need to be mentioned.

I. One-fluid Cells, with no Depolarizer.—The elements of this group are all immersed in a fluid to which nothing has been added to prevent polarization (*i.e.*, the formation of bubbles of hydrogen on the negative and of oxygen on the positive element of the cell during its period of activity).

Smee's Cell (1840).—Perhaps the best of this group is that devised by Smee. It consists of two zinc plates with one of platinized silver, suspended between the zincs, immersed in diluted sulphuric acid. The electro-motive force is about .47 volt.

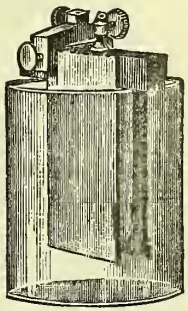


Fig. 142.—Smee's Cell.—This is a favorite with some manufacturers for a portable faradaic machine. In the author's opinion, it is far less satisfactory than Fuller's cell if the battery is a permanent one, or a Grenet cell if the battery is designed for transportation. It is active at first, but weakens rapidly from polarization.

Walker's Cell (1859).—Platinized carbon is used in place of platinized silver. It is cheaper than Smee's cell. E. M. F. = .66 volt.

II. One-fluid Cells, with Solid Depolarizers.—The best of this group is the cell devised by Leclanché.

Leclanché's Cell (1868).—The carbon element is packed in a porous cup, with the needle form of the black oxide of manganese surrounding it. This cup is then placed in a glass vessel, containing a rod of zinc and a solution of sal. ammoniac. The cup is carefully sealed to prevent evaporation and escape of its contents. E. M. F. = 1.48 volt, when the battery is not polarized.

Marié-Davy Cell.—Amalgamated zinc, acidulated water, carbon, and a paste of sulphate of mercury. E. M. F. = 1.52 volt.

Agglomerate Leclanché Cell.—The carbon is surrounded by plates of a special composition, which are bound around it by India-rubber bands. The internal resistance can be intensified by adding plates as desired. E. M. F. = 1.48 volt. The internal resistance with three plates = 1.8 ohm; with two = 1.4 ohm; with one = .9 ohm.

III. One-fluid Cells, with Liquid Depolarizers.—Of this group the Grenet cell is the most used for medical purposes.

Grenet's Cell.—The elements are two plates of carbon and one zinc plate (amalgamated). The zinc element can be lowered into the fluid or raised at will. It lies between the carbons. The depolarizer is bichromate of potassium. The active constituent of the fluid is dilute sulphuric acid. These two ingredients form what is known as the "red-acid fluid." These cells are of different sizes.

Trouvé's Cells (1875).—Similar to Grenet's, but of large size. E. M. F. = 2 volts. The internal resistance varies from .0016, when first

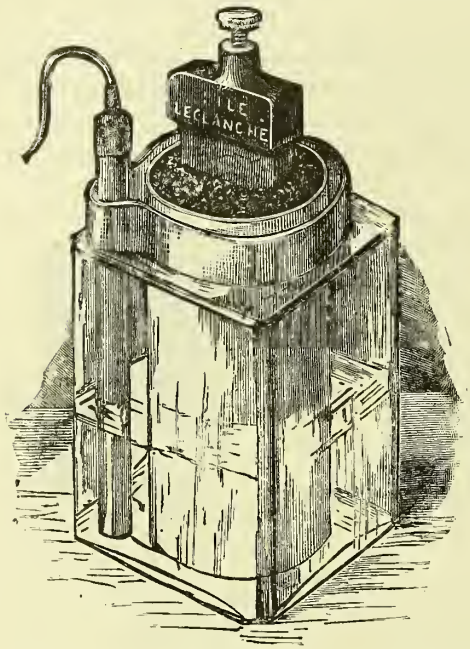


Fig. 143.—Leclanché's Cell.—The zinc rod (the one with its rheophore attached) is shown as immersed in a solution of ammonic chloride. The carbon element is seen to project slightly above the porous cup, in which, when the cell is properly prepared for action, it is packed with peroxide of manganese and afterward covered with pitch.

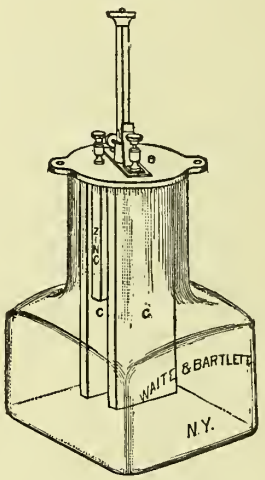


Fig 144.—Grenet's Cell.—The flasks come of all patterns, according to the taste of the various makers. In the form here depicted the zinc is lowered into the fluid by a straight handle. This is the cell most used in portable electrical apparatus. It is cheap, efficient, and easily repaired. Removing the elements and replacing them overcomes "polarization" in case the current grows weak from that cause. Some makers place the zinc in a drip-cup when the cell is not in use.

set in action, to .07 after the "spurt." The plates are raised and lowered by a windlass. The extent of immersion can thus be graduated. This form of element is known as a "plunge battery."

Fuller's Cell.—A porous cup containing zinc, mercury, and water is placed in a large glass jar containing red-acid fluid, in which a large carbon plate is immersed. The mercury keeps the zinc amalgamated. The elements are not removed when the cell is not in action. This form of cell is perhaps the best one yet devised to run the faradaic part of a cabinet battery.

IV. Two-fluid Cells.—In this group, the Daniell, Grove, and Bunsen cells are the most used. The two latter are not well adapted for medical purposes. The fumes which arise from some of them are unpleasant. Dynamos are now generally substituted for them in the mechanical arts.

Daniell's Cell (1836).—The so-called "sulphate of copper" cells (of various types) are modifications of that devised by Daniell. The elements are zinc and copper, separated in the original form by a porcelain or baked-clay diaphragm. The zinc is immersed in dilute sulphuric acid, and the copper in a saturated solution of sulphate of copper. E. M. F. = 1.079 volt. The solution for the zinc element may also be pure water, salt and water, or a solution of sulphate of zinc.

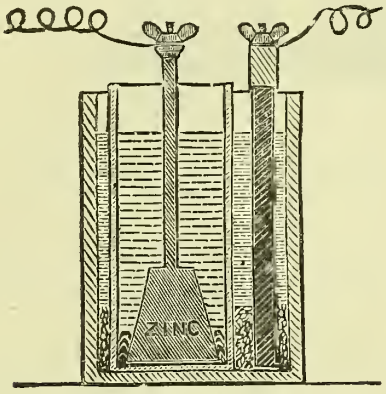


Fig. 145.—Fuller's Cell.—This is the best cell (in the opinion of the author) to use in connection with a permanent faradaic machine. It is not well adapted for transportation.

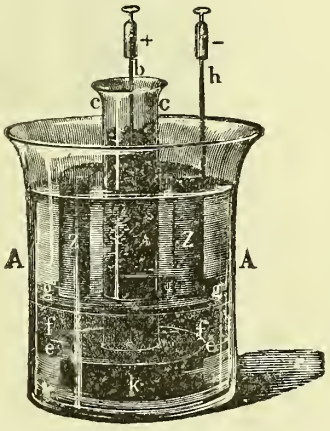


Fig. 146.—Siemens and Halske's Cell.—This cell is very efficient, but it is expensive to repair when the battery becomes exhausted. It is highly recommended by some European authorities for use in a cabinet or permanent office battery. In this country the Leclanché cell is more favorably regarded.

Siemens and Halske's Cell.—This is a favorite cell for medical batteries in Europe. It is a modification of the Daniell's cell and is expensive. A copper rosette is placed in a saturated solution of sulphate of copper at the bottom of the jar; this is covered with a porous diaphragm packed with *papier-maché*, on which the zinc rests surrounded by water. Water is added to the battery from time to time, and also crystals of the sulphate of copper. This form of cell is very constant; but it is *extremely difficult to repair* when out of order. As a permanent battery, such cells may last a long time with proper care; but they often do not, as the cells may become impaired from a multitude of causes (poor construction, improper use, etc.).

Hill's Gravity Cell.—This is another modification of the Daniell cell. It is used in medicine by many neurologists. A copper plate rests on

the bottom of the glass jar, covered with a saturated solution of sulphate of copper. The zinc element is a disk perforated by a large central opening, through which crystals of sulphate of copper may be dropped when the battery is inactive. A solution of sulphate of zinc floats, with-

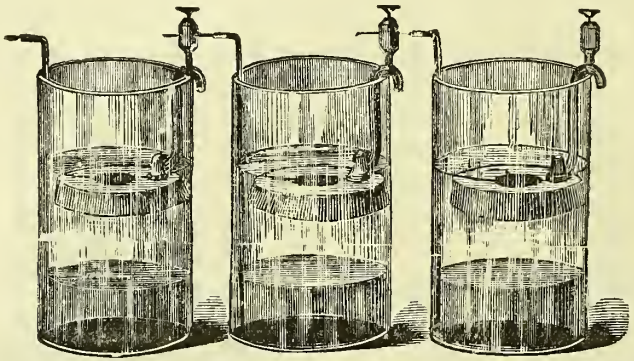


Fig. 147.—Hill's Gravity Cell.—This cell is employed very extensively in telegraphy, and is recommended by some authors for permanent medical batteries. When the jars are well paraffined at the top the cells do not "salt" badly. They require but little care when properly set up. Personally, I prefer a modification of this cell (in which the zinc is placed within a suspended porous cup) to the one shown in the cut. It requires less care, and is not affected by agitation. It also has a higher internal resistance.

out an intervening diaphragm, on top of the copper solution, and immerses the zinc disk. This battery must not be agitated, as the two solutions would then become mixed. E. M. F. = 1.068.

Grove's Cell (1839).—This consists of amalgamated zinc immersed in dilute sulphuric acid within a porous pot. Outside of this pot plati-

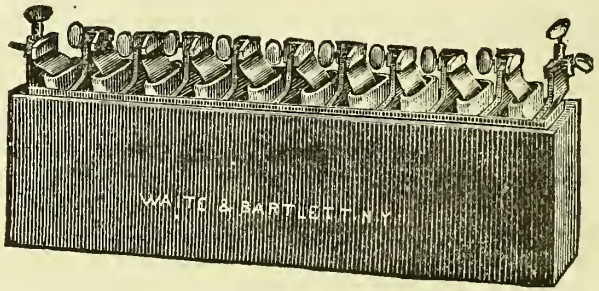


Fig. 148.—Grove's Cell.—This cell (shown here in the form of a battery) is not used in medical practice, chiefly on account of the fumes which arise from it. If used in the mechanical arts, it is a very expensive cell to employ. Dynamo machines have now taken the place of Grove batteries to a very great extent.

num is immersed in nitric acid placed in a glass jar. E. M. F. = 1.96 volt. The platinum is bent into an S-shape to increase its surface. Many modifications of this cell have been made for use in mechanical arts. The fumes arising from it are very objectionable.

Bunsen's Cell (1840).—This is a modification of the Grove cell. The platinum is replaced by artificial carbon in the form of a hollow cylinder, and a cylinder of zinc is bathed in dilute sulphuric acid. E. M. F. = 1.9 volt.

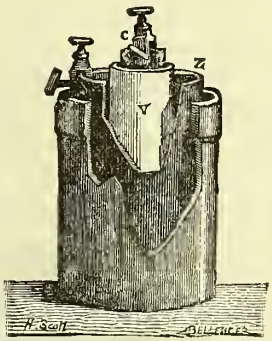


FIG. 149.—BUNSEN'S CELL.—Parts are represented as bitten away to show its arrangement: the cylinder of zinc (*z*); a porous cup (*v*); and the carbon (*c*) within it. This cell is not employed in medical batteries, for reasons similar to those given in connection with Fig. 148.

ATTACHMENTS TO A COMPLETE BATTERY.

Although it is not neeessary for a general medieal practitioner to have all of the attaehments to a battery such as are employed by neurologieal speeialists, still it is important that they be mentioned here, and their uses interpreted. The most important attachments to a cabinet or fixed battery for office use only are a galvanometer, a rheostat, a thermo-eleetric differential ealorimeter, a polarity ehanger or "eommutator," and a variety of rheophores and eleetrodes. Portable batteries do not require, as a part of their construetion, the first three of these attaehments, but they should possess the others.

THE GALVANOMETER.—When a galvanie current eirculates in a eoil of wire about a magnetic needle, it eauses deviations of that needle, whieh are modified by both the strength and direetion of the current defleeted into the eoil. This faet has led to the construetion of an instrument, called "the galvanometer," for the purpose of measuring the *strength* and *direction* of a current deflected into a coil beneath such a needle. When this instrument (properly made and ealibrated) is conneeted with a battery, the strength of current generated by any number of cells ean be determined in milliampères.* It is vitally important that the dial of a horizontal galvanometer should *not be divided into degrees of equal distances.* Such a galvanometer is absolutely worthless. The graduation of the dial should be *by tangents*, as shown on Fig. 150.

The defleetion of the needle grows less and less for every milliampère of current; henee a dial, to be accurate, should be earefully graduated so as to eorrespond with the needle-defleetions for different current-strengths. The first divisions on either side of the zero point on sueh a dial will be eoarse, but they should gradually grow finer and finer till the maximum point is reaehed. *Such a dial will not be graduated around its entire circumference*, as the maximum point will be reached before the

*Every galvanometer should measure at least quarters of the first milliampère to be considered worthy of indorsement.

90° of the circle on either side are required. One of my own galvanometers is graded into *equal degrees* on one half of the dial, and on the other half it is calibrated to milliampères.

Within the past few years the efforts of Erb, Eulenburg, and Bernhard in Germany, Gaiffe in France, and De Watteville in England have awakened the profession to the necessity of accurately measuring the current-strength employed upon a patient by means of a reliable galvanometer. To their views I lend my most hearty support. As well can I conceive of a boiler without a steam-gauge, or of a drug-store without a scale, as a galvanic battery without a galvanometer, provided its possessor

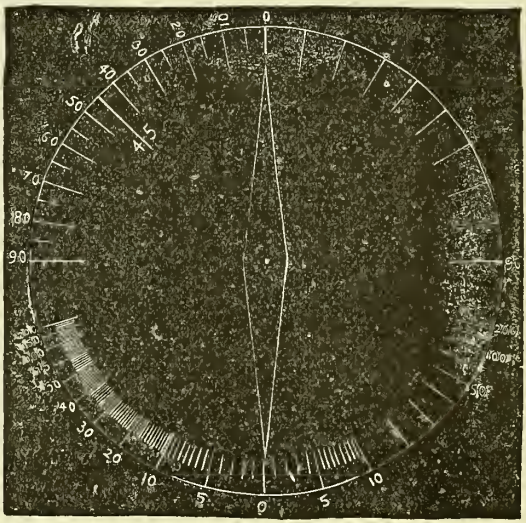


Fig. 159.—A Galvanometer Dial. (After De Watteville.) The lower half of the circle is graduated to milliampères; the upper half to degrees of equal distance. One serious criticism can be made of this dial, viz., that it does not indicate fractions of the first milliampère of current. To my mind, a galvanometer-needle deflection for the first milliampère of current should be sufficient to show at least a quarter or an eighth of a milliampère. This fault is common to all vertical milliampère-meters with which I am acquainted. Even Hirschmann's instrument does not entirely overcome it. I am at work at present upon a new form of milliampère-meter, which I hope will remedy this serious objection and at the same time allow the needle deflections to be read easily when the eye is on the same level as the needle.

aims at scientific precision in his treatment of patients by galvanism. Much of the neurological literature we now possess is materially lessened in value by the fact that the observations recorded lack scientific precision. If we expect to arrive at positive conclusions regarding methods of employing electricity for therapeutic or diagnostic purposes, we must have a more accurate and scientific basis for recording the strength of the current employed than the simple statement of an observer "that a certain number of cells were used" in each particular instance. Cells vary in their capacity and electro-motive force; they change in both respects from day to day, from use and polarization; the external resist-

ance afforded by different individuals is not uniform, although the poles may be similarly placed and all precautions taken against poor conduction; and many other sources of error may creep into observations

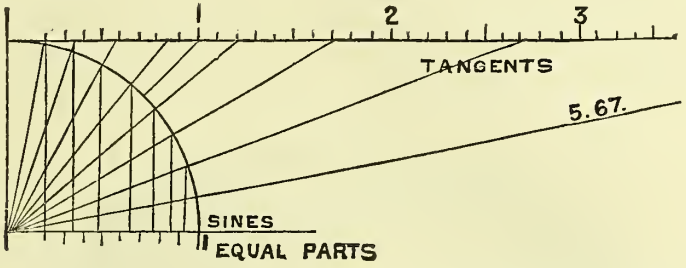


FIG. 151.—A DIAGRAM DESIGNED TO ILLUSTRATE THE METHOD OF TANGENT CALIBRATION. —The distances marked upon the straight line are uniform. When they are joined by imaginary lines with the centre of the circular dial, these lines intersect its circumference at points which steadily tend to approach each other; hence the first milliampère will produce a needle deflection which may exceed that produced by ten or more milliampères in some other part of the dial. The more sensitive the needle, the greater will be the distances marked upon the straight line, and the dial also, on either side of the zero point; hence a very sensitive needle, balanced so as to avoid unnecessary friction, will record eighths and quarters of one milliampère of current, if the coil be long enough.

practically made by "guesswork" only. The scientific world has now quite generally accepted the "milliampère" as the recognized standard of a unit of current-strength.

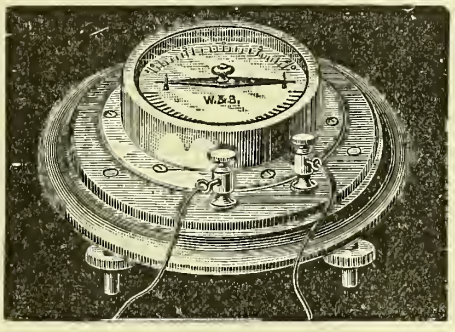


FIG. 152.—A HORIZONTAL MILLIAMPERE-METER. (After the Thistleton pattern.) The screw-feet allow of adjustment so as to insure a perfect leveling of the instrument. It is then revolved so that the needle (which will point north) rests at the zero point of the dial. Reversal of the current diverts the needle to the opposite side. One of the rheophores shown goes to a binding-post of the battery, and the other to one of the electrodes employed upon the patient. This instrument is very delicate, but the eye has to look down upon the dial in order to observe the deflection of the needle. If the instrument is placed lower than the eye, this objection is not serious. With it it is easy to detect small fractions of a milliampère of current, and it is much less expensive than a good vertical galvanometer and more accurate than most of those offered to the profession. I do not recommend them for measuring very high currents.

A milliampère-meter is therefore the instrument which medical practitioners should own, and all observations should be recorded from the deflections of its needle.

We shall probably be able soon to state with some positiveness the number of milliampères which are required to excite each of the more important nerves of the human body in health, and the exact limits between which contractions of certain muscles may thus be excited. Eulenburg and Weiss have already made a step in this direction.

One reason why the vertical form of galvanometer is commonly preferred to the horizontal is the fact that terrestrial magnetism does not

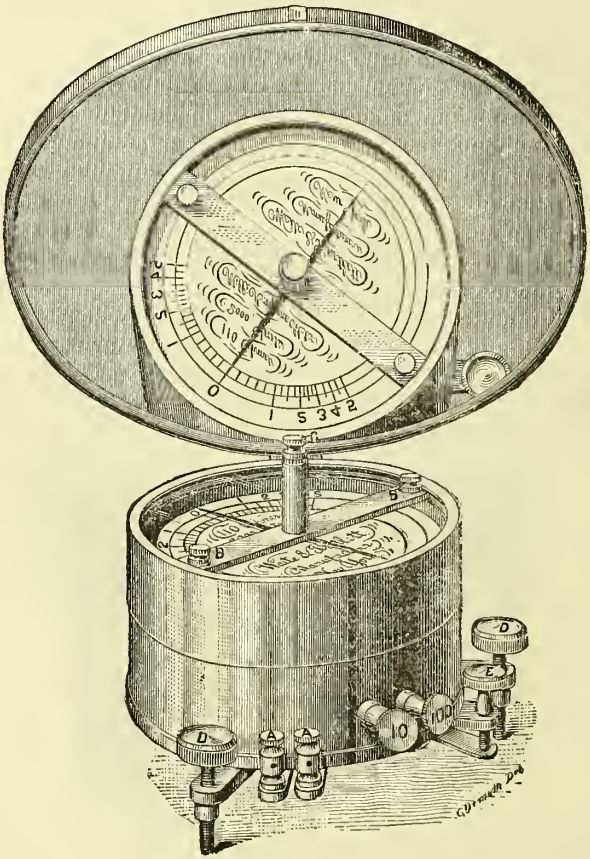


Fig. 153.—"Dead-beat" Milliampere-meter.

exert an appreciable influence upon it; hence observations made with such an instrument would be alike in all parts of the globe. The vertical galvanometer is, however, more expensive, provided it is accurately calibrated, than the horizontal, and the bearings upon which of necessity the needle rests are liable to cause more or less friction. At my request, Messrs. Waite & Bartlett, of New York City, have lately graduated some horizontal milliampère-meters with great accuracy (Fig. 152). I

can recommend them as reliable and comparatively inexpensive. They will not, however, accurately measure currents of over fifty milliampères. Before you purchase this important attachment to a battery, test it, if possible, by one whose accuracy can be relied upon. I would far rather have none than a poor one. At present I am using the so-called "dead-beat" milliampère-meter, made after a late design.* Hirschmann's vertical galvanometer with astatic needles, called the "absolute galvanometer," has been highly recommended of late. I have not yet purchased one, as I find my own to be perfectly reliable.

The instrument represented on opposite page measures from one-tenth of one milliampère to five hundred milliampères. It is contained in an all-metal case and carefully calibrated. It contains the Siemens' bell-magnet, and is dampened in its vibrations by the induced currents set up during the deflections of the needle, whereas in most instruments of the better class the oscillations of the needle average at least twenty-five before it comes to rest. In this instrument the vibrations seldom exceed from five to six. Shunts are provided in order to measure very high currents. I regard this instrument as fully equal, if not superior, to Hirschmann's expensive instrument.

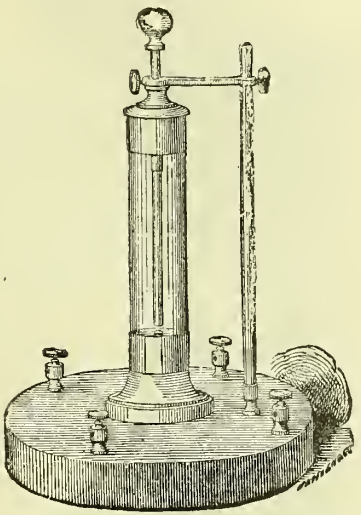


Fig. 154.—A Fluid Rheostat.—This instrument is used to throw additional external resistance into circuit. Coil rheostats are more reliable, because fluid is decomposed and causes polarization of the metal points when strong currents are used. Some of the graphite rheostats are preferable to many containing fluid, and are cheaply made.

The Rheostat. — This is an appliance to regulate the external resistance of a battery under varying circumstances. Several devices are made for this purpose, but the fluid rheostat (containing water, solutions of salt, solutions of zinc sulphate, etc.) is often all that is required for medical uses. It is cheap, easily managed, and modifies the current-strength. It is liable to polarize† when used too long, and does not act so well with strong currents as with weak ones. It consists of a glass tube filled with water or some prepared solution (preferably a forty per cent. solution of sulphate of zinc), through which a brass rod or an

* This is shown in a cut of my own cabinet battery, on a succeeding page, and in Fig. 153.

† We speak of an element as "*polarized*" when bubbles of hydrogen or oxygen accumulate upon it, and thus diminish its efficiency.

amalgamated zinc electrode is made to slide up and down, thus separating its lower end from a button at the bottom of the tube. When the current is sent through this rod, it is forced to pass through the depth of fluid that lies below it in order to reach the button at the bottom of the tube. By moving this rod, the amount of fluid which is thus interposed in the circuit of the battery can be graduated to any desired point. In this way a greater or less resistance can be made at will. A fluid rheostat is absolutely useless for measuring the strength of a current, but is an excellent appliance for modifying it.

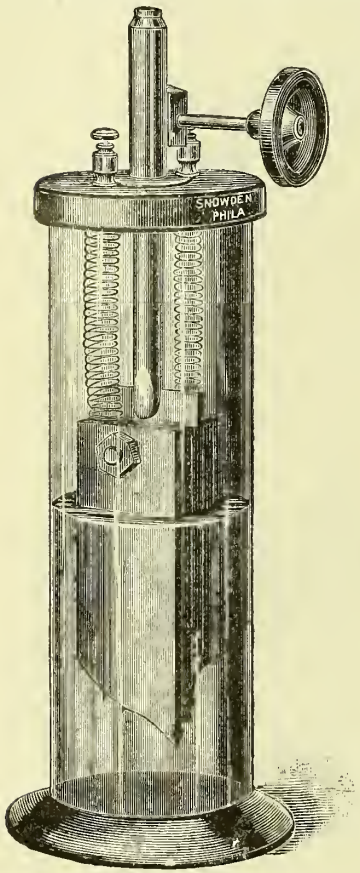


Fig. 155.—Rosebrugh's Fluid Rheostat. (*Canada Pract.*)

A new fluid rheostat has lately been devised by H. L. Bailey, which has been highly recommended by Rosebrugh, of Canada, and others. I have had no personal experience with this instrument. It certainly does not measure resistance (as a well-constructed coil rheostat may be made to do), but it may prove a valuable adjunct to an electrical outfit for practical purposes. Fig. 155 shows the instrument referred to.

The carbon plates of this device are made of a wedge shape, and have pyramidal pieces of sponge placed between them. As the plates are withdrawn from the fluid in the jar these sponges hold sufficient water to afford an extremely high resistance to the passage of the current. When the plates are fully immersed the resistance afforded is extremely small. It is claimed that from ten to one million ohms of resistance can be thrown into the circuit with this instrument. It is further claimed that by raising or lowering the plates of this rheostat the necessity of a commutator is dispensed with, even for the purpose of preventing a shock to the patient when the poles are applied or removed.

The Thermo-Electric Differential Calorimeter.—This apparatus is used in medicine to measure differences in temperature in homologous parts of the body, or of any two selected points. It is much more delicate than any form of surface thermometer, and is often very valuable as an aid in making a scientific diagnosis. Like many of the instruments

of precision, it requires experience to use it and it is somewhat expensive. The study of surface thermometry has not assumed the importance which, in the opinion of the author, it is destined yet to take. Waite & Bartlett, of New York, have constructed for me one of the most perfect instruments of this kind that I have ever seen.

The study of cerebral thermometry has already led to the discovery that the left hemisphere is normally hotter than the right (Hammond); that willed muscular action raises the temperature of the scalp over the motor centres called into action (Amidon, Gray, and others); that mental activity, emotional excitement, and merely arousing the attention cause a rise in temperature (Lombard); and that tumors of the brain or its envelopes are indicated by a localized rise in temperature over the site of the neoplasm. I have lately made some novel and interesting experiments in this field which I propose shortly to publish. In detecting inflammatory conditions of the abdominal viscera, this instrument has lately been employed with satisfactory results. I have lately made an improvement* upon this

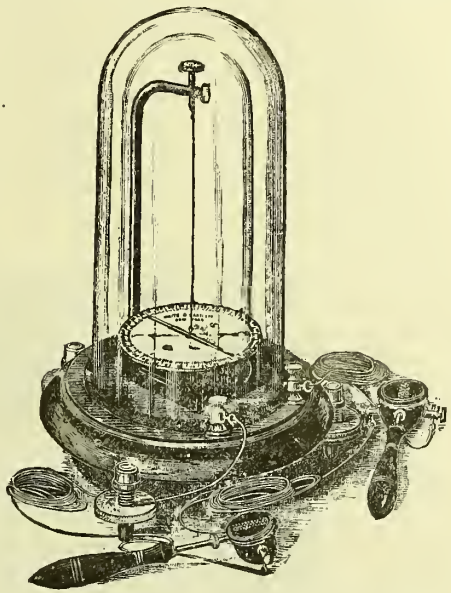


FIG. 156.—THERMO-ELECTRIC DIFFERENTIAL CALORIMETER.—Connect the two thermostats as shown in figure, viz: connect by means of one of the metal-tipped cords one binding-post of each of the thermo-piles to the two binding-posts on base of the galvanometer. Then connect the two remaining posts, one on each of the thermo-piles with each other. After so doing, place the thumb on the face of one of the thermo-piles and observe the direction of the deflection of the galvanometer-needle, then place thumb on face of the other thermo-pile, leaving the first uncovered, and, if the deflection is in the opposite direction to that first obtained, the instruments are properly connected. If, however, the second deflection is in same direction as obtained by pressing thumb on first thermo-pile, disconnect the two cords from either thermo-pile and interchange them, viz.: take cord from right-hand post and place in left, and cord from left post and place in right hand post; the deflections will then be as first alluded to, one pile turning needle in one direction and the other in the opposite direction.

*The improvement to which I refer consists in the addition of a polarity changer of my own construction, which enables me to reverse the deflection of the needle without removing the thermo-piles from the surface of the patient. If the needle, for example, shows a deflection of 15° when one thermo-pile is on the right side and 12° on the left side, the difference of 3° shows double the imperfection which exists in the thermo-pile thus tested, and the proper registration should therefore be 13½°. Such imperfections in thermo-electric piles are practically unavoidable and can be detected in no other way. As far as I know, this defect has never before been remedied. This has heretofore been the only serious drawback to the differential calorimeter, and the addition of this improvement renders the instrument far more valuable for accurate scientific purposes.

instrument which enables the physician to detect differences in the electro-motive force of the thermo-electric piles employed. This corrects all errors in observations made with this instrument.

The Current-Selector.—This device is now added to all of the modern galvanic batteries, whether designed for office use or for transportation. By it, the *number of cells desired can be thrown into circuit.* If a circular dial studded with buttons (which represent the cell-connections) is used, the bar which revolves and impinges upon the buttons acts as a connection between the button on which it rests and the metallic pivot on which it revolves.

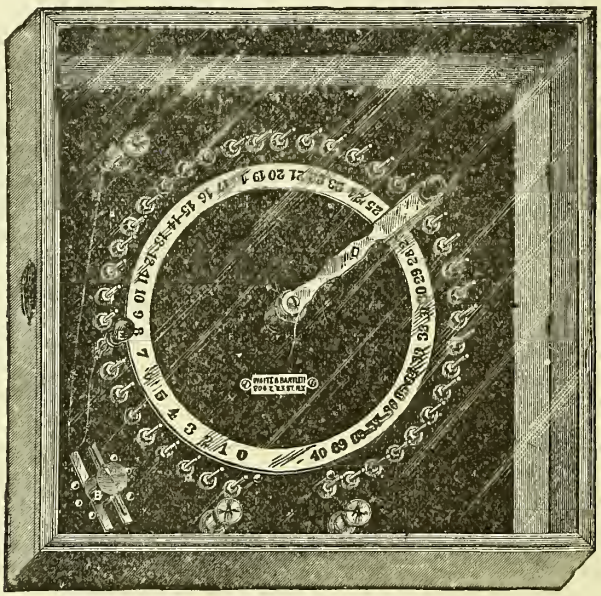


Fig 157.—A New Form of Current Selector.—This allows of a selection of any desired number of cells from any part of the battery, thus insuring equal wear and many other advantages.

It is important that this bar be broad enough to touch each button before it leaves the one behind it, otherwise the current is apt to be broken when the intensity of the current is changed. I have known of serious results from such an accident when a large number of elements were being used upon the head of a patient. At my suggestion, a modification of the dial current-selector has been made by Waite & Bartlett, of New York City (Fig. 157).

Another form of current-selector is that employed by the same firm for some of their instruments. I greatly prefer it to any other kind for a portable battery, as it enables the operator to select the desired number of cells from any part of the battery, thus insuring an equal amount

of wear upon all of its parts. It consists of pins projecting from the dial-plate, each of which represents one cell. These may be brought into circuit by means of two metal caps, which are placed upon any of the pins desired; the number of pins between the caps will immediately tell the operator how many cells are being used. An objection to its

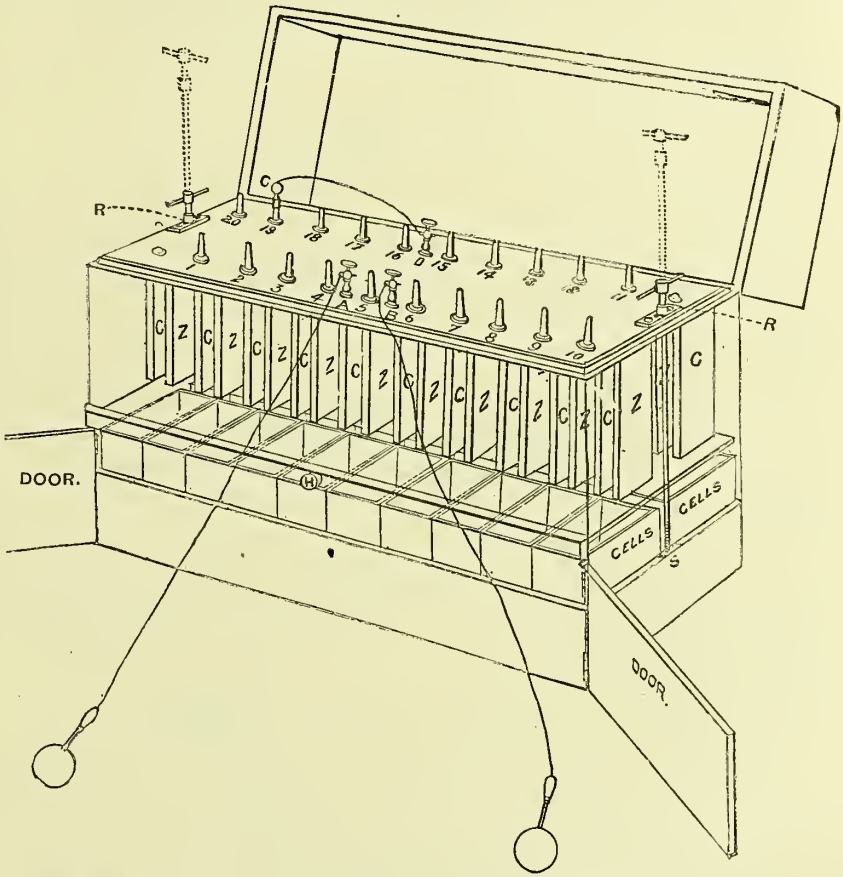


Fig. 158.—A Skeleton Drawing of the Pin Variety of Portable Galvanic Battery. *R*, handles by which the tray of cells is raised and lowered. *Z*, zinc elements. *C*, carbon elements. *A*, *B*, binding-posts. *D*, attachment of the stopper which fits over the pins. *H*, a rubber-covered diaphragm which separates the cells from the elements when the battery is not in use; this is removed when the cells are lifted so as to immerse the elements.

use is that it dispenses with a polarity changer,—in case the cells employed are capable of being thus selected.

Fig. 158 shows a portable galvanic battery of this make with the current-selector attachment described. In this form one stopper only is employed, however, and the cells in use are indicated by the numerals placed at the base of each pin. With this arrangement a polarity changer

is admissible, and is generally affixed to all so constructed. This is particularly to be desired.

One claim that is made in favor of this device is that this form of key-board prevents oxidation, as the wires which are usually employed to join the cell-elements with the current-selector in other apparatuses are entirely dispensed with. This advantage is an important one to those who live away from the large cities and are not sufficiently familiar with electrical apparatus to make their own repairs. Furthermore, the employment of the metal cap keeps the metal pins free from rust, and gives a perfect metallic contact because the pins are being constantly kept bright and clean.

In batteries which are formed of a large number of cells, it is best to have two dial current-selectors, so that a gradual increase or decrease of the current can be made without breaking the circuit. In one dial each button should represent one cell, while in the other each should represent from two to five cells. It is easier to make any desired combination of cells with rapidity by means of such an arrangement than if the dials were alike.

The Polarity Changer or Commutator.—Most galvanic batteries have a switch upon the key-board that is intended for the purpose of changing the poles at will without disturbing the rheophores or electrodes. The details of the many mechanical contrivances employed for that purpose need not be given here.

This attachment is almost indispensable to a battery designed for office or experimental work, since the reactions of the poles can thus be more readily studied. It is desirable, moreover, to have it attached to a portable galvanic battery.

It should be so arranged as to permit of *opening and closure of the circuit*, as well as the *reversal of the current.*

The Rheophores.—These are flexible wires which are necessary to conduct the electric current from the battery when in action to the patient. Insulated copper wire forms the best rheophore, as it is an admirable conductor. Tinsel threads insulated with cotton wrapping are more generally used, because they do not kink and are more flexible (although they are not such good conductors). They are connected at one end to the "*binding-posts*" of the battery, and at the other to the electrode. They should vary between four and six feet in length. I have some that are ten feet in length, which I employ when I examine the naked body of a patient lying upon a sofa or bed.

The Electrode.—In order to apply a current of electricity generated in a battery to the human body, various forms of electrodes are employed as termini to the rheophores. It is best to have a pair of handles to which different forms of tips may be screwed, according to the require-

ments of each ease. The tips may be made of plain metal, or of earbon or metal covered with sponge, chamois-skin, or eanton-flannel. The canton-flannel eovering is the cheapest and cleanest, and may be renewed at pleasure. Eaeh patient can thus have a elean eovering for the eleetrode at every applieation. Flat electrodes of large size are useful, espeeially when a neutral point for the current is desired. Small tips (motor-point eleetrodes) are generally employed to direet the current to some special muscle or group of muscles.

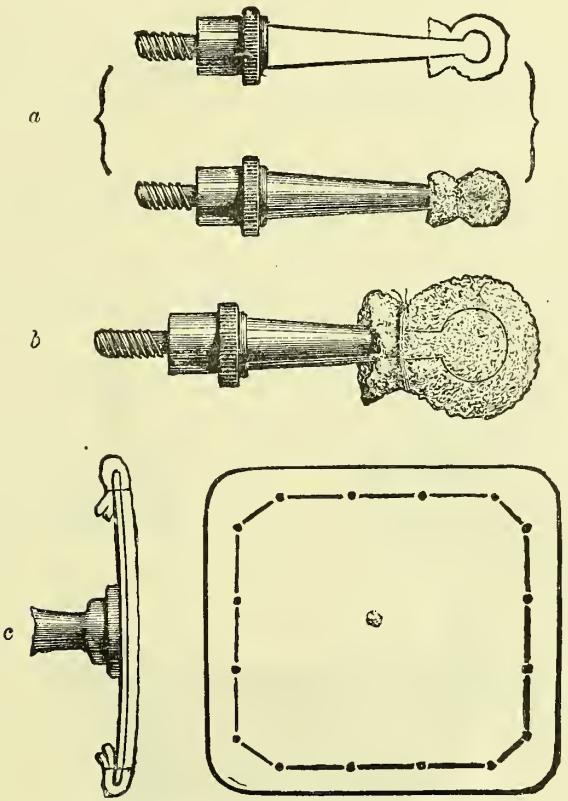


FIG. 159.—VARIOUS FORMS OF ELECTRODES (NATURAL SIZE), ADAPTED TO A SCREW HANDLE, NOT SHOWN IN THE CUT. (After Erb.) *a*, the "fine" electrode, or smallest size; *b*, the so-called "small" electrode; *c*, the "medium" electrode. All electrodes are covered with sponge (as in *a* and *b*) or flannel or chamois-skin (as in *c*).

The wire brush is employed chiefly in cases where anæsthesia exists. It is the only eleetrode that is used dry.

Most manufaeturers advertise a ease of eleetrodes designed espeeially for the application of eleetrieity to different organs. Seleetions may be made from these as desired. I have personally devised several modifications of electrodes.

A practical point may be mentioned here, viz., that the negative electrode (*cathode*) is the most painful to the patient, and produces the greatest chemical action. It is a well-recognized fact that a bullet does the most damage at its point of escape from any dense substance which it has penetrated. In the same way an electric current produces the

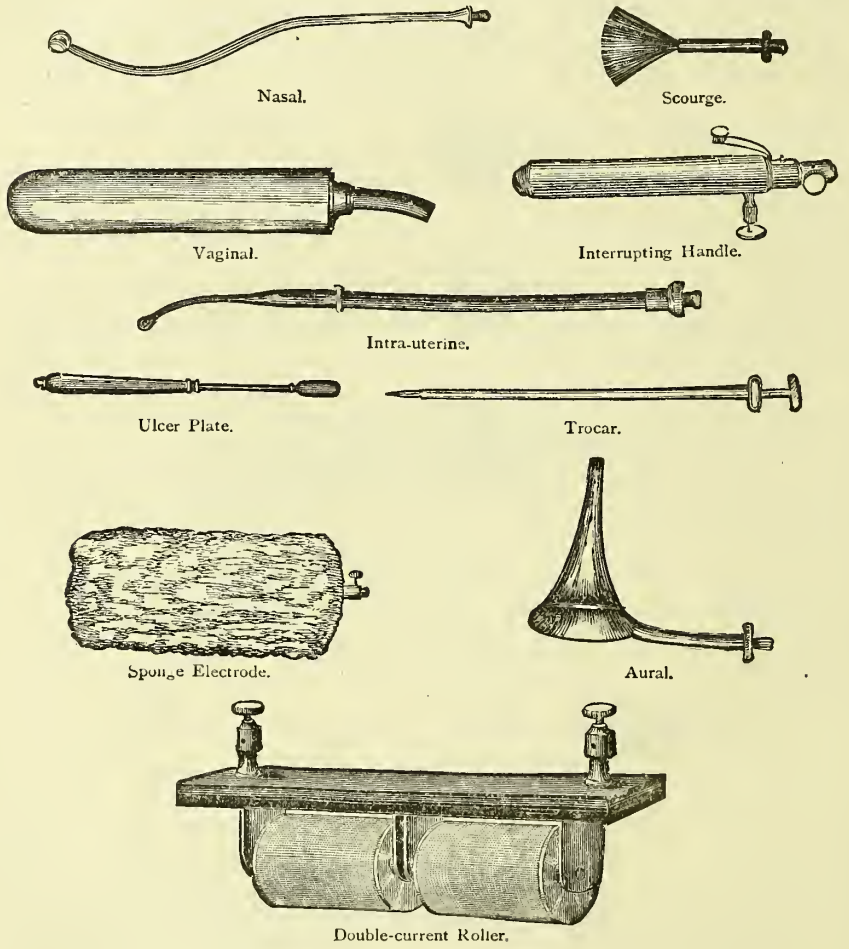


FIG. 160.—VARIOUS FORMS OF SPECIAL ELECTRODES.

most profound effects at its point of escape from the body, *i.e.*, the negative pole. It is not uncommon to see a reddening of the skin, and even vesication, produced by a strong current at the negative electrode, if kept too long in contact with it. The cathode is the "stimulating" or "irritating" pole (if such an expression is admissible) of a galvanic battery. The anode is, by contrast, the "sedative" pole.

For the treatment of special forms of disease by electricity, different types of electrodes have been designed by various neurologists. A device of my own, which simplifies electro-diagnosis and enables a medical observer to watch and compare the effects of electrical currents of definite strengths upon muscle- and nerve-reactions of opposed limbs simultaneously, has been described in Section II. By means of this device, the physician may sit at a key-board and excite different sets of muscles separately or simultaneously (without moving from his chair) by touching certain keys, as if playing the piano or working a type-writer. I have found it, moreover, of great assistance in demonstrating before large audiences the action of nerves and muscles upon a living subject.

Space will not allow of a detailed description of the electrodes shown, most of them being simple devices, which really require but a limited experience to use them in a proper way.

When you purchase a battery, two sponge-covered electrodes will probably be sent with the instrument. It is advisable, for the following reasons, to remove the sponges: (1) Cases have been reported where disease has been transmitted by sponge-covered electrodes. (2) In case the metal electrodes become oxidized, they can be readily cleaned. (3) When employed upon a patient's body, absorbent cotton, wet in salt water, can be placed upon the clean metal, and a piece of moistened canton-flannel may then be wrapped over both and fastened to the handle with a rubber band, thus insuring *absolute cleanliness* and *perfect conduction of the electric current.*

Patients of delicate sensibilities rebel against the use of sponges which for months or even years have been employed in case after case requiring electricity. Who would patronize a barber-shop where one towel constituted the entire outfit of linen? Is it right to ask of patients what you would yourself condemn? Furthermore, how can electrodes covered with dirt and other deposits under a sponge be perfect conductors of electricity? Absorbent cotton and canton-flannel are far cheaper than sponges, and can be thrown away after being used.

THE CHOICE OF A BATTERY AND ELECTRICAL APPARATUS.

In selecting a battery for purely medical purposes, the chief objects to be attained are, to my mind: (1) *cheapness;* (2) *constancy of the elements* and their *accessibility for repairs, cleaning*, etc.; (3) *durability of the elements;* (4) *a sufficient number of elements;* (5) *ease of renewal of the elements;* (6) *ease of introduction of any number of elements into the circuit;* and (7) an ability to select such as may be required *from any part of the battery.*

For the general practitioner it is necessary, as a rule, that a galvanic or faradaic battery *shall be arranged for transportation;* hence the cups

which hold the fluid should have a rubber cover, or some other device which will preclude the possibility of spilling the fluid. Again, some of the batteries manufactured are liable to become rapidly oxidized by the fumes of the battery-fluid. This tends to destroy their durability, and to cause difficulty in keeping them in good working-order. Finally, it is

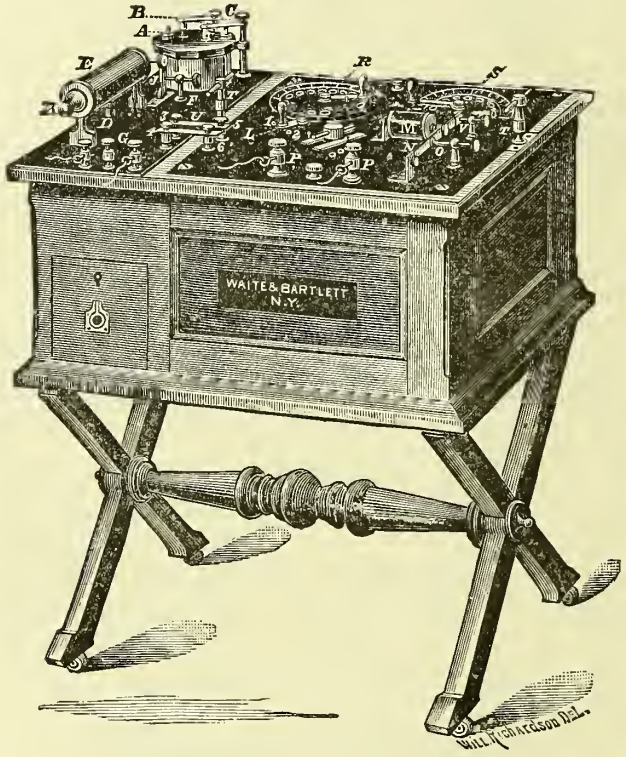


FIG. 161.—THE PHYSICIAN'S HANDY CABINET BATTERY. (*Author's model.*) The accompanying cut represents a light and compact form of cabinet battery, designed by the author. It is on castors, and can be wheeled about the consultation-room. This admits of the use of the instrument when the patient is in the gynæcological chair or upon the office lounge; or when any form of fixed apparatus, such as the laryngoscope, the inhaler or spray, etc., is being simultaneously employed. In some of my later models an immovable tray is placed beneath the battery for electrodes, and a movable shelf is also provided upon which a milliampère-meter, the solution of table salt, and the electrodes in actual use can be set. A glass cover protects the battery from dirt when not in use. *E*, faradaic coils; *K*, plunger; *G*, faradaic binding-posts; *F*, interrupter; *D*, drip-cup; *R*, current-selector of single cells; *S*, the same of four cells to each button; *M*, coil to work the interrupter for the galvanic current; *L*, switch to work or disconnect the interrupter (*V*); *P*, galvanic binding-posts; 3–5 and 4–6, connecting rods to allow of the action of *M*. The commutator lies above *P*.

very desirable that portable batteries should be as light as possible, and not too large to be handled easily.

The *attachments upon the key-board* of every portable galvanic battery should comprise a current-selector and a commutator. There should be at least four rheophores, in order to make allowance for breakage,

additional connections, etc. Several electrodes of different sizes and shapes should also be selected,—preferably a large, a medium, and a small one,—a wire brush, and an interrupting electrode. These can be added to as circumstances demand.

For office purposes, a CABINET BATTERY has some decided advantages over a permanent one placed in an adjoining closet or cellar and connected, by means of wires, with a key-board in the consulting-room. A cabinet battery can be easily wheeled about, and is readily repaired. The cabinet should be so arranged as to allow the back and front of the compartment for the cells to be removed, in case the battery needs repairs or a renewal of the fluid. The connections of the battery with the key-board should also be made as easy of access as possible; this decreases the expense of alterations or repairs when such becomes necessary. They should be protected, moreover, as far as possible, against oxidation and dirt.

The cabinet battery which I use in my own office was made, under my special direction, by Waite & Bartlett, of New York City, and is as nearly perfect as one could desire. It contains *forty cells of the Leclanché pattern*, which are equivalent to sixty of the gravity cell. The connections and the cells can be exposed and easily reached by removing the front and back of the case. The cut on the following page represents its special features better than a long description. Considerable expense in constructing such a battery may be saved in the case, and by dispensing with some of the accessory apparatus shown.

The perfected office cabinet battery (Fig. 162) was made after my own designs. It consists of a handsome cabinet, arranged for storing the cells in the under part as shown. The upper part consists of a shallow closet suitable for affixing the upright switch-board as shown. The closet contains forty cells of the kind generally known as the "Gonda-Leclanché," for use as the galvanic part of the apparatus, and four extra cells to be used in running the faradaic part of the apparatus. The switch-board is provided with a universal current-selector for the galvanic part of the apparatus, by means of which any cell or series of cells can be brought into the circuit; thus doing away with the necessity of using cells from the same part of the group, in order that the cells can be worked and used up evenly. It is also provided (1) with an attachment for interrupting the galvanic current; (2) a wire rheostat, for interposing resistance into the circuit; (3) a pole-changing attachment; and (4) a milliampère-meter for measuring the current-flow during treatment. By means of a milliampère-meter and the wire rheostat, the resistance afforded by the patient at the time of the sitting may be readily measured. The apparatus also has a complete faradaic attachment; this latter being provided with fast and slow contact-breakers, as well as a

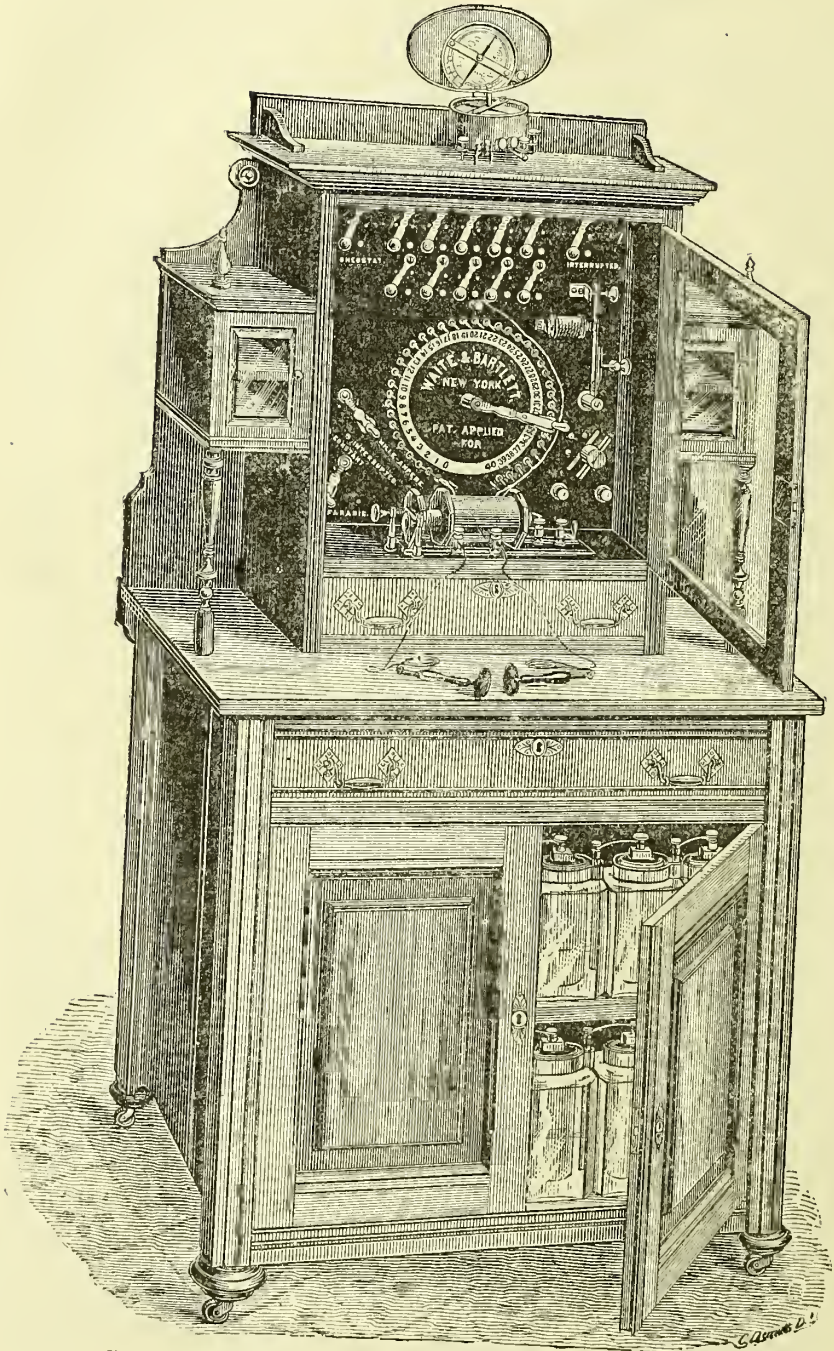


Fig. 162.—The Perfected Office Cabinet Battery. (*Author's model.*)

contact-key to be worked by pressure of the finger at the will of the operator. This latter attachment is of special use in electro-diagnosis. The cabinet is mounted on rubber-tired castors of large diameter, and can readily be moved on finished and polished floors without marring the same. It is also provided with a large drawer and closets for the electrodes. The milliampère-meter attached to this cabinet is separately shown in Fig. 153.

I believe this cabinet to be the most complete for practical work in electro-diagnosis and general electro-therapeutics that has ever been offered to the profession. [I am having a new cabinet so arranged for my own use as to contain a storage battery, so that I can employ the electric lamp or cautery at will. This is charged as circumstances require. It can be removed from the cabinet and transported for examinations or treatment at the patient's residence.]

A PERMANENT BATTERY is somewhat cheaper to construct and takes up less room in the office than a cabinet, because no case is required; but, in my opinion, these two advantages are not sufficient to render it preferable to the other for office or experimental work. I have known several of my medical friends to discard it (after a thorough trial) for a cabinet battery. If a permanent battery is deemed preferable by any of you (for reasons of your own) rather than a cabinet battery, be sure and place your cells on shelves in your office or waiting-room, and not in a cellar. The wires will not be so liable to corrode from dampness, and the cells will be constantly under your eye, so that you can see when they require attention.

The *gravity cell* makes a very serviceable and durable permanent battery for office work. It has one advantage over some other cells, viz., that it has great constancy of action and that its activity can be renewed by the addition of crystals of sulphate of copper to the fluid when necessary without disturbing the cells. For this reason the sulphate-of-copper cell, in some one of its various forms, is employed exclusively in telegraphic lines. Unfortunately, it tends to "salt" excessively, and the jars and elements become incrusted unless they are carefully watched and taken care of. Again, it cannot be transported about the room to suit the convenience of the patient or the physician during his examination so well as some other cells adapted for a cabinet battery. It is also difficult in many cases to repair the wire connections of a fixed battery (running, as they often do, through partitions and plastered walls to reach the key-board) when they become inefficient from any cause. I personally prefer the Leclanché cell to any other yet devised for a cabinet battery.

The engraving on page 644 represents the perfected cabinet so arranged as to be detached from the cells and screwed to the wall. The

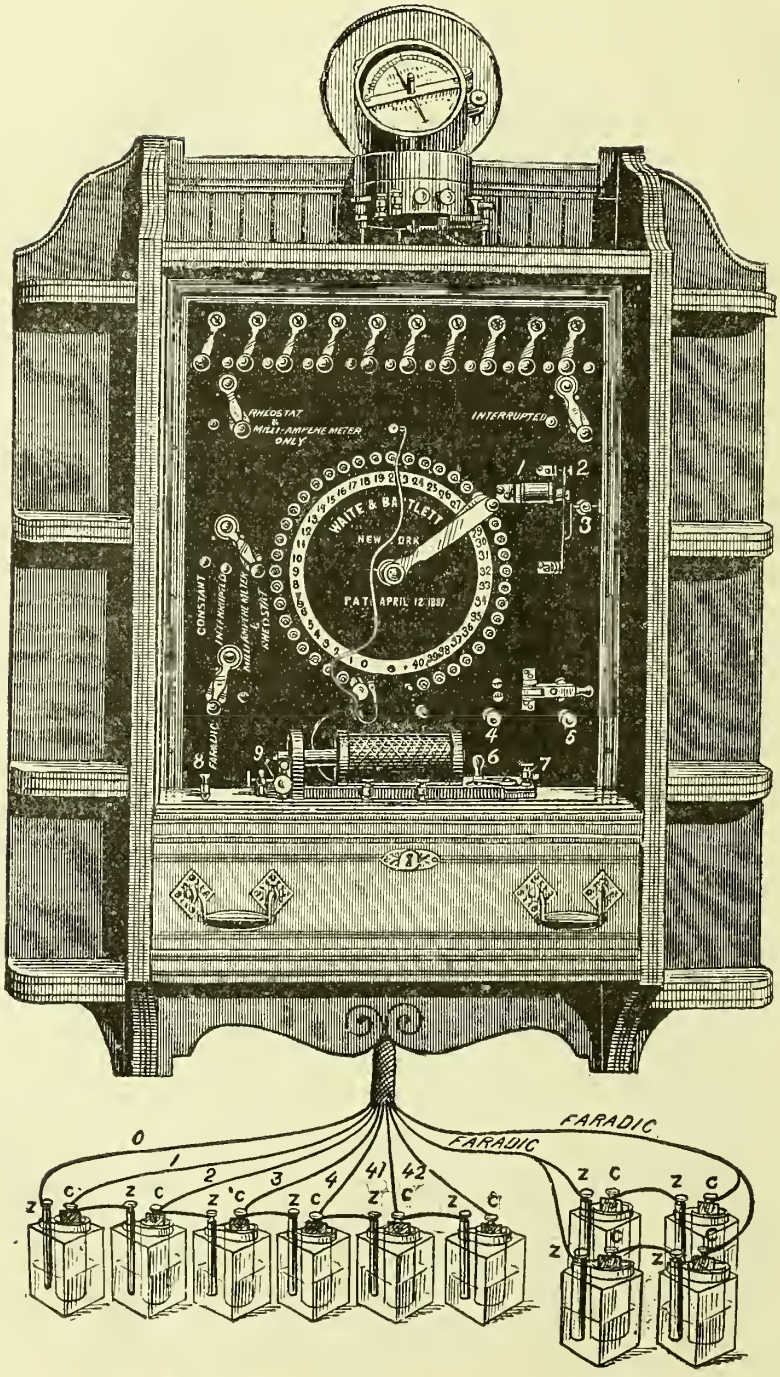


FIG. 163.—THE PERFECTED WALL-CABINET. (*Author's model.*)

cells may be placed in any convenient place, as closet or cellar. It is the same in all of its attachments as the perfected cabinet just described.

Respecting the selection of the cheaper forms of batteries for general medical use, it is important that accuracy of workmanship shall not have been sacrificed in order to lessen the cost. The construction of the primary and secondary coils of a faradaic machine and the adaptability of the interrupter to slow and rapid breaks in the circuit should be looked into before a decision is reached. Poor coils and a bad interrupter render a faradaic machine almost worthless. A "*drip-cup*" containing mercury, in which the zinc element is placed when the battery is not in use, is a desirable feature in a faradaic machine.

Do not buy a magneto-electric machine whose motor power is furnished by a crank to be turned by the hand. It is practically useless for medical purposes when compared with a good faradaic instrument.

The Grenet cell is now used by most of the manufacturers of electrical apparatus for a portable galvanic or faradaic battery. It is the best cell for this purpose, in my opinion, for many reasons that I have already expressed in print.* A thirty-cell galvanic battery of this type gives all the current that is required by the general practitioner.

Every galvanic battery should have a *commutator* on the key-board. Without this appliance electro-diagnosis becomes difficult.

THE CARE OF A BATTERY.

The best battery is liable to get out of order. It is an easy matter, as a rule, to correct the trouble if the construction of the apparatus is thoroughly understood. The following hints may aid the reader in obtaining a satisfactory current with a minimum expense:—

1. *Keep your battery clean and bright in all its parts.* Close the case when the battery is not in use, and thus keep out dust, grease, and moisture. Emery-paper is useful to keep the metal connections free from rust. Remember that a small amount of dirt, grease, or rust will often arrest the action of any battery.

2. When the battery fails to act properly, examine the cells first and see if *the fluid requires renewal.* The "red-acid fluid" is easily made by adding one part of commercial sulphuric acid to ten parts of cold water; when cooled, one part of finely pulverized bichromate of potassium should be added and mixed well. This is the fluid commonly employed in portable batteries with cells of the Grenet pattern.

3. If the fluid is found to be fresh, and if the zinc and carbon elements are in good order and the zinc well amalgamated, *examine carefully all the screws and other connections attached to the elements* and see if they have become oxidized. Sometimes they become rusted or so

* *N. Y. Med. Journal*, 1885.

covered with accumulated dirt as to render the passage of the current impossible. Occasionally the carbons may be disconnected and baked in an oven to render a Grenet cell more active. Soaking the elements (*in situ*) in hot water which does not reach the connections will generally suffice to cleanse them.

4. If the *cell has become polarized* when in action (by bubbles of hydrogen which accumulate upon the carbon and of oxygen upon the zinc element), lifting the zinc out of the fluid and replacing it immediately will suffice to overcome this trouble, whenever the cells are of the Grenet pattern. These bubbles of hydrogen and oxygen set up a counter-current in the cell, which will weaken and may even neutralize the original current.

5. *Examine the interrupter*, the *buttons of the current-selector*, and *the commutator* for rust or dirt, and clean each thoroughly when the trouble appears not to be due to the elements or their immediate connections.

6. If a *drip-cup is furnished* with a faradaic or galvanic battery, be careful to place the zinc element in it when the battery is not in use.

7. In portable galvanic batteries, be sure to *place the rubber-covered diaphragm over the cells* before closing the case and to screw it down tightly. This prevents the fumes rising and oxidizing the connections of the elements when not in use.

8. Be sure that the *rheophores are perfect* before they are used upon a patient. The wire used in their manufacture is liable to become broken or oxidized by use. This is especially true of the flexible, cotton-covered cords generally furnished with batteries. The electrodes may be tested by employing a galvanometer, if an imperfection is suspected and cannot be found.

9. The *wires* that run from the cells to the buttons of the current-selector or the commutator may be seen on the bottom side of the key-board of a battery. They can be examined for imperfections when the other parts of the apparatus appear to be perfect.

10. *Do not short-circuit a battery.* By this we mean, do not allow a battery to run down, or, more technically, "polarize," by the poles being brought into contact without an interposed body (such as animal tissue) for any length of time. For example, galvanic cells which have a low internal resistance (as a Grenet cell) become polarized in a few hours when the poles are connected by a short wire which affords little if any resistance to the current.

11. *Keep your electrodes clean.* As I have stated before, it is well to cover them with fresh canton-flannel for every patient. This is an act of precaution which will impress people with your regard for their feelings and for their safety from contact with infectious matter. Sponges

are too expensive to be renewed so often. Absorbent cotton may often be placed between the electrode and its covering with advantage.

STATIC ELECTRICITY.*

An exposition of the different forms of generators which may be employed, and the various methods of application of this therapeutic agent, together with hints respecting the care and management of induction machines and the selection of apparatus, seems to be advisable in a work of this character. Most works on medical electricity are singularly deficient in this field.

We owe to the ingenuity of Otto V. Guericke, the inventor of the air-pump, the first electrical machine where friction was employed as the

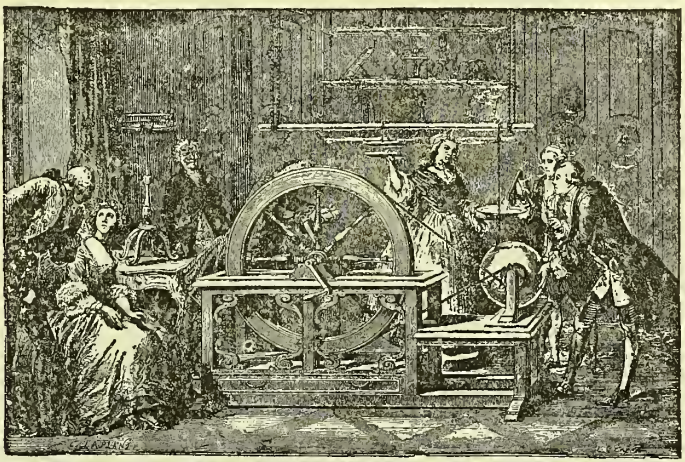


FIG. 164.—HAWKSBEE'S ORIGINAL ELECTRICAL MACHINE. (From *Leçons de Physique* of the Abbé Nollet, published in 1767.) The globe is of glass, and positive electricity is collected upon a conductor suspended by silken cords from the ceiling.

exciting agent. It consisted of a ball of sulphur which was turned upon its axis by hand-power. An assistant grasped the ball with his hands, and, by so doing, served as a conductor for the escape of the positive electricity to the earth. This primitive affair gave feeble sparks, which could only be seen in total darkness.

Hawksbee substituted later a globe of glass for the ball of sulphur. He obtained more satisfactory sparks with the positive electricity thus generated.

Later still glass tubes were used, with hand rubbing; and they entirely superseded the globe as generators until the middle of the eighteenth century.

* A portion of this discussion of static electricity originally appeared as a contribution to *The Physician and Surgeon*, Ann Arbor, Michigan, 1886.

In 1767, Hawksbee's original machine was revived in a modified form by Professor Boze, of Wittemberg; and for a time it came into general use. The cut of this machine (Fig. 164) is taken from the *Lecons de Physique* of Abbé Nollet.

The collector was hung from the ceiling by silken cords; and the hands of an assistant were used as rubbers upon the globe of glass.

In 1768, Ramsden, of London, invented the so-called "plate machine." The glass plate was supported by wooden uprights, and the

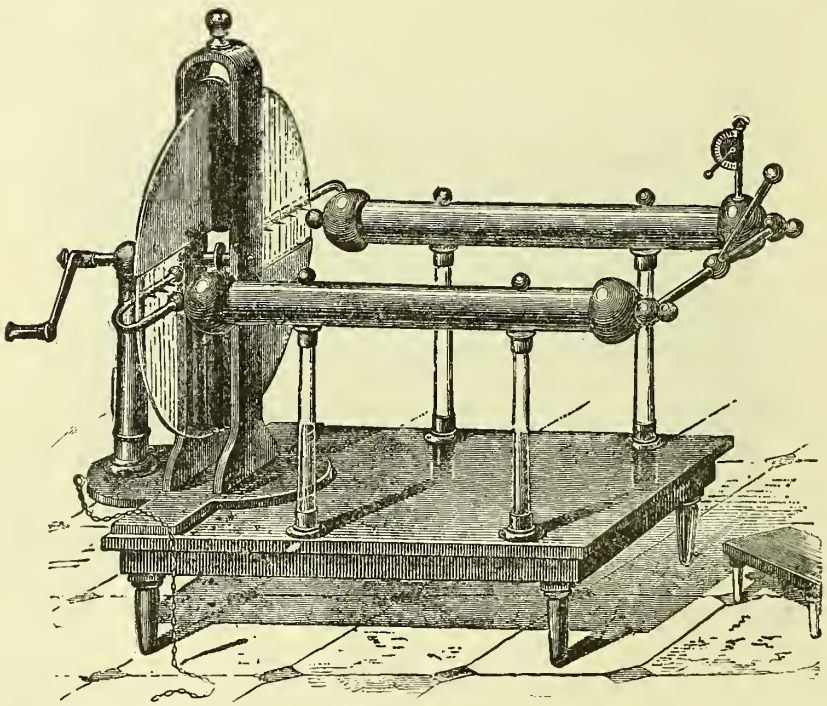


FIG. 165.—RAMSDEN'S ELECTRICAL MACHINE. (Invented in 1768.) It has sector-shaped pieces of oiled silk to prevent a loss of electricity from the glass plate while passing from cushion to cushion.

friction was made by means of two cushion-rubbers. The collectors were of metal; and two combs of metal were employed to draw off the electricity from the glass plate. The cushions were "grounded" by means of metal supports, so that the negative electricity which accumulated upon them could escape to the earth. In 1776, Von Marum modified Ramsden's apparatus so as to obviate this loss.

Nairne next modified the machine of Ramsden by substituting a cylinder of glass for a single glass plate, and by adding an attachment for collecting the negative electricity by means of an insulated conductor

placed in communication with the rubbers. This was the first machine that satisfactorily furnished both positive and negative static electricity.

Probably the first electrical apparatus which can properly be said to have been a true "*induction machine*" was described as early as 1788, by W. Nicholson, before the Royal Society of London. He called it the "*electric doubler*." It was built somewhat upon the plan of the machine now known as the Toepler model. It had three disks, attached to a common hub. These touched upon pins of metal at two points during each revolution, and passed between two pairs of insulated metal plates without touching them. They deposited their electricity upon a metal ball, which they also passed during each revolution. This ingenious

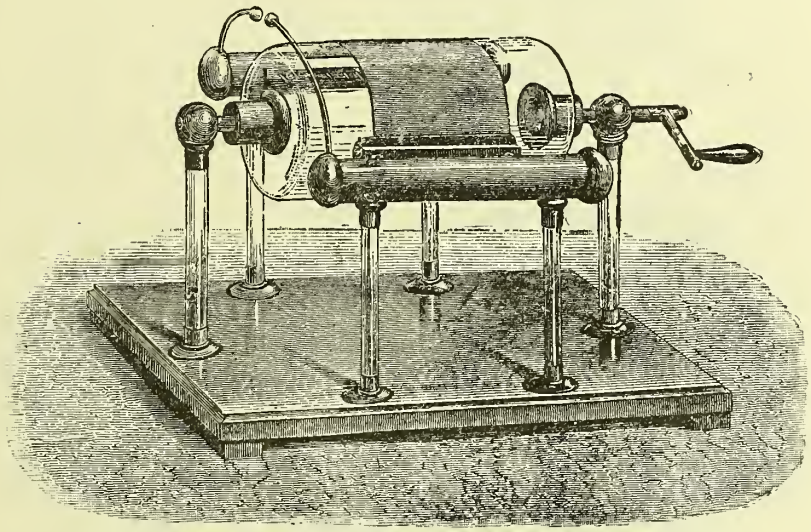


FIG. 166.—NAIRNE'S ELECTRICAL MACHINE.—The cylinder of glass revolves between two separately insulated conductors, one attached to the rubber and the other to a metal comb.

little instrument could to-day be made quite effective by slight modifications. For some unexplainable reason, it was apparently thought to be of little value, and even its existence is not mentioned by any standard author on electrical subjects with which I am familiar. A cut of the machine was published, however, together with the inventor's description of the machine, in an old work entitled the "New Royal Encyclopedia of Arts and Sciences." (See Figs. 167 and 168.)

Lane and Adams both perfected frictional machines during the eighteenth century, in connection with which the Leyden jar was used for medical purposes. Some of the cures reported by these crude machines are fully as startling as those now obtained by improved apparatus.

In 1840, Sir W. Armstrong devised a machine by which the friction of cooled steam against the sides of minute orifices, through which it escaped under a high pressure, became the generator of static electricity. The boiler was insulated by glass legs, and became negatively electrified. The jets of steam conveyed the positive electricity and deposited it upon a metal plate studded with points, upon which the jets were directed. This machine proved very powerful, but difficult to manage, and totally unfit for general use. It made a deafening noise, and saturated everything near it. One of these machines gave a spark of twenty-two inches.

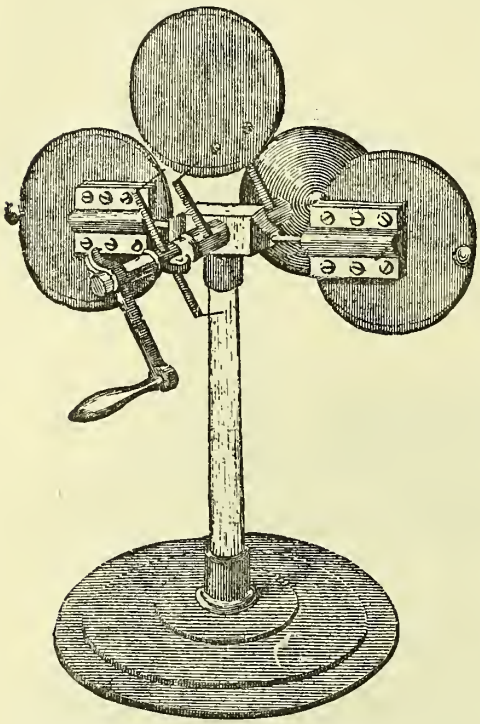


FIG. 167.—NICHOLSON'S "ELECTRIC DOUBLER." (The first induction machine invented.) Reproduced from the original cuts made in the eighteenth century. The lighter portions of the cuts are made of glass.

To Holtz, of Berlin, we do not owe the discovery of the first induction-machine, as many suppose. His apparatus was not perfected until 1865. Although the original model seems crude in comparison with our present instruments, still it cannot be denied that it contained the principle which formed the starting-point of all the later improvements; and many of the mechanical details of the original instrument are to-day generally used.

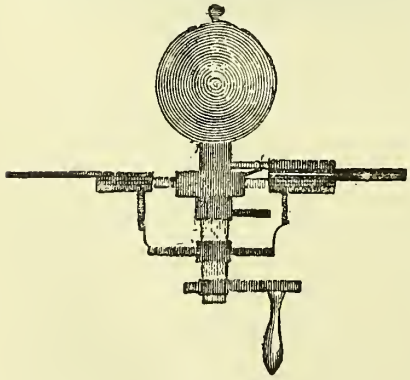


FIG. 168.—THE SAME MACHINE, VIEWED FROM ABOVE.

There is a modified form of an induction machine which is now sold quite extensively to the medical profession. It is known as the Toepler machine, or the Voss machine. It can be made with one or more revolving plates. The fixed plates are larger than the revolving ones, and have usually a central opening. They may, however, be divided or per-

forated centrally.* They are furnished with paper collectors and disks of tin-foil. The revolving plates have metal buttons attached to one of their faces. These buttons impinge upon metal brushes as the plate is revolved. The buttons rest on tin-foil cemented to the glass. The fixed plates are placed as close as possible to the revolving plates.

It is claimed that this machine will work in all weathers. I have not found this to be strictly fact; although it is not as much affected by dampness as an ordinary plate machine.† This machine is usually not encased,—a defect which I have remedied with satisfactory results. It is lighter and less expensive than the improved patterns of the Holtz model; but it is far less satisfactory in medicine, because it generates a much smaller quantity of electricity and has less intensity. The spark elicited may be a moderately long one (when compared with the radius of the revolving plates); but it is rather a thin spark at best,—thus confirming the view expressed by me respecting the quantity generated.

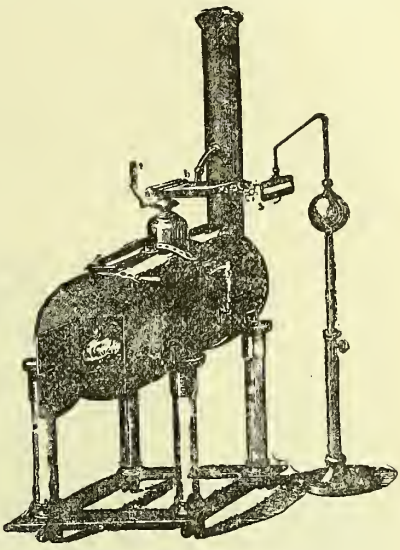


FIG. 169.—AN APPARATUS FOR GENERATING FRICTIONAL ELECTRICITY BY STEAM. (Devised by W. Armstrong). The legs upon which the boiler rests are of glass. The negative electricity generated by the machine when in action accumulates upon the boiler, and the positive electricity is collected by the comb upon which the steam-jets are directed.

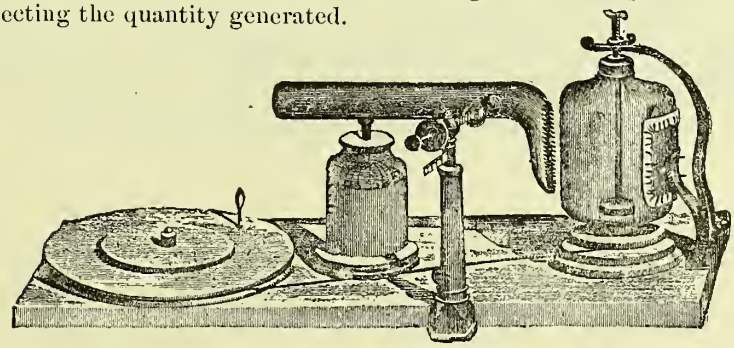


FIG. 170.—ONE OF THE OLDEST MODELS OF A CYLINDRICAL STATIC MACHINE.—The rubbers are grounded, and a Leyden Jar is connected with the positive electricity stored in the receiver.

THE PRINCIPLES OF STATIC INDUCTION.—The application of the principles of static induction, as demonstrated in the machine devised by

* I have lately had one so divided, which works admirably.

† This opinion is supported, moreover, by the fact that some manufacturers of these machines give to their purchasers explicit directions respecting the drying of the plates.

Holtz, is difficult to fully explain without devoting more time to the general subject of electrical induction than is deemed wise. It may be roughly summarized, however, as follows :—

Any body when electrified has the power, to a greater or less extent, of exerting (even through an intervening substance, which in this instance consists of a plate of glass) a peculiar effect upon the electrical state of another body closely adjacent to it in position. It tends to draw from the opposed body that variety of electricity which it does not itself possess. Now, if an intervening substance happens

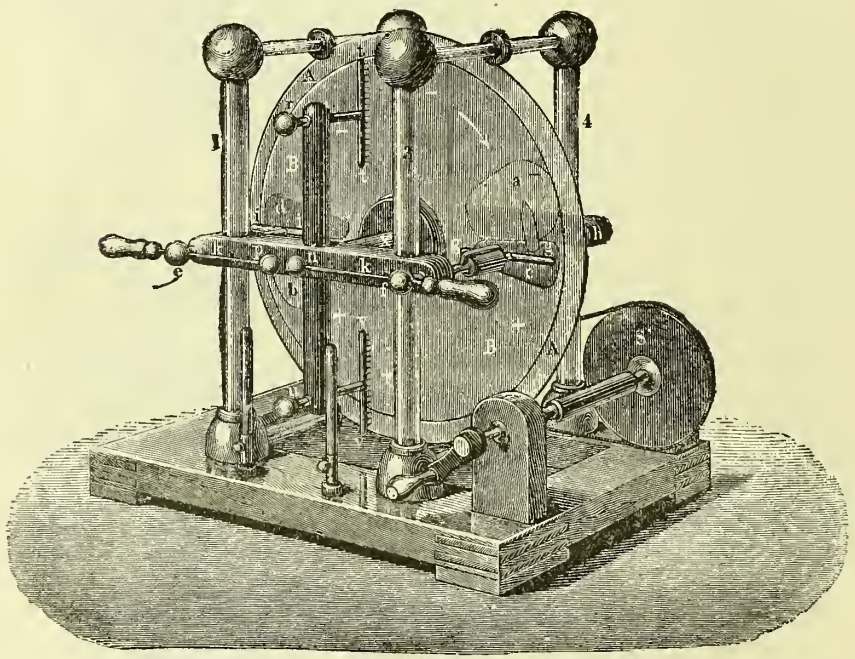


Fig. 171.—The Original Model of Holtz's Induction Machine with Vertical Plates. The same inventor also perfected a machine without win lows or armatures, in which two horizontal plates revolved in opposite directions. This machine is shown in a subsequent cut.

to exist between the two bodies, the electricity drawn toward it by induction may be deposited upon the corresponding side of that substance, and a proportionate amount of electricity of the opposite variety is abstracted from the intervening body. Hence, the intervening body becomes either positively or negatively electrified on one side, as the case may be.

In the induction machine, the intervening substance happens to be the revolving glass plate; and the opposed bodies are the two paper collectors and the two metal combs of the machine, which are separated by the revolving plate of glass.

In all induction machines, the charge is *practically constant when once established*, provided the mechanism be perfect and the plates kept absolutely dry. Under such conditions, it ought never to fail to produce its full effects when the wheels are set in revolution. This is a great desideratum in medicine.

In the original Holtz model only one stationary and one revolving plate were used. Both were circular in shape. The stationary plate had openings or "windows" cut in it. Paper collectors were glued to the stationary plate, so made as to project from it and to come in close contact with and to face the openings in the stationary glass plate. The revolving plate was insulated by legs of glass, while the stationary plate was not. Metal combs were used as terminal attachments to the inner end of the two poles of the machine. They faced the revolving plate and almost touched it.

You will find all of these mechanical features practically preserved in the improved models of to-day. The revolving and stationary plates have been increased in number, simply to augment the quantity of electricity generated. The stationary plates are no longer circular; they are made in two pieces, to allow of "windows." Two paper collectors are glued to each stationary plate. These terminate in points, which project into the "windows" made by dividing the plates. The poles of the machine have metal combs on one end and a brass ball at the other. Extra combs have been added to draw off residual electricity, which accumulates in excess; but these are "grounded."

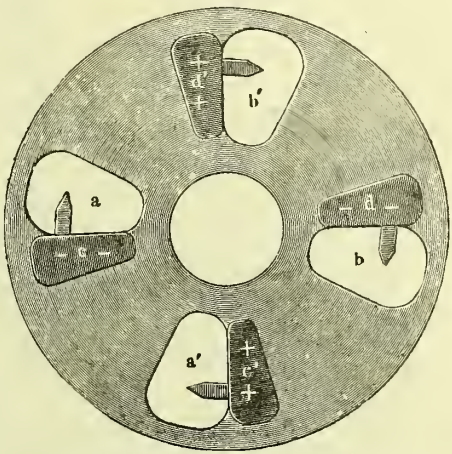


FIG. 172.—THE STATIONARY PLATE OF THE ORIGINAL HOLTZ, SHOWING ITS ARMATURES AND WINDOWS, WITH THE PROJECTIONS UPON THE ARMATURES.

Furthermore, the machine has been encased, simply to protect it from atmospheric changes. Cat-skin rubbers have been added. They are of use only as a means of exciting the plates when, from any cause, induction shall have ceased. We call them the "chargers" of the machine.

There have been many mechanical modifications made from time to time of the original model, which have not been here specified by me; but as they do not in any way affect the principle of electrical induction, they are not of importance in this connection.

In the original Holtz machine, a charge was primarily effected by rubbing a piece of ebonite briskly with cat-skin until it became highly charged with negative electricity, and then applying it closely to one of the paper collectors on the stationary plate of the machine. By the "law of induction" the comb opposed to this paper collector becomes electrically excited immediately. It at once deposits *positive electricity* on the side of the revolving plate nearest to the comb, and takes *negative electricity* away from the revolving plate. Thus the revolving plate becomes *positively electrified* to a very high degree at this point.

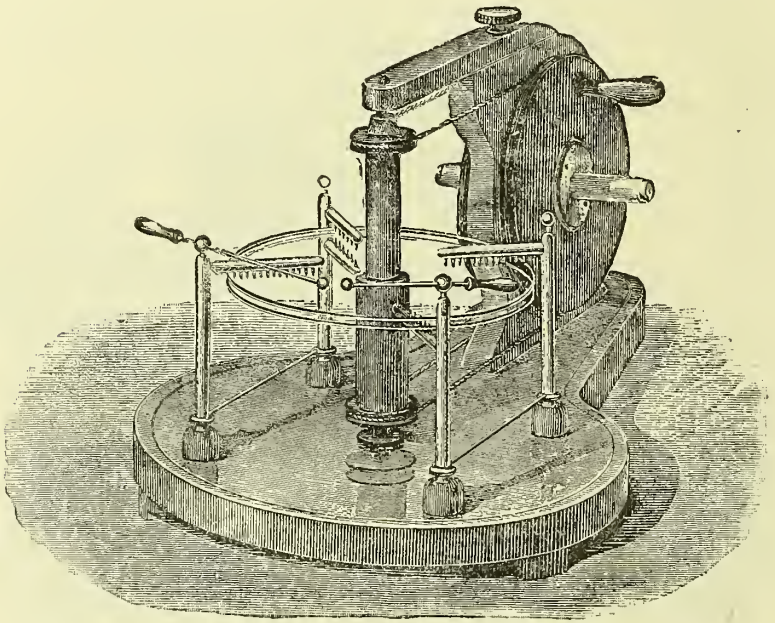


FIG. 173.—HOLTZ'S STATIC INDUCTION MACHINE, WITH HORIZONTAL PLATES.—The plates have neither windows nor armatures, and they revolve in opposite directions.

Now, when the wheel is made to revolve to that point where it meets the other paper collector upon the stationary plate, induction again takes place. Negative electricity is deposited (1) by the collector on the opposite side of the revolving plate (the side nearest to the paper collector), and (2) by the metal comb; at the same time positive electricity is taken from the adjacent side of the revolving plate by the collector, and also by the metal comb, from the opposite side of the revolving plate. This interchange of electricities charges the "positive pole" of the machine.

The revolving plate (now excessively charged with *negative electricity*) goes on to the next paper collector. Here a similar exchange

of electrical conditions occurs. The negative electricity is taken from the revolving plate by both the paper collector and the metal comb, and positive electricity is given to the plate in exchange from both of these sources. Hence the "negative pole" becomes highly charged.

As long as the revolving wheels are kept in revolution, this interchange of electricities continues at each of the poles; hence, the accumulation at each pole soon becomes sufficiently great to allow of an escape from pole to pole in the form of a spark, or into the atmosphere as a "luminous brush" easily seen in the dark.

STATIC ELECTRICITY AS COMPARED WITH GALVANISM.

It has been computed that the *electro-motive force* of a Holtz induction machine is 52,000 times as great as that of a Daniell cell (or 52,000 volts). It is not affected by the velocity of rotation.

The *quantity* generated is proportionate to the velocity of rotation and the number of wheels employed.* It is modified also by the moisture present in the atmosphere.

The *internal resistance* of the machine diminishes rapidly with increased velocity of rotation. It is not influenced by atmospheric conditions.

STATIC ELECTRICITY IN MEDICINE.

The revival of static electricity (or franklinism) as a therapeutical agent from the oblivion into which, for nearly half a century, it had unaccountably sunk, has been occasioned by several factors. Among these factors the following may be prominently mentioned:—

(1) The awakening of the profession at large to the fact that *electrical currents of different kinds have distinct therapeutical actions.*

These are not to be attributed to or confounded with the strength of the current employed, or its methods of application. The effects of faradization, galvanization, and franklinization upon animal structures differ widely in many respects. The time has come when an intelligent physician cannot justly condemn all forms of electrical treatment of any individual case, because he has failed to obtain satisfactory results with one of the above-mentioned currents alone; even if he has employed that particular form of current with the highest possible skill and judgment.

This is an error into which many are unwittingly led. I could report (if space would permit me to do so) the details of several cases where a failure to employ the proper current proved most disastrous to patients. One instance of this character (which was happily aborted) impressed me so forcibly at the time that it is possibly worth narrating:—

* On this account I have lately increased the size of the driving-wheel, so as to insure rapid revolution of the plates of the machine.

A patient, who had accidentally severed the musculo-spiral nerve by a pistol-bullet, was sent to me some years since for diagnosis, and to confirm or reject an opinion which had been expressed by a physician of prominence, namely, that the only hope of cure lay in a surgical operation for the uniting of the severed ends of the nerve by sutures. This opinion, as I found, was based upon the fact that the *faradaic current* had failed to produce any movement in the paralyzed muscles, and that several months had already elapsed since the accident, during which time the hand was steadily becoming more and more deformed by contracture of the flexor muscles of the hand and forearm.

My examination of the patient showed, however, that a *galvanic current* produced violent contractions of the paralyzed muscles when *passed through the injured nerve* (one pole being placed upon the sternum as a neutral point, and the other upon the musculo-spiral nerve); and the galvanic reactions of the nerve and its muscles furthermore indicated marked "degeneration" as having developed in the nerve below the point where it had been divided. Thus, the question of the advisability of an operation was decided positively in the negative. The nerve had already united.

In about eight months the injured nerve was completely restored by the use of the "static spark," the contracture had disappeared, and to-day the patient can see no difference in the usefulness of his hands.

(2) The *improvements which have been made in machines for the generation of static currents* for medical purposes have had much to do with the revival of this method of treatment.

Some of the cases reported in the earlier cyclopedias and antiquated works on electricity are fully as startling as those now encountered when treated with the improved machines; but, on the other hand, many failures to obtain good results must of necessity have occurred in olden times from the imperfect apparatus depicted in the scientific works referred to. Later, I will discuss the various improvements which have been made from time to time since Holtz first devised the present model of an induction machine (1865).

(3) *Improved methods of administration* of static currents have added materially to the effectiveness of this agent as a cure of disease. Some of these methods were unknown in earlier times (as far as my research goes to show).

(4) It is now known that a *considerable quantity*, as well as *length of spark*, is essential to the successful use of a static machine in medicine. Many of the static machines sold to-day are practically worthless, save as a toy, because they do not produce a sufficient quantity of electricity. The requisites of a static machine for medical purposes will be touched upon later.

(5) Experimentation with this agent seems to have confirmed the views of its enthusiastic advocates of the present day, and to support the accuracy of many of the observations reported in old scientific works. The incredulity of the past is rapidly being overthrown in respect to this method of treatment; and the special fields in which it proves of the greatest service are being definitely mapped out by those who are scientifically recording the results of its administration.

For the past few years I have devoted considerable attention to the

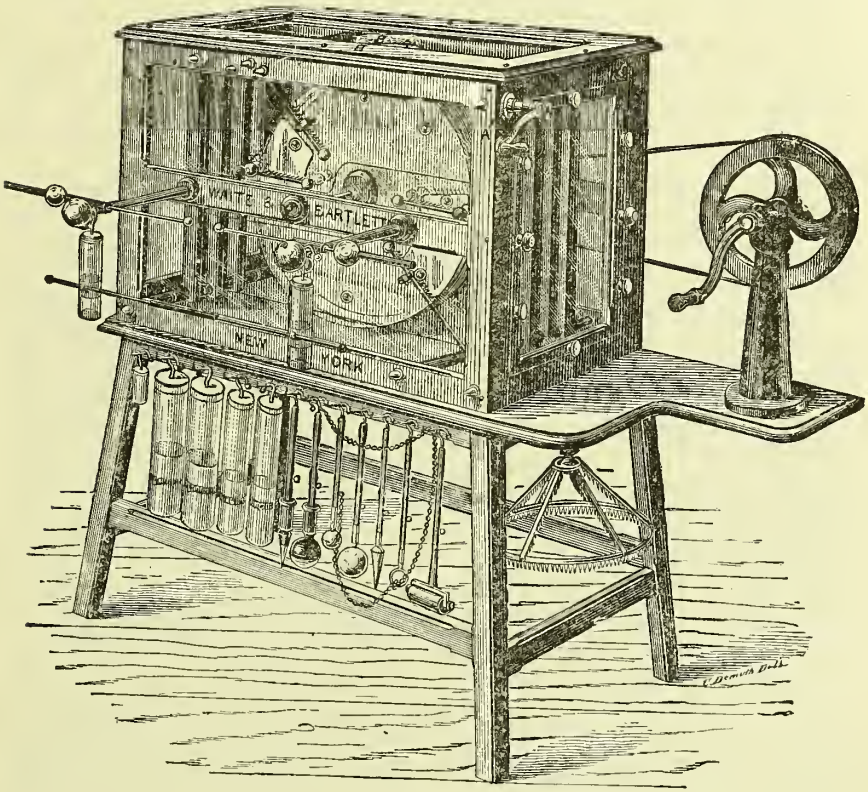


Fig. 174.—Holtz Induction Machine, as Improved by the Author.

improvement of the Holtz Induction Machine. Some of the results of my experimentation have already been published. The machine now described (Fig. 174) is in some respects an improvement upon the one which I originally introduced to the profession through the *Medical Record* of October 17, 1885. I have modified the charger originally used by me, so that it now bears upon the *outer revolving plates above the metal combs*, instead of passing between the central revolving plates. I have found that the application of cat-skin at this point on the outer

plates awakens the machine into action (when not charged) with greater rapidity and certainty than at any other part of the machine. Furthermore, there is no longer any difficulty in making the contact between the charger and the glass when the wheels are revolving rapidly;* while, with the old charger, the springing of the rubbers often rendered their insertion between the revolving plates (which are in extremely close approximation) a matter of some little annoyance at times. This modification in the charger has entailed a slight change in the mechanism by which the rubbers are brought into play when needed.†

*A *slight touch* of the charger of short duration upon revolving wheels, repeated at intervals of a second or two, is more effective than a long-continued application to the wheels of an induction machine.

† In some models of the present day, the charger cannot be raised from between the revolving plates. This is a serious defect.

NOTE.—The following description of this machine is quoted from the author's article in the *Medical Record*, October 17, 1885:—

"I would call the attention of the profession to an improved static machine which has been lately devised by me.

"It is the result of many months of experimentation, and is the outgrowth of the dissatisfaction which all other devices for generating electricity by friction have afforded myself, as well as others who have employed them. By those who have had experience with static machines, it is generally conceded that they are frequently charged with difficulty and give but a feeble spark during damp weather; and that they are particularly unsatisfactory and of little service during the summer months, when such conditions are liable to prevail. It is also conceded that some form of motor is generally required to run a machine of large size, because the hand is soon fatigued in overcoming the friction of the plates upon the rubbers, in addition to that produced by the bearings of the axle and the belt which connects the driving-wheel with the axle. Furthermore, it is now well recognized that plates of large size (16 to 24 inches in diameter), and several of them, are absolutely essential to a machine which is intended for medical use. Small single-plate machines do not give sufficient quantity or length of spark to be of any practical benefit as a curative agent. Again, it has been found by experience that building a glass case over a static machine does not thoroughly protect the plates of the instrument from dampness. No cabinet-maker can make joints of wood which will not admit of much moisture when the outside air is impregnated with it; and chloride of calcium, if placed within the case, will not absorb all the dampness that enters and collects upon the plates and metal of the machine.

"I found by experience that all the electrodes of a static machine (being insulated by glass) were liable to be easily broken; and that the poles of the instrument, when by accident exposed to outside violence or a blow, were also liable to cause a breakage of the glass windows in the case of the machine (which they perforate in most of the later models).

"In some machines, the case is, moreover, too small for the plates and allows of an escape of more or less of the electricity generated. The first machine which I made upon the present plan had this objection. It would give the operator an occasional shock in consequence of this defect when the hand was used as a motor, and it lost a large percentage of the volume of electricity generated by "grounding" that proportion which jumped to the metal parts of the case.

"Without entering further into numerous difficulties which I have had to encounter and overcome, I may summarize the more important improvements made in the machine shown in the accompanying cut, as follows:—

"(1) The case is so constructed that all of its joints are *packed with soft rubber* before its screws are tightened. This prevents the entrance of moisture from without, and makes the machine a useful one at all seasons of the year. Soft rubber now constitutes the best packing for steam apparatus, and is also used in hermetically sealing fruit-jars in preference to any known material. All imperfections in the joints of the case are perfectly sealed in this instrument.

"(2) The *doors of the case* are so arranged as to be drawn tightly in contact with a frame covered with soft rubber by means of milled screws.

"(3) The glass in the case is put into the frames with putty, which is impervious to air.

"(4) The poles of the machine perforate the wooden portion of the case, instead of the glass windows. They are insulated with hard rubber, and the apertures are protected by soft-rubber packing placed between hard-rubber buttons and the wood. Thus the danger of breakage of the case by accident is decreased without impairing its impermeability to dampness or allowing of leakage of the electricity generated when the machine is in use, while the strength of the case is materially increased by this modification.

"(5) The axle is so built as to reduce the friction to a minimum and to allow of its being oiled without opening the case.

"(6) The driving-wheel is very large, and rests upon a cast-iron support. This insures both ease of motion and durability.

"(7) The glass plates of the machine are nine in number. Six of these revolve, and three are stationary. The stationary plates are of peculiar shape. The revolving wheels are made of carefully selected glass, so as to be as true as possible and bear evenly upon the rubbers as they revolve.

Again, although chloride of calcium is not required during the cool months in this particular machine for the purpose of drying the air contained within the case (on account of the rubber packing between all the joints of the case, which almost hermetically seals it), I have found it desirable to use this or some other means of artificially drying the plates *during the summer months;* because the air is then excessively laden with moisture. To allow of the introduction of a tray containing chloride of calcium without opening the doors of the case I have been forced to modify the wood-work of the machine somewhat, and I have also raised the lower level of the stationary glass plates about two inches. By this means I can now slide a tray nearly the whole width of the case underneath the plates, and thus expose the air within the case to a large absorbing surface, which deprives it of moisture very rapidly.

I hope in time to so perfect my system of packing the joints and the openings in the case (entailed by the parts of the machine which must of necessity perforate it), as to make it absolutely air-tight at all seasons of the year. When this feat is accomplished, the necessity of chloride of calcium* or any artificial dryer within the case will have been entirely dispensed with; but until cabinet-makers can be found who never make mistakes, or a better material than highly finished and shellacked wood† can be obtainad from which to construct the framework of the machine, I fear this scheme will never be perfectly accomplished. Practically, however, this necessity is not so great as it might at first seem; because during the summer months the diffusion of static electricity into the atmosphere is so great as to seriously interfere with a satisfactory application of this agent to a patient by the methods known as "insulation,"

"(8) The exeitants consist of cat-skin rubbers so *arranged as to touch the outer plates* when the machine loses its charge. This eharger is a great improvement over all others previously employed by me. Metal buttons are also placed upon the outer plates, which as materially assist in charging.

"(9) The collectors have tinsel attachments which aid in gathering the electricity generated.

"(10) The electrodes are made with handles composed of hard rubber instead of glass. They are therefore less liable to be broken, and are as perfectly insulated.

"(11) Each machine is provided with three pairs of Leyden jars of different sizes. It is arranged also with hooks upon which the electrodes may be hung when not in use.

"In conclusion I would say that I have produced with the machine here described (24-inch plates), and now in my office, a spark of eleven inches in length during a muggy day in August, when most static machines would fail to charge. It runs, after a few turns by the hand, for nearly a minute without any power, and generates without interruption. Any boy of seven years of age can run it without fatigue for half an hour.

"The machine here described is manufactured by Waite & Bartlett, of New York City, who have been industriously occupied for several months in perfecting it under my guidance.

"The cost of this machine has been materially reduced from the schedule prices of other makers for similar instruments, rather than increased by the improvements made."

* In several instances I have known the *chloride of lime* sold in commerce to be placed within the case of a static machine. The result has been to almost ruin the metal parts of the machine. It took a mechanic nearly a week in one instance to restore the effectiveness of the instrument.

† It has been computed that fifty coats of shellac-varnish are requisite to prevent the penetration of gases through stone.

the "indirect spark," and the "static wind," in spite of a perfect generator. These methods, as well as other forms of application of static electricity, will be described later.

In other respects than those enumerated, the modified Holtz induction machine introduced to the profession by myself some time since remains practically unaltered. Its effectiveness seems to have been pre-ëminently satisfactory to those who have used it, and the quantity and length of spark which can be elicited is as nearly an approach to the maximum of its theoretical quantity and power as could be hoped for.

No static machine can give off a spark greater than the radius of the revolving plates. I have frequently demonstrated a spark of eleven and a half inches from a wheel of twelve-inch radius. Furthermore, I think I can justly claim to have so improved all previous models built on the original Holtz plan as to insure a continuance of the charge throughout nine months of the year without recourse to artificial means for drying the plates. When properly cared for and handled, there is little necessity even for a charger during these months.

Before I pass to the consideration of static electricity as a therapeutical agent, it may be well for me to state that the cost of a static induction machine with plates of twenty inches diameter or over must of necessity be large; although the cost has been materially reduced of late by competition and improved methods of manufacture.

Again, it is impossible to transport a static induction machine from house to house without danger of breakage and the employment of a cartman; hence it becomes a part of a physician's office outfit only, and cannot be used in medical practice except by bringing the patient to the machine or going to some expense and risk in transporting it.

Finally, a static machine of the induction model requires a certain amount of care; otherwise the effectiveness of the instrument is liable to deteriorate, and its component parts to become more or less injured.

There is another form of static machine (already described as the Toepler model), which has been sold extensively to the profession. It has no case to protect it from the atmosphere. It can therefore be more readily transported, and it costs much less to manufacture than the induction model; but, on the other hand, it is far less effective, and cannot be favorably compared with the more expensive machine as a part of a physician's office outfit. The *quantity* generated by such a machine is necessarily small; and it is more or less seriously affected by atmospheric changes. In spite of the fact that some of the later authorities on electricity speak in its praise, I cannot give it an unqualified indorsement. It may serve the requirements of scientific institutions admirably; but it is, at best, but a make-shift for the neurologist. I think that I am sustained in this opinion by those who have had experi-

ence with the two models, when provided with all their latest improvements. I have been experimenting for some months to devise a cheap static machine which patients can use at their homes, and I think I have succeeded in producing a tolerably effective instrument; but I should never advise a physician to purchase one for his own use, if he could afford to buy an improved Holtz induction machine.

A Static Outfit.—The cost of an improved induction machine of the latest pattern varies from $250 to $350, according to the size and number of the plates; hence, this is a matter to be carefully considered before purchasing one. It is advisable, in my opinion, to have not less than six revolving and three stationary plates. The revolving plates should not be below twenty inches in diameter. I prefer one with twenty-four-inch plates, for medical purposes, over those of less power.

The *attachments* which should be purchased with such an instrument comprise:—

(1) An *insulated platform.* These may be made to seat one, two, or more persons at a time. I use for legs the heavy glass insulators employed by telegraph companies upon their poles. They are very strong and cheap, and have another advantage, namely, that they can be screwed up and down upon a wooden pin which perforates their central orifice. This admits of leveling the platform, in case the floor of the room has settled.

(2) A *set of electrodes.* This item comprises a large and small brass ball, a metal point, a wooden point, a roller of metal and of wood, an umbrella-electrode, some sponge-covered electrodes, a pistol-electrode, and a ring to hold the chain away from the patient during the applications. The handles should be long, and made of hard rubber or of glass.

(3) A *set of brass chains* of varying lengths.

(4) A *set of hooks* for attachment to the ends of the chains.

(5) A *set of heavy insulated rheophores* of varying lengths.

(6) *Three pairs of Leyden jars* of different sizes. I use those of 3-inch, 1½-inch, and 1-inch diameter, respectively.

(7) A *wooden chair* or *stool* which fits the insulated platform.

(8) A *connecting brass rod*, for use when the Leyden jars are employed.

(9) Some pieces of cat-skin.

(10) Several bottles of well-selected chloride of calcium.

The Care of an Induction Machine.—A few suggestions of practical value may be made upon this subject.

It is advisable, in the first place, that an induction machine should be placed in a perfectly dry room, *well lighted by the direct rays of the sun;* and, when possible, in close proximity to a window which shall allow the sun's rays to fall directly upon the glass plates of the instru-

Carbon Electrode, round end.

One and a quarter inch Brass Ball.

Brass Brush.

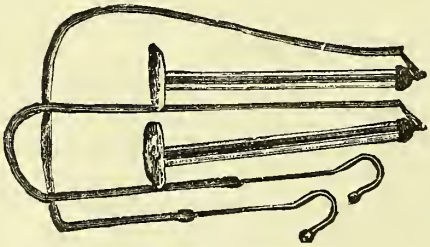
Rubifacient.

Wood Ball.

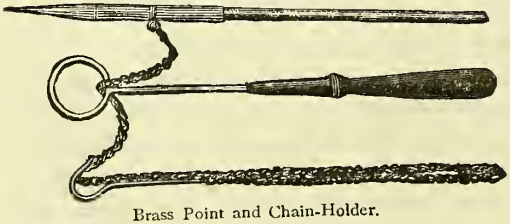
Wood Point.

Two and a quarter inch Brass Ball.

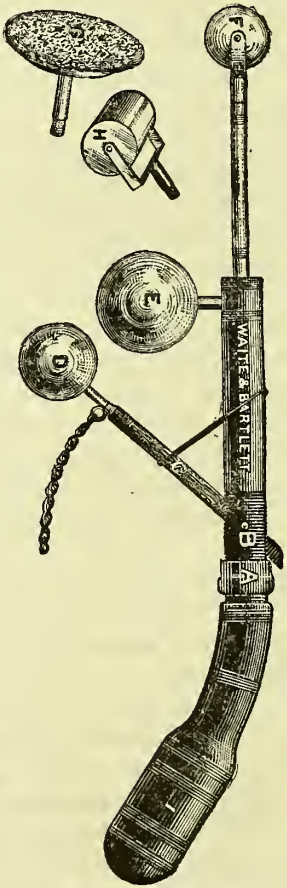
Spinal Roller for Massage.



Handles and Sponges.



Brass Point and Chain-Holder.



Morton's Pistol-Electrode.

FIG. 175.—ELECTRODES EMPLOYED WITH AN INDUCTION MACHINE.

ment. By this step we obviate dampness, and thus insure the greatest effectiveness of the machine.*

In the second place, the *metal parts* of the machine and the metal electrodes should be *rubbed briskly every morning* with dry chamois-skin or silk. Accumulated moisture on the poles or electrodes is a serious drawback to successful static applications.

In the third place, although the *metal parts* of the machine are shellacked when made, they are apt after a lapse of time to require *repolishing* with emery-paper, powdered emery, or rotten stone. A light coat of shellac-varnish should be given these parts after their brightness has been restored, and all grease or moisture thoroughly removed from them.

Again, it becomes necessary, at intervals, to *oil the bearings* of the wheel-axle and the plate's axle; also to occasionally tighten the leather belt,† and to re-shellac the case if it becomes blistered by the sun. The latter step tends to exclude the entrance of moisture within the case through the pores of the wood.

During the summer months *fresh chloride of calcium should be constantly kept within the case.* It should be renewed whenever sufficient fluid appears in the tray to become evident to the eye. Unless the case is packed with rubber, this method of artificially drying the air must be employed at all seasons of the year. A few drops of petroleum on the floor of the case help to prevent the accumulation of atmospheric moisture upon the plates.

Occasionally, the best machine will lose its charge. Should it do so, you will probably find that one of the following causes has led to this result :—

(1) The servant, or some inquisitive person, may have *turned the revolving plates in the wrong direction;* this causes the accumulators to lose their electrical state and thus to arrest "induction" through the glass plates.

(2) *Atmospheric moisture may have entered the case* and been deposited upon the plates. In all models that I know of, but my own, this occurrence must of necessity be very frequent, as no safeguards exist to prevent it.

(3) The instrument may have been left, after an application to a patient, with both the *poles* "*grounded*" by means of the chains dangling from them and resting upon the floor. This oversight may not prove serious in dry, cold weather; but, it is never advisable to leave the chains attached to the poles when the instrument is not in use.

* I have my own in a bay-window, where the afternoon's sun has free access to it.

† Thumb-screws beneath the driving-wheel post are provided for this purpose in my model.

(4) The plates may have loosened from the axle; and, in consequence, some may fail to revolve properly. To obviate this occurrence, double nuts should be used on the plate-axle.

(5) The *combs may have become displaced*, so as to touch the glass or to bear an improper relation to the paper collectors.

(6) The *case may be too small* for the plates; and thus allow of escape of the electricity to the ground. This will be very apparent to the eye when tested in darkness.

The Charging of a Machine.—It is well to know what steps are necessary to start a static induction machine, in case it loses its charge. I have seen a few instances where the owner of such an instrument has worked himself into a heat of passion as well as of body by fruitless attempts to obtain a spark, while a patient calmly waited with expectancy for the successful termination of his feat. Some of my readers may have had such an experience. I suggest, therefore, that they follow the directions given, with some regard to their details:—

(1) See that the *plates and charger are dry*. If not, you can easily render them so by exposing the machine to strong sunlight, and by putting an abundance of chloride of calcium in trays at the bottom of the case. This may require some hours of delay. Always open the door of the case if the sun's heat be used; and *close them tightly* (by means of the milled screws which perforate the door) as soon as the machine regains its charge.*

(2) After you have got the plates thoroughly dry, start them in rapid revolution by turning the driving-wheel *from left to right* as you stand facing it. Now *apply the chargers lightly near to the edge of the revolving wheels for a second or two*, and then sweep them across their face at intervals of a few seconds, until the machine starts. The poles should be approximated to within one-half inch, and the chains should not be connected with the poles.

(3) If the machine fails to start, in spite of these directions, you can then *take a piece of cat-skin* and *warm it thoroughly over a gas-jet*. Then set the wheels in rapid revolution and apply the warmed cat-skin as a rubber (to the plate with the buttons on it) as *close above the metal*

* If you cannot spare the time for these procedures, a large alcohol-lamp may be lighted within the case. The air may thus be heated sufficiently to temporarily render the machine useful. I am aware that I have been criticised (in a carping spirit) for offering this suggestion in print; but, as a *temporary expedient*, it oftentimes proves a valuable aid in rapidly regaining a lost charge, and rendering an induction machine efficient.

I have frequently known the nozzle of a hot-air furnace (such as is used in giving a hot-air bath to a patient beneath the bed-clothes) to be directed into the case of an induction machine for the purpose of drying the plates when very damp. At one time I tried to build a machine with a tube passing through the case, by means of which the air in the case might be heated indirectly without opening the door; but I found it impracticable, for many reasons.

comb as it is possible to hold it. This seldom if ever fails; but it requires the opening of the door of the case.

(4) Be sure that the *poles are well dried* with chamois-skin before the machine is put in action; also, that the poles are closely approximated, but not in contact.

METHODS OF APPLICATION OF STATIC ELECTRICITY.

Static electricity can be applied in several ways to a patient. Each of these methods has some therapeutic effects which are peculiarly its own. Moreover, the sensations experienced by the patient during the application are greatly modified by the method employed. For these reasons, it is necessary to go into greater detail respecting the management of a static machine than that of any other electrical apparatus in medicine with which I am familiar.

We can apply the static current to a patient in the following ways :—

(1) By the "*indirect spark.*"

(2) By the "*direct spark.*"

(3) By the "*Leyden-jar spark*" or "*static shock.*"

(4) By "*static insulation.*"

(5) By the "*static breeze.*"

(6) By the "*static induced current.*"

The Indirect Spark. — To administer static electricity by this method, the patient is first placed upon the insulated platform, and sufficiently removed from all surrounding objects to prevent the escape of the charge from the patient to them. The machine is then connected with the patient by a chain, which is either held or simply attached to the stool on which the patient sits. The chain must be sufficiently elevated from the floor to prevent "grounding" of the current. It may be attached to either the positive or negative pole of the machine, according as the operator may desire positive or negative insulation. A chain is then attached to the other pole of the machine, and is "grounded." This can best be effected by attaching it to the gas-fixture or a faucet attached to a constant water supply. If this is not convenient, the chain may be thrown upon the floor, when not carpeted, in case the generating power of the machine is ample.

The *poles of the machine are now widely separated* and the wheels put in rapid motion. You will notice that the hair of the patient immediately rises; and, in a dim light or total darkness, you should perceive a peculiar purplish light escaping from the tips of the finger-nails, the hair, and other parts of the body which are more or less pointed. The rapidity of this escape is influenced (1) by the extent of the charge; (2) by the proximity of a part to some surrounding object; and (3) by the condition of the atmosphere, as regards its moisture. As the patient

moves his finger-tips near the door-casing or some article of furniture not insulated, you may be able to perceive this escape of electricity, even in a strong light.

Now we have a condition which is known as "static insulation." If the machine is a powerful one, it may be carried to a high point. The patient happens to be charged, in Fig. 176, with positive electricity, because he is connected with the positive pole of the machine.

The final step consists in presenting to the part which you wish to influence a brass ball on the end of an insulated handle. This electrode is connected, as you see, with a gas-pipe by means of a brass chain. A water-pipe makes an equally good connection. When this ball reaches a certain degree of proximity to the patient,* you notice that a discharge

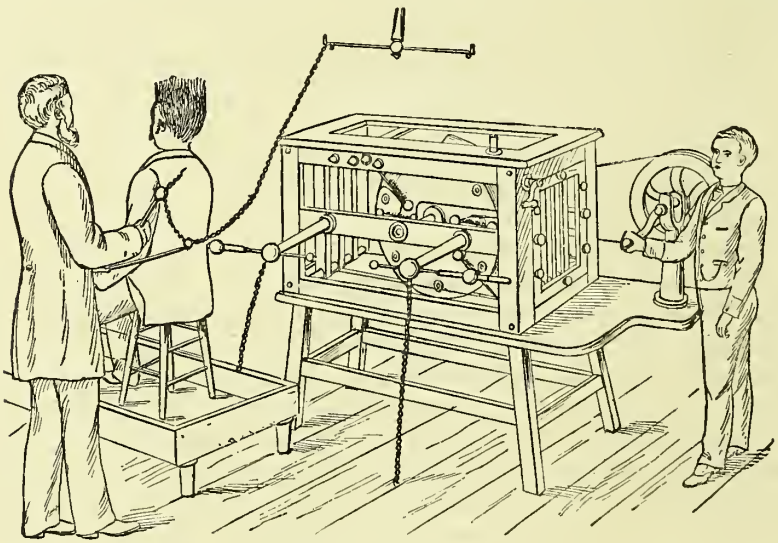


Fig 176.—The Indirect Spark.

of the accumulated electricity occurs in the form of a "spark." This is known as the "indirect spark," because the electricity takes an indirect course (through the earth) to form a circuit. It leaps from the patient and escapes to the earth down the gas-pipe or whatever grounding the electrode may chance to have.

The *length* of the "indirect" spark is directly proportionate to the *generating power of the machine*—supposing, of course, that all other factors in the application are equal (such as the humidity of the atmosphere, the completeness of insulation, etc.).

*The length of the spark elicited depends on the power of the machine, the dryness of the atmosphere, and the perfection of the insulation of the patient. I frequently have drawn a spark of eight inches by this method.

The *volume* of the spark is modified by the *size of the brass ball* on the end of the electrode. A large ball will produce a heavier spark than a small one.

The therapeutical effects of this method of application will be discussed later. I would call attention, however, to the violent muscular contractions which occur with each spark.

This method is somewhat painful. The withdrawal of a spark leaves a "weal" or lump, which somewhat resembles a recent mosquito-bite. They almost entirely disappear when friction is employed after the application; hence, it is my custom to rub the part with my handkerchief, if exposed (the face or hands, for example), after the application.

I would caution you here, in passing, against giving static sparks (by any of the three specified methods) to a patient on his first visit. You are apt to frighten a patient, unless he is well prepared for it. Again, patients grow tolerant of this method of treatment after a while; hence, you can gradually increase the volume and length of spark at successive sittings without endangering the patient's confidence in you or creating alarm. You can regulate the length of the spark by the speed of revolution of the plates of the machine.

For some hours after such an application the patient feels a sense of heat at the spot where the spark occurred. This is not at all unpleasant to many. Some patients even speak of it as agreeable.

By using a *wooden ball* in place of a brass one, a number of very fine sparks are simultaneously elicited—giving to the patient a feeling aptly compared to a "shower of sand." This electrode is admirably adapted for use about the eye or the face, although this is not the limit of its usefulness.

Finally, it is not essential to this form of application the that clothing be removed; as the finest silk or woolen fabric is not injured by it. This is a great point in favor of static application, especially in the treatment of females.

It is customary to use a second electrode with a *ring of brass* attached to the insulated handle, through which the chain is passed before it is attached to the ball-electrode. This is to keep the chain away from the patient, so that sparks will not be caused at points where you do not desire them to occur. A little practice will enable you to handle both with one hand, while you turn the wheels of the machine with the other. Sometimes it may be necessary to have the patient stand rather than sit upon the insulated platform while these applications are being made.

Let us pass now to the second method enunciated.

The Direct Spark.—By this method, the circuit between the poles of the machine included the patient only. He sits on the insulated

platform, which is connected with one pole of the machine; or one pole may be directly attached to some particular extremity of the patient, when the effects of the current are to be concentrated as much as possible upon that member. The electrode is attached to a chain, which is fastened to the other pole of the machine. The length of spark to be administered is regulated by the *extent of separation of the poles of the machine* and the speed of revolution of the plates. The farther apart the poles, the longer and more severe is the spark.

The ring electrode is employed (as in the former method) to protect the patient from an accidental contact with the chain attached to the electrode.

In neither this nor the method previously described are Leyden jars employed.

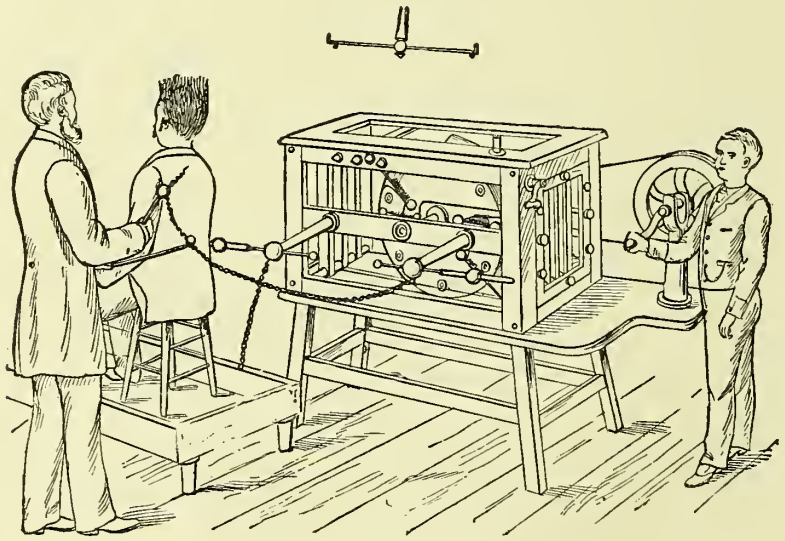


Fig. 177.—The Direct Spark.

I question, personally, whether the selection of the poles for the attachment of the electrode has much, if any, influence over the therapeutical action of the "direct" spark. If it has, I have not as yet clearly formulated in my own mind any deduction respecting this point.

Static Shock, or the Leyden-Jar Spark.—This method of application is accomplished by first *attaching a pair of Leyden jars* to the poles of the machine, and *connecting their outer covering of tin-foil by a brass rod.*

The poles of the machine are then brought into close approximation; because the strength of the shock is modified (1) by the *size of the jars*, and (2) by the *separation of the poles.*

As this method is, at best, a very severe form of application, it is well to begin with very small jars, and to place the poles as nearly in

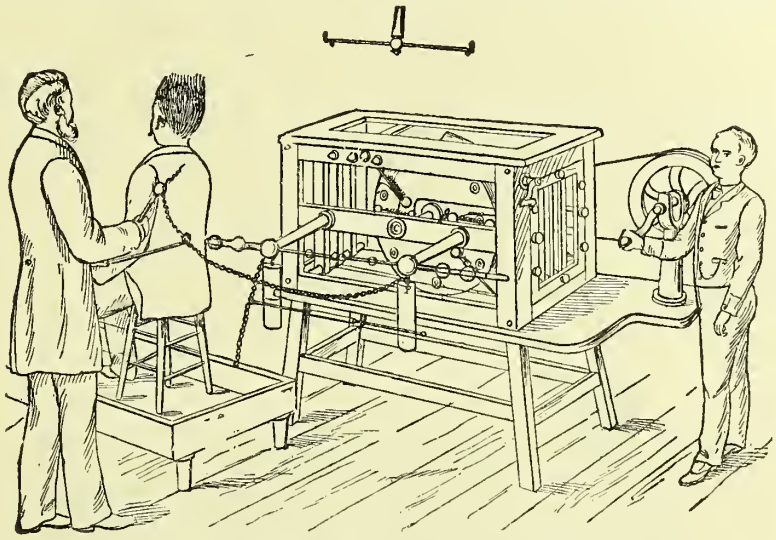


FIG. 178.—SHOCK WITH LEYDEN-JAR DISCHARGE.

contact as possible (without actually touching each other). They can then be separated at will, as the exigencies of the case seem to demand.

The chains are arranged in a similar manner to that described in the preceding method (direct-spark application).

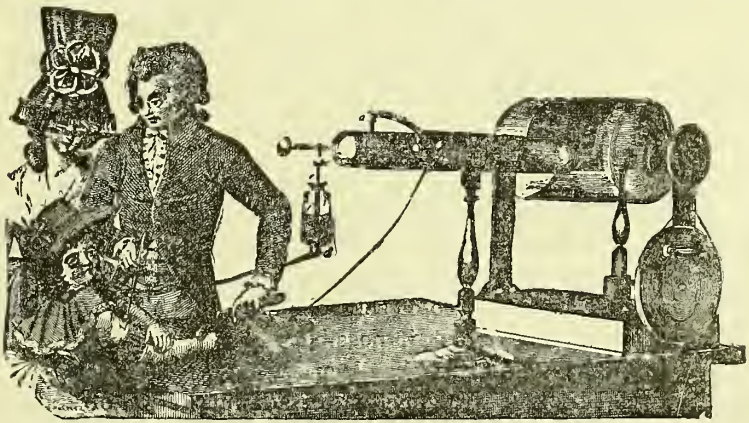


FIG. 179.—AN APPLICATION OF THE LEYDEN-JAR SHOCK DURING THE EIGHTEENTH CENTURY.
(Copied from an old English work.)

This method is best applied to the bare skin. The polarity of the electrode is not, to my mind, a matter of much consequence.

I advise you to handle this form of treatment with extreme caution.

I have several times accidentally received a moderate static shock, and I can assure you it is not associated with pleasurable sensations.

Static Insulation.—This method has already been described in connection with the administration of the "indirect spark." It is, perhaps, the most agreeable of all methods of static treatment. The patient is simply charged for a variable space of time (three to twenty minutes) with either positive or negative electricity. The pole of the machine is attached to the insulated platform on which the patient sits or stands. The other pole is "grounded" by a brass chain running to the floor, a water-pipe, or a gas-fixture.

The *poles of the machine are as widely separated as possible* before the wheels are set in revolution.

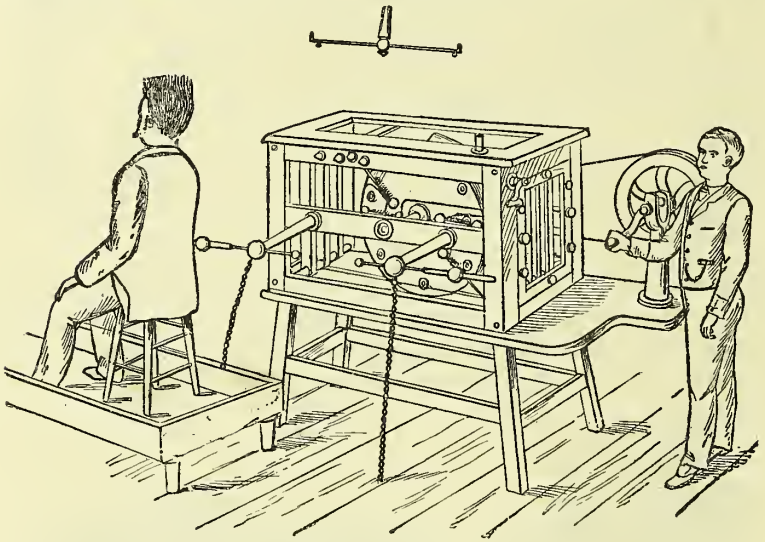


Fig. 180.—Static Insulation.

No pain is experienced. The hair becomes erect, unless very much oiled. The patient experiences a peculiar "tingling sensation," with a tendency toward perspiration if the administration is long continued. If you approach the patient too closely, a spark is elicited at the nearest point. This should be avoided, if possible.

Its therapeutical effects will be discussed later.

The Static Breeze.—This method of administration of static electricity consists in the withdrawal of the static charge from a patient by means of an *electrode of metal or wood, which is pointed.*

If the breeze be *indirectly* induced, this electrode is grounded by a chain attached to a gas-pipe, a water-faucet, or placed in contact with a wood floor when the other connections are not easily accessible. The

patient is first insulated (in order to retain a charge), and is then connected with one of the poles of the machine by means of a chain, which

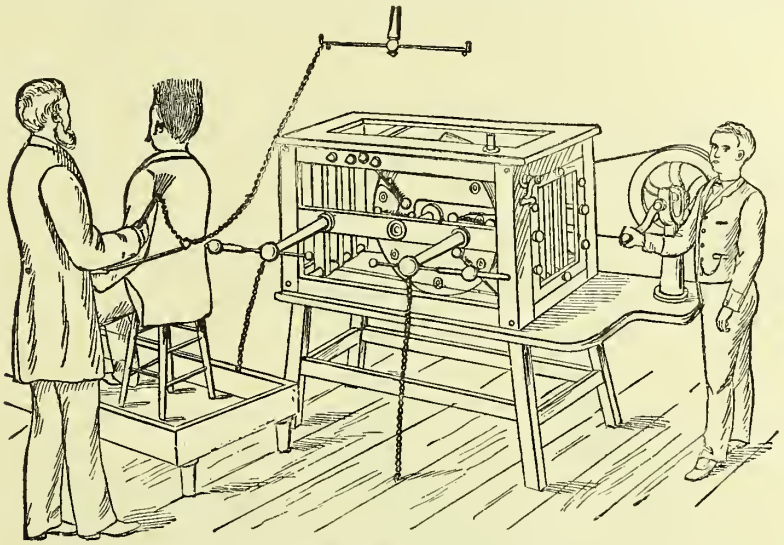


Fig. 181.—The Indirect Static Breeze.

he either holds or fastens to the platform upon which he sits. The electrode is then employed.

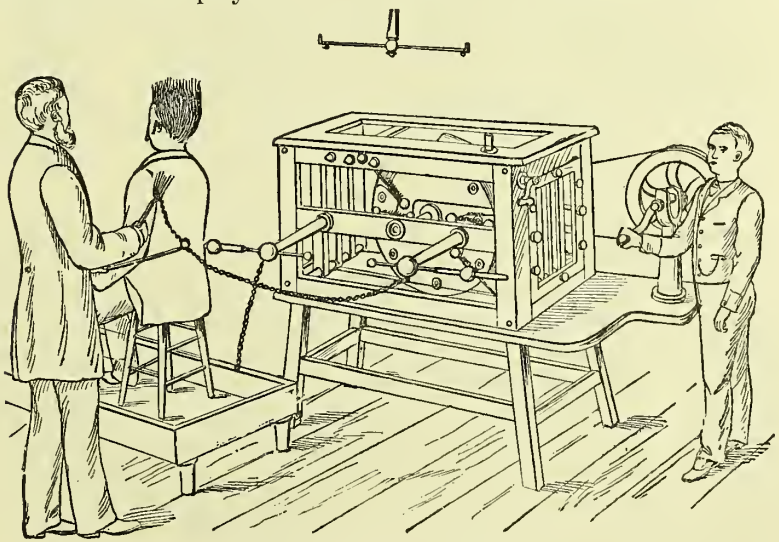


Fig. 182.—The Direct Static Breeze.

When the breeze is *directly* induced, the insulated stool is connected with one pole of the machine, and the electrode with the other pole

If the electrode be *a metal one*, the electricity is drawn rapidly from the patient at the point which is nearest to the electrode, and a sensation resembling that of a breeze is experienced at the spot where the electricity escapes. Single or multiple points may be employed on the electrode.

In either of these methods, when the electrode is *composed of wood*, the sensation is modified, to a certain extent, by the poor conductivity of the wooden point. Most patients compare the effect of such an application to a "shower of sand" concentrated upon the point of withdrawal of the charge.

When this method is employed about the eye, the wooden ball or wooden point is usually preferable to one of metal.

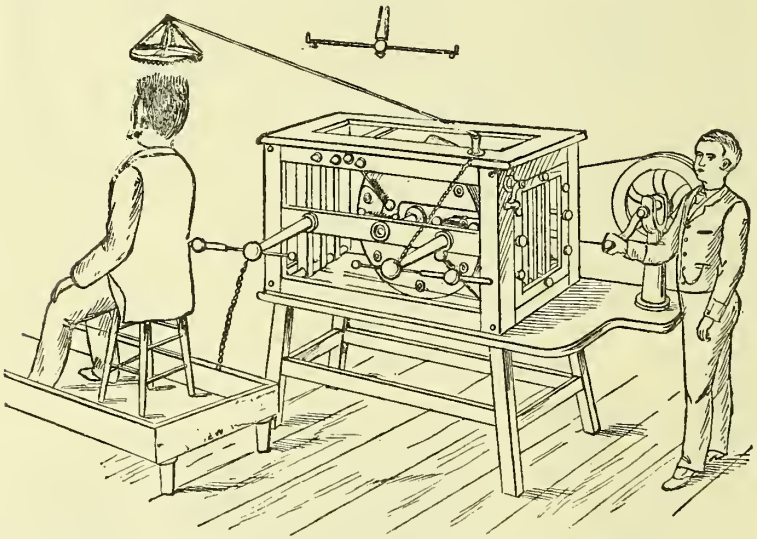


Fig. 183.—The Electrical Head-Bath, a Variety of Administration of the Static Breeze.

When application to the head and scalp are deemed requisite, a metal cap studded with points is hung over the head of the patient by a chain, which is grounded. This cap is known as the "umbrella electrode." It should not touch the patient's head or hair, when he is placed beneath it upon the insulated platform. The numerous points of the electrode draw off the electricity through the hair and scalp, which passes from the machine to the patient, and produce a sensation which is particularly pleasant. A "strong wind" is felt permeating the hair and encircling the head.

Static Induced Current.—To convert a static machine into what, to all practical purposes, may be considered a "Faradaic" instrument, some slight modifications only are required.

The discovery of this method may justly be attributed to the investigations of Professor W. J. Morton, of New York; although Matteucci first devised an instrument which gave shocks by induction simultaneously with the discharge of a Leyden jar. (See Fig. 795 of Ganot's work on "Physics," by Atkinson.)

To produce this form of current, it is necessary to first *hang a pair of Leyden jars* upon the arms of the machine. The size of the jars employed modifies the strength of the current; hence it is necessary to have jars of different sizes as a part of the static outfit. You now attach the chains or, by preference, insulated wires, which serve to connect the machine with the patient, *upon the hooks that rest upon the outer coating of the jars.* Finally, you attach to the other end of each rheophore an electrode for use upon the body of the patient. The electrodes may be

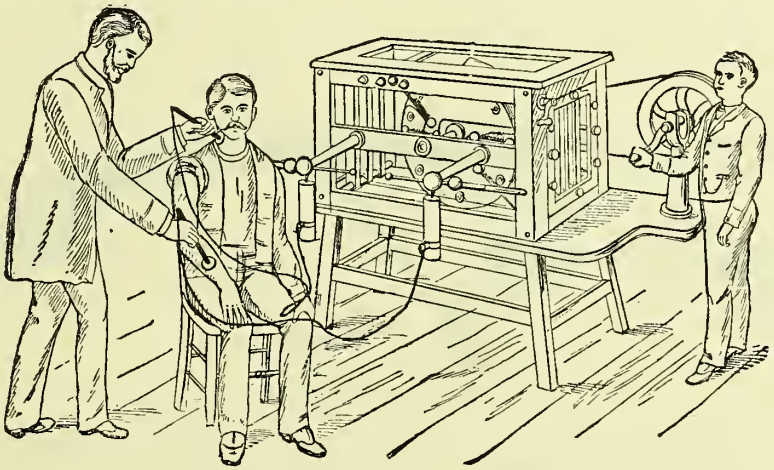


FIG. 184.—THE STATIC INDUCED CURRENT.

of metal without any covering, or ordinary sponge-covered electrodes may be employed (care being taken that the insulating handles are somewhat longer than usual).

Before the machine is set in motion, *its poles should be approximated closely.* This step is important, because the separation of the poles intensifies the current, as long as a spark will pass between them. There are two factors, therefore, in determining the strength of the static-induced current:—

(1) The size of the jars.

(2) The extent of separation of the poles.

Dr. Morton has devised an ingenious electrode which allows of an application of this form of current to a patient without disturbing the poles of the machine; but it is not an essential part of a static outfit, because an interruption of the current can be accomplished without it.

This electrode represents a simple mechanical means of putting into practice the method discovered by him in 1880, of converting the static electric charge into dynamic electricity or current. Electric nerve-and-muscle reactions had previously been obtained by means of the interrupted galvanic and the faradaic currents. It has also been noticeable that the "spark" discharged on a nerve motor-point, or over a muscle, produced the characteristic reactions. But the spark was painful, and difficult to direct accurately, particularly about the face and head. To avoid these objections, Dr. Morton arranged this electrode, by means of which the disruptive discharge or spark of static electricity takes place between two brass balls, one of which is in relation with the "ground," while the other is connected to an ordinary moist-sponge electrode. This in turn is applied at the point desired of the patient charged on the insulated platform.

As a result, for every static discharge occurring between the two brass balls there is a dynamic discharge or current at the point where

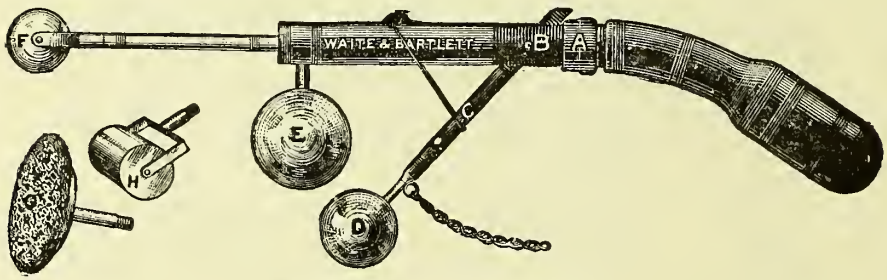


Fig. 185.—Morton's Pistol-Electrode.

the sponge is applied, and the nerves and muscles may be stimulated, or rather "irritated," exactly as by the ordinary interruptions of battery currents, direct or induced.

This electrode is another means of converting static into dynamic electricity, based upon the principle described under the name of "static induced current" by Dr. Morton.

This "static induced current," as has been already stated, was obtained by attaching ordinary sponge electrodes by their connecting rods to the outer layer of tin-foil on the Leyden jars. The patient, in other words, replaced the usual connecting rod between these coatings. On putting the machine in motion and causing a spark between the poles, a "current" was felt by the patient, no insulation of course being required. The advantage of this method is that the ordinary Holtz machine may when required be called upon to perform the work of an ordinary faradaic or induction machine.

The greatest event after its discovery, in the history of medical

statical electrization, or franklinism, was the invention of the Holtz or induction machine in 1865. Next in importance, perhaps, is the method discovered and put into practice by Dr. Morton, in 1880. of converting the static discharge into a dynamic discharge or current, and the electrode represented on opposite page is the only novel electrode of any importance not bequeathed to us by the medical electricians previous to 1880.

The difference between the "static induced current" and the faradaic current is this: The former has a *fixed polarity and direction*, and *greater electro-motive force*. It is far less painful, also, than is the faradaic current when the electrodes are widely separated.

My attention has lately been drawn by Dr. Morton to another device of his for the prevention of the escape of sparks from a sponge-covered electrode while employing the static induced current. It consists in applying a coil of copper wire closely upon the flat surface of the metal end of the electrode which is covered by the sponge. This is done in order to prevent the concentration of the current at any one point on the surface of the electrode while in use.

He has also been experimenting of late upon the effects of deriving currents for medical purposes from a helix of insulated wire wound upon each of the Leyden jars of a Holtz induction machine.

I have not yet tested the working of either of these later appliances but I shall do so soon, when I am provided with the necessary apparatus.

PART II.

ELECTRO-THERAPEUTICS.

Galvanic Measurement and Dosage.—Quite an active discussion of this important subject has been indulged in through the columns of various journals during the past year. Prominent among the participants in this discussion may be mentioned, Drs. Rockwell, Martin, Engelmann, Massey, and Bailey.

The fact must be apparent to any one who has watched the progress of electro-therapeutics with interest that the professional mind is at last becoming keenly alive to the necessity of knowing exactly how much electricity is being administered to a patient at each sitting. The method commonly employed in the past of noting the number of cells used (even this precaution not being always taken by some writers) is only an approach to a scientific record of a case. It offers no opportunity for advancement in our efforts to bring electro-therapeutics to a tangible basis of scientific precision.

A few hints previously offered respecting the important subject of galvanic dosage may therefore be repeated here with possible benefit to the reader.

Galvanic cells differ in their *electro-motive force* according to their mechanical construction, viz., the character of the elements employed, the internal resistance, etc. On the other hand, the *quantity* generated depends upon the size of the elements or the extent of their immersion, and the character of the excitant, when all other conditions are practically similar.

For example, two galvanic cells of identical make and size will not generate the same quantity when one has been exhausted and the other has been freshly filled; or when one is polarized and the other is not polarized.

The electro-motive force has no relationship with the size of the elements, but the quantity generated has. A stream may flow five miles an hour, be it a brook or a river; but the quantity of water that passes a given point in the same interval of time is not the same in each case, as the merest child can easily understand. On the other hand, a lake at a height of one hundred feet will exert no greater pressure through a pipe leading from it to the ground than would a tea-cup at the same elevation, if kept constantly filled. The quantity is determined in the first instance; the pressure (or electro-motive force) is illustrated in the latter. The bursting of water-pipes in our dwellings is not induced by the size of the distributing pipe nor by the size of the reservoir, but by the height of the original source of supply.

Again, a bullet, when propelled from a weapon with a given charge of powder, will penetrate a board more deeply than a piece of iron. So it is with electric currents. A current of a definite electro-motive force will travel faster through some tissues than others; will penetrate the skin of a thick palm with greater difficulty than the thin skin on the back of the hand; will be facilitated in its passage by large electrodes and retarded in proportion as their size is reduced; will be aided in many instances by the saturation of the electrode (especially with a saline solution), and will be retarded by the absence of such conditions; will be aided by a close approximation of the electrode to the surface of the body, and retarded by an imperfect approximation of the electrode.

Currents derived from a galvanic battery invariably enter at the positive pole (the anode) and escape at the negative pole (the cathode). Now, a bullet, when shot through a board, tears most at its point of exit. We may consider the negative pole as that which tends chiefly to stimulate the parts with which it comes in contact or upon which its effects are indirectly exerted, while the positive pole is to be clinically regarded rather as the sedative pole, in contradistinction from the negative. The positive pole is acid in its reaction, while the negative is alkaline.

When we bear the essential facts of electro-physics in mind, many

of the difficulties of electric measurement are more clearly appreciated, as well as some clinical facts that are indirectly related to this subject.

To accurately determine the dose of electricity which the patient is taking, several factors have to be considered:—

In the first place, the number of milliampères (the unit of current-strength in medicine) may be measured by a reliable milliampère-meter.

This instrument must be thrown into the circuit,—*i. e.*, between the positive pole of the battery and its negative pole,—the patient being similarly interposed and constituting the main resistance offered to the current which is supposed to be passing. The resistance of the rheophores and of the galvanometer itself must be added to that of the patient in determining the total of external resistance offered to the electric current,—provided the electrodes admit of a circuit.

The importance of having a reliable milliampère-meter (if any is employed) must be apparent to the merest tyro in electricity. Unfortunately for science, the market is flooded to-day with cheap instruments that are absolutely worthless for scientific purposes. Many of them are never tested individually before being sold, even if made upon scientific principles. Others fail to record more than thirty or forty milliampères of current, and on that account are not adapted for use when very high currents are employed, even if deemed reliable as far as they go. Considerable ingenuity has been shown of late in the attempts of professional electricians to perfect this instrument, the utility of which is becoming more generally recognized each day, and which will soon constitute a very important part of each electrician's outfit. Perhaps one of the best instruments yet introduced to the profession is known as the "dead-beat" milliampère-meter, and is manufactured by Messrs. Waite and Bartlett, of New York.

This instrument (Fig. 153) is so called because the oscillations of its needle (which in most galvanometers amount to from fifteen to sixty before the needle comes to rest) are dampened by the suspension of the needle in a cylindrical block of copper by means of a fibre. This block becomes electrified by an induction and creates an opposing current, which reduces the oscillations to three or four after the current has been shut off or reversed. By means of two shunt-coils this instrument can be made to accurately record as high as five hundred milliampères, or as low as one-tenth of one milliampère. The mirror allows the operator to read the needle-deflections with as much ease as he would those of a vertical galvanometer. Rockwell, in a late publication, joins with me in the view that this instrument is a great improvement upon any other hitherto at our command for the measuring of current-strength.

The "absolute galvanometer" of Hirschmann is another desirable instrument, but it is too expensive for general use in the profession.

The needle, however, rests upon two points which are liable to get bent or to oxidize.

Respecting the employment of the galvanometer in the measurement of galvanic dosage, M. Vigouroux has lately contributed a paper to the Biological Society of Paris, in which he raises a point not generally considered, viz., whether the electro-motive force which propels the current through the external resistance afforded by the galvanometer, the rheophores, and the tissues of the patient should not be taken into account along with the reading of the galvanometer. He cites, as an example, that to give two patients a current of five milliampères, thirty-six Daniell elements were required in one and only twelve in the other. Both received the same quantity; but one received it under three times the pressure of the other. In any given case, as he states, the galvanometer fails to show this point. He believes that currents are modified in their physiological attributes by the electro-motive force as well as by the quantity and density. He therefore suggests that the quantity as shown by the galvanometer and the electro-motive force, which is easily ascertainable, after noting the number and the character of the cells employed, be alike recorded in each observation. By multiplying the electro-motive force by the quantity, we obtain the energy of the current in "volt-ampères" or "watts."

Respecting this point, this observer suggests that with an equal number of "watts" a current of great intensity and feeble electro-motive force would have a totally different therapeutic action from a current inversely constituted.

He employs the Deprez-d'Arsonval galvanometer with a vertical needle. This instrument is perfectly astatic. He also employs a special commutator, which is so arranged as to place at will the galvanometer in the circuit of the electrode or in derivation. It is graduated for fifty volts.

The only criticism that I would make to the views of this observer is that possibly the resistance in the rheophores, the electrodes, etc., have not been uniform in all his experiments. Much of the electro-motive force may have been expended in trying to overcome an imperfect conduction outside of the tissues of the patient. In that case the patient would not receive the current under as high a pressure as the electro-motive force of the battery would indicate.

In the second place, mere measurement of current-strength by the milliampère-meter is not sufficient for exact and scientific electrical treatment of morbid physical conditions.

If an experiment be made with a fixed number of cells of the same pattern and freshly filled, by passing the current through animal tissues with electrodes of different sizes placed upon identical spots of the same

individual, it will be found that the milliampère-meter will record a larger number of milliampères when the electrodes are large than when small, and that the smaller the electrodes the greater will be the discomfort to the patient, in spite of the fact that the amount of current is less than when the electrodes are increased in size.

Now, the *density* of the current is a very important factor in electro-therapeutics, especially so after the current has reached the diseased portion of the body which we wish to influence by it. All electrical currents tend to diffuse themselves to a greater or less extent after they enter animal tissues: hence, if the diseased part is deeply situated there is of necessity more diffusion and less relative density than if the part be superficial, and therefore nearer to the electrode. Zenner puts this point in a late lecture very clearly, when he says, "The density is in inverse proportion to the size of the conductor through which it flows: therefore, with the same current-strength it is greater when we apply small, less when we apply large, electrodes. When the area of disease is near the surface we often apply small electrodes in order that as dense a current as possible should enter it; but if we wish to affect a deep-seated part, the spinal cord for example, as the current becomes very much diffused before reaching it, it is necessary that a large quantity of electricity should enter the body, and for this reason we apply large electrodes."

We have already noted the important fact that the density of the current affects the current-strength. A patient will feel a very dense current of five milliampères (administered of necessity through a somewhat small electrode) more perhaps than one of twenty milliampères entering the same region of the body through a large electrode.

In the third place, some basis of measurement of electrodes must be generally accepted by the profession before electrical dosage can be considered as placed upon a positively scientific basis.

Erb has suggested that an electrode of 10 sq. cm. be regarded as the normal size. Remak has proposed that a series of graduated electrodes of 10, 15, 20, etc., sq. cm. be employed, and the size recorded as each application is made. It has been suggested also to express the relation between the current-strength and the electrode by making the milliampères the numerator and the size of the electrode in square centimetres the denominator of a fraction.

By such a system of record the results of treatment in the hands of different observers could be critically analyzed. Some satisfactory deductions relative to electro-therapeutics might possibly be then established beyond contradiction, provided that due care be taken relative to the saturation of the electrodes, the pressure employed upon them, the conduction of the rheophores, the skin of the patient, etc.

In the fourth place, it is very important that the placing of electrodes upon the cutaneous or mucous surfaces should be based upon scientific principles.

Erb has shown, in some diagrammatic cuts incorporated in his book, the different areas of diffusion that ensue whenever the electrodes are placed near together or wide apart. When near together the greatest density lies almost in a direct line between the electrodes, especially when applied to a similar surface of the body. When we wish to obtain the greatest possible density in deeply situated parts, or when the special effects of either pole of a galvanic battery are to be attained, the electrodes are to be widely separated.

The sternum is probably the best point upon the cutaneous surface of the body, in the vast majority of cases, for the application of the so-called "neutral electrode,"—*i. e.*, the pole whose effects upon the diseased part are the least to be desired. Another point commonly used for this purpose is the nape of the neck. This point is most easily reached on account of the clothing, but it is too abundantly supplied with muscles to be as desirable as the sternum.

A wide separation of the electrodes during a galvanic application is a very important point to insure in case the effects of either pole upon some special part are particularly to be desired. Thus, for example, when the electrical formulæ of individual muscles or special nerve-trunks are being tested to determine if the "reaction of degeneration" exists or is absent, the experiment should never be made when the neutral pole is sufficiently near to the active pole whose effects are being studied to influence the reactions obtained. Again, in the treatment of disease we sometimes wish to influence the diseased part exclusively by means of the anode or cathode. In such a case, the poles of the battery should be as widely separated as possible.

In the fifth place, the conductivity of the skin to electrical currents is modified by several factors which must be carefully considered in every case.

Among these the *saturation of the electrodes, the employment of salt in the saturating solution, and the amount of pressure exerted upon the electrodes* when applied to the skin are worthy of special mention. Patients afflicted with dropsical conditions offer a less resistance than those in whom the tissues are normal.

These minor details are not to be ignored by those who endeavor to do scientific electrical work in the treatment of disease by galvanism.

If you wish to test the value of these suggestions, put a patient and also a reliable milliampère-meter into the circuit of a galvanic battery. First, use dry or metallic electrodes and note the current-strength of a definite number of cells when they are applied lightly and also firmly to

the skin. Then cover the electrodes with absorbent cotton and wet them thoroughly in plain water, noting, after so doing, the results shown by the galvanometer of light and firm pressure. Finally, add a teaspoonful of table-salt to the water and again thoroughly wet the electrodes and the skin of the patient with this saline solution, noting for the third time the current-strength obtained by a light and firm application of the electrodes. In each experiment be sure that the battery has the same number of cells in action and that polarization has not been allowed to occur. This can be insured in most batteries by raising the elements from the fluid while the cells are not in use.

The effect of firm pressure upon the electrodes and the use of salt is to lessen the resistance; hence, the current-strength is often heightened by so doing. It has been wisely suggested that the handle of electrodes be furnished with a spring gauge which will enable the observer to know positively that the pressure exerted upon the electrodes in any given case is uniform at each sitting.

In the sixth place, the employment of a good rheostat in galvanic applications is very advantageous and oftentimes almost indispensable.

The conductivity of the skin varies in the same individual with the condition of the surface. When wet, as for example with perspiration, or somewhat dampened by a humid atmosphere, it is better than when dry or parched. This tends to explain in many cases why patients feel a galvanic application of a definite number of cells more at some times than at others. The milliampère-meter will, when employed, always show the reason of this.

Again, it is often necessary for scientific record to determine the exact resistance which any part offers to the passage of a galvanic current. This can be accurately measured by a coil rheostat. For example, the tissues of a patient may cause a deflection of the needle of a galvanometer, placed in the same circuit as the patient, of twenty milliampères with thirty freshly filled Grenet's cells. Now drop the patient from the circuit and place a coil rheostat in his stead, adding sufficient resistance by means of shunts in the rheostat to bring the needle-deflection to exactly twenty milliampères. The resistance indicated in the rheostat marks the resistance of the tissues of the patient, the rheophores, and the electrodes, which were traversed by the galvanic circuit when the needle-deflection was first noted.

In the cabinet battery which I have devised for the use of physicians (Fig. 162) I have lately incorporated a reliable coil rheostat, which may be connected or disconnected at the will of the operator by means of a switch. This I regard as a most valuable improvement upon the original model.

The rheostat enables us, furthermore, to gradually increase or decrease the current-strength without a danger of breaking the current

(an accident not without danger when very high currents are being employed). We do not have to touch the battery when this instrument is employed, but simply turn on its full capacity and graduate its strength by the rheostat alone. Many modifications of the fluid rheostat have been made with the view of removing the many objectionable features of this instrument. I have for some years personally discarded fluid rheostats in my practice because of the repeated annoyances they have caused and the uncertainties which attend their use.

I must confess that on reading the description of various instruments I fail to see any practical advantage to be gained by their use over a well-constructed coil rheostat. Yet, on the other hand, there is without doubt a field for a perfect fluid rheostat. They are cheaper to construct; they are somewhat easier for a novice to manage; they can be transported with little additional weight or inconvenience; and they materially aid an operator in graduating the current without danger of suddenly breaking it while the application is being made.

A physician in general practice wants an apparatus that is light, not too bulky to transport easily, and one that is reliable under all conditions. This is the great desideratum, and, unfortunately, less easily furnished than desired. A reliable milliampère-meter, a serviceable fluid rheostat, and a good galvanic battery are perhaps the three most important parts of an electrician's outfit for general use at the homes of his patients.

The general practitioner is apt to become easily confused by a superabundance of switches, plugs, and other electrical devices. He wants his electrical outfit as simple and inexpensive as it can be made without sacrificing delicacy, reliability, and durability in any part of his apparatus. If he strives for scientific attainments he will sooner or later be able to judge himself of the defects of his outfit, and supply the wants with greater discretion than when he originally purchased it.

At the present time, gynecologists are testing very extensively the method first advocated by Apostoli for the treatment of uterine fibroids in which currents of from one hundred milliampères upward are being frequently employed.

The question of galvanic dosage has been brought prominently forward in a controversial conflict between some of the advocates of this method and electricians during the past year; some of its adherents being accused of serious errors in the computation of the actual currents which have been employed in this therapeutic procedure.

The whole controversy, to my mind, seems to turn upon two factors which do not appear to be fully decided. These are: (1) the internal resistance of the batteries employed to generate the current; and (2) the external resistance which the rheophores, the galvanometer, and the animal tissues traversed by the circuit together help to constitute.

Now, the employment of a coil rheostat will enable any one to determine the exact resistance offered by the tissues in each individual case with little loss of time or labor.

The external resistance to the passage of a galvanic circuit is a very important factor in modifying the current-strength which the milliampère-meter indicates. To repeat what has already been said, there are only three factors in Ohm's law, any one of which can be easily figured when the other two are known. Ohm's law is as follows: The *electro-motive force divided by the resistance equals the current-strength.* The electro-motive force (E) is estimated in *volts;* the resistance (R) in *ohms;* and the current (C) in *ampères.* To put this in a mathematical form, the following equations are applicable to the solution of any such problem:—

$$C = \frac{E}{R} \text{ or } E = C \times R \text{ or } R = \frac{E}{C}$$

Finally, in the seventh place, the length of the sitting is a factor in electrical dosage.

This factor must, unfortunately, remain—for some time at least—a matter of pure empiricism. As a general rule, the weaker the current the longer may its application be prolonged. Still there are, without question, certain individuals who are more tolerant of electrical currents than others; and the physical conditions of each patient have to be taken into consideration before the duration of a *séance* can be decided upon. Of necessity, the experience and judgment of the operator will always prove of material service in deciding such questions as they arise; and it is here that the success of some and the lack of success of others may possibly lie.

The tendency of the age is toward the use of much stronger currents than were formerly considered judicious by the German investigators. Especially is this true in the treatment of some of the graver diseases, and in many conditions where electrolysis and the galvano-cautery are now successfully employed.

In concluding my remarks concerning galvanic dosage, I trust the many aspects of the question may now be more clearly understood by some of my readers; and that the necessity of a more complete electrical outfit than many general practitioners now possess may be apparent to them. These remarks apply only to galvanic currents, and not to faradaic or static applications.

We have now discussed the essential points pertaining to electro-physics and electro-diagnosis, and there remains now for us to consider the uses of electricity in the treatment of diseased conditions of various organs and tissues.

Before we pass to details of the practical part it may be well for us

to review in a general way some of the laws which should govern us in applying electric currents to the different tissues, and the objects to be attained by the employment of faradaism, galvanism, and static electricity.

GENERAL ELECTRO-THERAPEUTICS.

The rapidity and completeness of reported cures of nervous affections by the use of electric currents upon living tissues during the last quarter of a century leave no room for doubt that this agent is particularly valuable in the treatment of paralysis, neuralgia, spasmodic diseases, disturbances in the sensibility of the skin, and many disordered states of the brain, spinal cord, and peripheral nerves themselves. We have undisputed facts which prove also that blood may be coagulated with safety within some aneurismal sacs by the galvanic current, that the life of the ovum may be destroyed in extra-uterine pregnancy, that animal tissues may be disintegrated by chemical changes induced within them by this agent, and that neoplasms may be removed without hemorrhage by the cautery loop.

Our present ignorance of the molecular and nutritive changes in tissues (as the cause or result of disease) renders it impossible to do more than speculate upon the theory of the therapeutical action of electricity in many cases; but, on the other hand, our empirical knowledge of these effects is none the less valuable because we are unable to explain them. The same criticisms would otherwise hold good in reference to almost all of the drugs employed in medicine. None of us know exactly *how* they produce their specific effects.

Concerning speculation upon electrical effects on living tissues, Erb remarks as follows: "What appears more natural than that neuralgia and spasms could be relieved by the sedative action of the anode, with production of anelectrotonus, and that anæsthesia and paralysis could be cured by the exciting action of the cathode, with production of catelectrotonus? But, apart from the fact that we are not certain that an increase of irritability really occurs in one group of cases and a diminution in the other, it must be remembered that electrotonic action disappears very rapidly after the cessation of the current, while the curative effects of the current are more or less permanent."

Now, we may summarize the general principles which regulate the use of electric currents as follows:—

(1) They may exert, under certain circumstances, a *stimulating* or *irritating effect*. This is, perhaps, the basis of the most varied applications of electricity to disease.

(2) They may exert, when properly applied, a *sedative action* on nerves or nerve-centres.

(3) They may be made to exert a *catalytic action* upon neoplasms, enlarged glands, etc.

(4) They are capable of causing *electrolysis.* This action is one which has lately come into prominence.

(5) They create *heat* under certain conditions. The galvano-cautery is to-day assuming a very prominent place in some of the departments of surgery.

Let us now discuss each of these special actions separately, noting the general points of interest pertaining to each which will aid us in properly treating our patients. Electrolysis and the galvano-cautery have been treated of in previous pages.

Stimulating or Irritating Effect of Electricity.—This is indicated in many diseased conditions encountered by the neurologists as well as by the general practitioner. Among these the following may be prominently mentioned :—

Some of the various forms of cerebral and spinal diseases.

Depressed irritability of some special nerve-trunks.

Abnormal resistance to conduction of electric currents, exhibited by the motor or sensory nerve-filaments of some part.

As a counter-irritant to some pathological conditions.

Trophic disturbances of special regions (skin, nails, hair, etc.).

Vaso-motor depression.

Atrophic changes in muscles.

As a means of indirectly affecting the nerve-centres through the sensory nerves, thus influencing respiration, circulation, phonation, vaso-motor paths, peripheral organs, the muscles, etc.

The methods of application which are best adapted to accomplish irritating or stimulating effects are differently stated by authors. Personally, I do not confine myself exclusively to faradaism or galvanism.

The *faradaic current* is more commonly employed for this purpose than the galvanic. The electrodes should be selected, as to their size and shape, in accordance with the parts to be acted upon ; they should be well moistened with salt water, and kept closely in contact with the skin. The wire brush is the best electrode to stimulate the nerves or other tissues of the skin. It should be used dry. I prefer the secondary faradaic current to that of the primary coil for stimulating effects.

If galvanism is employed as a stimulant, *Remak's plan*, of moving the well-moistened cathode rapidly over the nerve-trunk or muscle to be stimulated, with a current sufficiently strong to cause strong wave-like contractions, is a good one. Another method, termed by this author "*terminal labile stimulation*," consists in stroking the tendinous end of a muscle with the cathode so as to affect the entire length of the muscle. In both of these methods, the anode is kept stationary upon

some indifferent or neutral point—the centre of the sternum by preference, or the nape of the neck.

One of the most vigorous methods of stimulation consists in *rapidly changing the polarity* by means of a commutator, when the galvanic battery is employed.

The Combined Current.—Another method which I employ (not generally mentioned in text-books) consists in connecting a galvanic battery, by means of a rheophore, with a faradaic instrument, thus bringing *both a constant and induced current* to bear upon the tissues at once. The rheophore which connects the batteries joins the positive binding-post of the galvanic instrument with the secondary coil of the faradaic; the two rheophores connected with the electrodes run from the negative binding-post of the galvanic and from the secondary coil of the faradaic instrument. The two instruments (faradaic and galvanic) are thrown into action simultaneously, and the strength of the current employed is graduated by the number of cells used in the galvanic battery and by the extent of the overlap of the secondary coil of the faradaic instrument. I have obtained some remarkable results by the stimulation thus produced in various forms of trophic disturbances of the skin and muscles.

The stimulation of nerve-fibres (when obstacles exist to their conduction) should be performed peripherally from the site of the lesion in sensory nerves, and as centrally as possible in motor nerves (Erb). Degenerated and atrophied nerves and muscles require a direct effect of the currents employed. For these reasons, the site of stimulating electrical applications depends upon the situation and character of the lesion and the object to be attained.

Modifying Effects of Electric Currents.—The *irritability of nerves and muscles* may be influenced by electric currents.

In certain diseased conditions, we may expect a favorable result from such an action. Thus, for example, in some types of paralysis, in anæsthesia, in certain vaso-motor disturbances, and in depressed states of cerebral and spinal activity, the irritability of nerves or of muscular fibres is diminished; hence we resort to the so-called "catelectrotonic action" of electricity as a means of stimulating and restoring the normal irritability of the tissues affected.

It is now generally accepted as proved that *feeble faradaic currents* will accomplish this end. Galvanic currents, when applied for this purpose, give more positive results, however, than faradaic.

In order to *increase irritability* by galvanism, the negative electrode should be applied in a stabile manner (*i.e.*, without being moved) to the part upon which this effect is to be produced; and the strength and duration of the current should be steadily increased. When the muscles

or motor nerves have been exhausted by over-exertion, excessive fatigue, etc., this action (termed by Heidenhain the "refreshing action" of galvanism) is particularly indicated.

Those conditions in which the *normal irritability of nerves or muscles is intensified* demand the so-called "anelectrotonic action" of electricity. These conditions comprise all irritative states of the sensory, motor, and vaso-motor tracts within or without the brain and spinal cord; hence, we employ this action in neuralgias, spasmodic affections, hyperæsthesia of any of the cerebro-spinal nerves, headache, excitation of any of the special senses, cerebral and spinal irritation, etc.

In order to *decrease the irritability of nerves or muscles*, we may employ very powerful faradaic currents. We may also begin by employing a feeble faradaic current and gradually increasing its strength to the highest point of endurance; then maintaining it at this point for some time; and subsequently reducing it gradually to the feeblest current perceptible to the patient. This method is known as the "*increasing induction method.*" Electrodes, well moistened and of large size, should be employed and kept immovable upon the same points during the application. It is often advisable to repeat this procedure several times at one sitting (Erb).

When the galvanic current is employed for the purpose of decreasing irritability, the positive pole is made fast at the point to be influenced. The current is increased in strength and maintained at its maximum for some time, after which it should be decreased gradually until it cannot be perceived by the patient. The gradual decrease of the current-strength prevents the marked temporary increase of irritability which is liable to follow this method when this step is omitted.

Static electricity exerts in many cases an immediate beneficial effect upon neuralgic pains (especially upon sciatica) and upon the various spasmodic affections, as, for example, chorea, paralysis agitans, tremor, contracture, etc. These effects are obtained, in some cases, when galvanism and faradaism have proved of no benefit. I should never regard a case as incapable of benefit by electric treatment until static electricity, in the form of insulation, the electric wind, or the spark, had been thoroughly tested. I have had better results with this form of current in tremor than with galvanism or faradaism.

Some forms of pain (as, for example, the pains of ataxia, sciatica, trigeminal neuralgia, muscular rheumatism, etc.) are oftentimes relieved by a few applications of static electricity. My experience with this agent has convinced me that its effects are often satisfactory in cases where pain is a prominent symptom, when galvanism has been tried without benefit. I have found that insulation and the abstraction of heavy sparks from the seat of pain give the best results.

This therapeutical agent will be discussed separately, later in this volume.

Catalytic Action of Electrical Currents.—Under this heading we include (1) an increase of absorption produced by dilatation of the capillary blood-vessels and lymphatics; (2) an increased capability of tissues for imbibition of fluids, through an increase of osmotic processes; (3) changes in the disassimilation and nutrition of nerves, on account of their stimulation or refreshing effects; (4) changes in the molecular arrangement of tissues, caused by electrolytic processes; and (5) the results of the transportation of fluids from one pole to the other (Remak and Erb).

Remak has shown that muscles become congested and greatly swollen when subjected to galvanism. They are rendered tense, and (according to this observer) absorb water more freely than muscle which has not been galvanized.

Changes of a marked character may be induced in the skin by galvanism. These have been studied by Erb, Remak, Bollinger, and others.

The vaso-motor nerves may be influenced by electrical currents. This is shown by many of the later investigations,—prominently those of Lowenfeld, which apparently demonstrate that contraction and dilatation of the vessels of the brain result, respectively, from antero-posterior and transverse currents through the head from a galvanic battery.

Although we are, as yet, unable to speak with positiveness regarding the certainty of the catalytic effects of electrical currents, or to map out the forms of disease which are to be regarded as specially indicating these catalytic effects, still it may be said that the following states have been successfully treated by electrical currents, and that the cures are probably to be attributed to a catalytic action. (1) inflammatory affections of the nervous system, including sclerosis, myelitis, neuritis, etc.; (2) arthritis and chronic exudations into joints; (3) glandular enlargements; (4) hard cicatrices, periosteal swellings, and fibrous adhesions; (5) contusions, sprains, extravasations of blood, and other results of traumatisms.

The *galvanic current* is the one that is generally employed when catalytic effects are desired. In diseased conditions of the brain, spinal cord, or any of the deeply seated organs, the faradaic currents are not usually productive of benefit.

The "stabile method" of application of the galvanic current is preferable, to my mind, when catalytic action is to be attained. The strength of the current should be sufficient to easily overcome the resistance offered, and the duration should be sufficiently prolonged to accomplish changes in the tissues subjected to its influence. One pole

is placed, as a rule, at an indifferent point (the sternum by preference), and the other over the tissue diseased. Sometimes, as in the case of the brain, for example, the poles are placed upon either side of the diseased part. Although there are exceptions to the rule, it is well to use the anode or positive pole over the diseased part when pain is present, when symptoms of active irritation exist, or when the morbid processes are very active. The cathode or negative pole is best adapted to influence chronic morbid processes, such as sclerosis, indurations, etc. Erb recommends that the polarity of the current be changed several times in either case; he doubts the infallibility of the rule given, although it is theoretically sound. Chvostek urges the use of short and moderate currents for a few minutes (three to ten) when catalytic action is desired. In this way, he believes, the vaso-motor and trophic nerves are more impressed than by any other method.

Respecting the catalytic action of *faradaic currents*, a difference of opinion exists between authors of note. One thing is certain, viz., that strong currents are required, and that the currents must be passed directly through the diseased part to accomplish marked results. Glandular tumors have been resolved by this method with great rapidity in some recorded instances.

Galvanization of the Cervical Sympathetic.—This method has afforded relief, according to published cases, in vaso-motor and trophic disturbances of the nerve-centres, the eye, viscera, muscles, joints, and skin. Thus, for example, cases of cure of epilepsy, atrophy of the optic nerve, Basedow's disease, progressive muscular atrophy, lead-palsy, scleroderma, chronic rheumatic arthritis, bulbar paralysis, neuralgias of various types, and many other conditions have been reported by means of this method. Respecting this step, Erb wisely remarks as follows: "There can probably be no doubt of the correctness of a part of these observations, but this does not by any means imply that the cervical sympathetic is responsible for such results."

When we review the structures which compose the neck and recall the numerous connections which exist between the sympathetic cords, the pneumogastric nerve, the brain and cervical segments of the cord, the medulla oblongata, the brachial and cervical plexuses of nerves, etc., it becomes clear why De Watteville applies the term "sub-aural galvanization" and Erb the term "galvanization of the neck" to this special procedure.

The steps required to influence these parts by Meyer's method consist in the application of a small electrode (cathode) under the angle of the jaw and adjacent to the hyoid bone, and then crowding it backward and upward against the vertebral column, the positive electrode (of larger size) being placed over the seventh cervical spine. The current

may be stabile, labile, or interrupted; or the polarity may be changed from time to time during the sitting of from one to three minutes. Six to ten galvanic cells of the Grenet variety are sufficient. The application may be unilateral or bilateral, according to the demands of the case.

Corning has devised an instrument which insures carotid compression with galvanization of the neck for the treatment of cerebral hyperæmia and some other morbid conditions.

Benedict places the positive pole in the jugular fossa, and the negative pole upon the superior cervical ganglion.

General Faradization.—This method of administering electricity was first employed by Beard and Rockwell. By this procedure the entire body is subjected to secondary faradaic currents of varying intensity. It is applicable chiefly to those forms of nervous disturbance which are associated with general debility, poverty of the blood, special diatheses and cachexiæ, hysterical affections, skin diseases, persistent chronic inflammations, and other results of low vitality or functional derangements of the organs.

To apply this method, the patient must be undressed or very loosely clothed. The feet are immersed in a bowl of tepid water with a little salt added, in which the cathode is also placed after being connected by means of a rheophore to the binding-post of the secondary coil of a faradaic machine. The anode is held in one hand of the physician, and his other hand (well moistened in salt water) is applied to all parts of the surface of the patient's body. If the subject can bear it, a large electrode covered with absorbent cotton and flannel, or with a soft sponge, is employed in place of the hand. The application should begin at the head and terminate at the feet, the strength of the current being modified from time to time as the feelings of the patient may demand. The extremities and back should have vigorous stimulation, the nerves of the neck should be influenced by a much weaker current, and the cœliac plexus should be influenced by a stabile application of a few minutes over the epigastrium. The entire duration of the application occupies from ten to twenty-five minutes. It may be applied as often as three times a week if necessary.

Personally, I can attest the efficacy of this treatment as a general tonic. I have witnessed immediate effects from it in some of my cases, and I employ it constantly in a modified form.

In case the hand of the attendant is to be employed as an electrode, I would advise the use of an instrument which I have devised as an improvement over the way originally described by the inventors of this method. It is called the electric bracelet. It is placed upon the right wrist of the attendant over a pad of wet absorbent cotton, and the rheophore is screwed into the binding-post upon it. The right hand is then

wet in salt-water and used as previously described. By this instrument the hand of the attendant is alone subjected to the current, and the fingers can detect muscular contraction in the patient even when too feeble to be seen easily. As an adjunct to massage, I employ this useful instrument with decided benefit.

General Galvanization.—The steps required by this method are similar to those previously described, except that the constant-current battery is employed in place of a faradaic machine.

Central Galvanization.—The cathode is placed over the epigastrium. This electrode should be of large size. The anode is stroked over the forehead, with a current of about two milliampères, for two minutes; then made stabile over the cranium for about two minutes; then moved up and down the neck on each side for the same duration; finally it should be moved along the length of the spine for about five minutes. This method was a favorite one with the late Dr. Beard, who reported cures of gastralgia, hysteria, hypochondriasis, nervous dyspepsia, and many of the symptoms of cerebral and spinal neurasthenia by its continued use. In two cases of gastralgia in which I personally employed it for some time I obtained an absolute recovery.

The Electrical Bath.—This method of administration of electricity to a patient may be accomplished by using a metal tub, or one which is composed of a non-conductor. If a metal tub is employed, the patient must be protected from actual contact with it. This is usually accomplished by means of wooden slats or some other medium of support for the patient when immersed. If the bath-tub is of metal, one rheophore of the battery employed is attached to the tub, while the other is attached to an electrode held by the patient or placed in contact with his body. If the tub is of a non-conducting material, both electrodes may be placed in the water. The electrodes employed should be very large (often running the entire length of the tub), in order to allow of as great a diffusion of the electricity as possible. The fluid in the tub may be simple water, or, preferable, a solution of salt, soda, or an acid.

The battery employed for a bath should have very large plates, so as to generate an abundant *quantity* of electricity without a very high electro-motive force.

Personally, I am not a strong advocate of this method of treatment. It violates one of the fundamental principles of electrical treatment of localized affections in that it does not confine the polar action to the part or parts diseased. In the second place, I have not found its tonic action to equal that of general faradization or general galvanization.

Strong claims have been made in its favor as a remedy for tremor (especially of the alcoholic and mercurial varieties) and for chronic

articular rheumatism, but I am not yet convinced that they are to be regarded as well established.

If the reader desires to try this method of treatment in any case, it is well to know that the temperature of the bath, as well as the strength of the current employed, should be modified by the condition of the patient. The duration of the bath should never exceed thirty minutes, and ten minutes will generally suffice. The current should be strong enough to be perceived by the patient in all cases. The elements of the cells employed to generate the current should be large, in order to insure *quantity* as well as electro-motive force.

The Relief of Painful Points.—One of the most generally useful effects of electricity is the relief which it affords in many cases to pain. Of all the methods of treatment of neuralgia now employed, I consider electricity, in some of its various forms of application, by far the most efficacious. Personally, I have almost discarded internal medication for the relief of this class of sufferers.

In the majority of subjects afflicted with neuralgia, painful points may be detected along the course of the affected nerve or its branches. These are situated, as a rule, where the nerve gives off a branch or bifurcates, and also where it passes through a foramen. Sometimes it is necessary to make pressure along the course of the nerve to detect the existence and seat of these points.

Now, it should be remembered that the successful electrical treatment of neuralgia depends largely in some cases upon the *direct treatment of these painful points.* They seem in some way to have a relationship with both the production and relief of some types of neuralgia, as well as spasm of the muscles, ataxic symptoms, and other forms of nervous diseases. These points may be the seat of a localized periostitis, a circumscribed inflammatory exudation, a neuritis, an enlarged gland, and many other conditions which create nervous phenomena. In a few instances the symptoms even of ataxia have been relieved, by the electrical treatment of painful points in the region of the spinous and transverse processes of the vertebræ, by men of note, among whom may be mentioned Brenner. Remak, Meyer, Legros, and others.

The steps which you should employ in the treatment of painful points are as follow: 1. Use the galvanic current, employing from three to eight Grenet cells. 2. Apply the anode to the painful spot, and keep it stationary at that point. 3. Place the cathode at some indifferent point, preferably the sternum. 4. Do not use a current which will be excessively painful to the patient, nor exceed five minutes in the application. I frequently do not allow the duration of the current to exceed two minutes at a sitting. It is advisable, in persistent cases, to make the applications daily.

Of late some experiments have been made, with apparent benefit, by having patients of this class wear over the painful points *a piece of metal*, connected with another piece of metal (which is also in contact with the skin) by an insulated wire. The best metals are zinc and copper. They should be brightly polished before the application, and should have a piece of dampened linen between them and the skin. They may be worn continuously for weeks, or changed each day on retiring and rising.

Some authors recommend the employment of *very feeble galvanic currents for an hour or two at each sitting*, the anode being placed over the painful point. Le Fort goes so far as to suggest the propriety of applying such currents continuously for weeks, by means of ordinary rheophores and electrodes, when fatty changes, contractures, or reflex paralyses are to be combated.

Electrolysis.—When a galvanic current is concentrated within animal tissue by a close approximation of the electrodes, or when, by means of the "polar method" and insulated needles, a galvanic current of high intensity is made to traverse some selected spot upon the human body, there is apt to be a chemical decomposition of the water and salts, and a coagulation of the albuminous elements of the tissue thus acted upon. The salts are then separated into their bases and acids, while the water is simultaneously decomposed into hydrogen and oxygen. The positive pole attracts to it the acids and the oxygen; the negative pole attracts to it the alkalies and the hydrogen. For this reason the insulated needles tend to become oxidized when they are connected with the positive rheophore. They do not become so when attached to the negative rheophore. The free alkalies deposited at the negative electrode are apt, on the other hand, to cause destructive effects upon adjacent tissues. These are greatly in excess of that produced by the oxidation of the metal points of the insulated needles when joined to the positive rheophore.

When we wish to test the strength of the current which we propose to employ for electrolysis, it may be easily done by sending the current through the white of an egg for twenty or thirty minutes. In that time it should coagulate the albumen.

In order to reach the parts upon which we most desire to perform electrolysis, it is often necessary to perforate the skin and the muscles. To do this, needles are employed. They should be insulated with hard rubber, collodion, or shellac, except at their point for one-half inch, and the uncovered part should be gilded, as a rule, in order to prevent its oxidation. They should be from two to five inches long; should be strong enough to penetrate tissues without a liability of breaking; should be as small as is consistent with the current-strength to be employed; and should be so arranged as to enable the operator to adjust them in a

handle to which one of the rheophores of the battery may be attached. Ordinary sewing-needles strung on a wire may be employed in treating superficial nævi, tumors, etc., if you lack the instruments specially designed for the purpose. You may shellac them for insulation if deemed best.

It is very important, in some cases, that the insulation of the needles employed be as perfect as possible; and that the tips of the needles be triangular or lancet-shaped, in order that they may penetrate the skin with ease. The needles and handle required can be bought of any manufacturer of electrical appliances.

The battery employed for electrolysis need not be unlike that for ordinary medical purposes. Twenty-four of Grenet's cells will produce a sufficient intensity of current, provided they are freshly filled. Robin's statement that a current of forty-five milliampères is requisite must be based upon a very limited external resistance. It is well to use a battery of greater power than is actually required, so that fresh cells can be added without breaking the current during the operation, when deemed

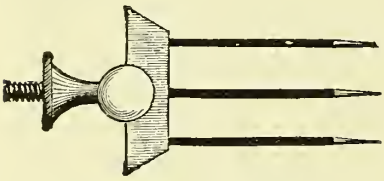


Fig. 186.—Electrode for Electrolysis (with three insulated needles).

a

Fig. 187.—Electrode for Electrolysis.—The needles are fine and numerous, and are arranged as shown at *a*. This is an excellent device for affecting the cure of diffused nævi, small glandular tumors, etc.

necessary. Personally, I use insulated copper wire for rheophores when performing electrolysis, in preference to the tinsel cords commonly employed for electric applications. They are less flexible than the tinsel cords, but they are vastly superior to them as conductors.

Electrolysis has been employed for the following purposes, with more or less success:—

1. The coagulation of blood in aneurismal sacs.
2. The relief of nævi and erectile tumors.
3. The cure of cystic tumors.
4. The cure of goitre.
5. The cure of ecchinococci.
6. The cure of ovarian cysts, and those of the broad ligament.
7. The cure of urethral stricture.
8. The cure of malignant and fibroid growths.
9. The cure of hydrocele.
10. The destruction of the fœtus in extra-uterine pregnancy.
11. The removal of cicatrices, polypi, and other new formations.
12. The destruction of superfluous hairs and their follicles.

Since many of these conditions come under the observation of the neurologist, either as a factor in the causation of nervous symptoms or as co-existing troubles which demand relief, it may not be inappropriate to hastily dwell upon the electrical treatment of such conditions in a volume of this character.

Respecting the *treatment of large aneurismal sacs* by this method, statistics show that the operation merits more general recognition, as a means of possible cure of intra-thoracic and abdominal aneurisms which cannot be safely treated by ligation, than it has received. Nine cures out of thirty-seven cases have been collected by Duncan from various sources; and Bartholow has since collected others which have been benefited by it, although not positively cured. In none of these cases, so far as I can ascertain, was the current-strength measured by a galvanometer. There is reason to hope that the operation may become more generally employed when the steps of the procedure necessary to its success are determined with greater exactness. There seems to be a doubt, as yet, regarding the best method both of generating and directing the current, so as to prevent suppuration and secondary hemorrhage. In two cases of blood-sacs upon the face, intrusted to my care, I have succeeded in consolidating the tumor and effecting its radical cure by this means without suppuration or other complications. I employed the positive rheophore for the needles and placed the cathode at the nape of the neck in both cases. The duration of the two sittings in each case was about ten minutes, and twenty-four Grenet cells were employed. I believe that the anode produces the firmest clot; hence the danger of hemorrhage on the withdrawal of the needles is less than when the cathode is employed. The risk of embolism, as a result of disintegration of the clot, does not seem to be so great as one might at first imagine.

The employment of electrolysis in cystic tumors of the neck, the ovaries, and the thyroid gland, has been resorted to by many experimenters of note. Among these may be mentioned Althaus, Amussat, Ultzmann, Clemens, Semeleder, and others. Some of the cases reported seem to point toward this method of treatment of these diseased conditions as potent and comparatively devoid of danger if properly carried out.

The *treatment of urethral stricture* by electrolysis I have always heretofore combated, chiefly because I think it less safe and far less certain than gradual dilatation. I must confess, however, my prejudices may be more or less without foundation. My perusal of the reported cures by this method has not, as yet, carried to my mind a thorough conviction of the permanency of the cure. The method still seems to me to lack absolute precision, which should, to my mind, form the basis of all surgical procedures within that canal. I am having made some

modifications of urethral electrodes which I believe will insure greater precision than any yet devised.

Ordinary *cases of goitre*, and the *enlargement of the thyroid gland which accompanies Basedow's disease*, have been cured by electrolysis. Rockwell and Butler have reported some astonishing results in the treatment of exophthalmic goitre by galvanism of the thyroid gland. Rockwell places the cathode over that body and the anode over the solar plexus, combined with the employment of the anode in the auriculo-maxillary fossa and the cathode over the cilio-spinal centre (cervical segments of the spinal cord) at each sitting. His cases of reported cure required from fifty to sixty-nine sittings. Needles were occasionally employed upon the goitre. This treatment was supplemented by the use of iron, zinc, digitalis, and ergot; and a restricted diet, with instructions regarding the necessity of the repression of the emotions and passions, was enforced.

The *arrest of extra-uterine pregnancy* by electrolysis, and also by shocks transmitted through the sac from Leyden jars charged with static electricity, from a galvanic battery, and also from a faradaic machine, constitutes, perhaps, one of the most successful and remarkable contributions to medicine.

The *treatment of cancer* by electrolysis has been followed by satisfactory results in some cases, according to the observations of Beard, Butler, Mussey, and Neftel. The question of accurate diagnosis of the cases reported as cured must still be considered as unsettled. The results apparently obtained should certainly awaken the profession to a trial of this method of treatment of a malady which internal medication, caustics, and the knife seem powerless to combat.

Bartholow reports a cure of four out of six cases of *fibroid tumors of the breast* by electrolysis. The remaining two patients failed to continue treatment for a sufficient length of time. He did not employ needles in any of these cases.

The *relief of hydrocele* by the introduction of two needles connected with the rheophores of a galvanic battery and brought within a half-inch of each other at their points has been reported by Rodolfi, Frank, Bartholow, and others. Some of the cases reported as cured required only one application.

In dismissing this subject it may be well to summarize the effects of electrolysis, as follows :—

1. A feeble current tends to cause dilatation of the capillaries and the lymphatic vessels, and thus to aid in absorption.

2. A stronger current decomposes the salts and the water of tissues, and coagulates the albuminoid elements.

3. A disintegration of the tissues immediately adjacent to the pole

which produces the effects previously described takes place, with an escape of bubbles of gas, when the decomposition of tissues is active.

4. As an eschar may be formed by a current of great intensity, it is maintained by some authors that the cicatrix which results from such a slough is soft and pliable if the eschar has been made by the anode, and dense, with a tendency to contract, when due to cathodal action. I am unable to confirm or deny this statement.

5. The danger in electrolysis is that of "doing too much" rather than too little. The former error cannot be repaired; the latter can by repeated sittings.

6. When an escharotic effect is desired, it is well to have the needles made of zinc. The decomposition of the chloride salts forms indirectly the chloride of zinc, because the liberated chlorine attacks the needle. This is absorbed by the tissues adjacent to the needle, and an escharotic effect is thus produced. In the treatment of malignant growths such needles, with currents of weak intensity, and long sittings, seem particularly well adapted. This method is almost painless, and has produced excellent results in some cases reported.

7. The introduction of needles into the tissues is not an absolute necessity when treatment by electrolysis is indicated. The same effects to a lesser degree may be obtained by placing the electrodes in contact with cutaneous or mucous surfaces.

8. The employment of iron needles has been suggested for the rapid coagulation of blood, on account of the styptic effect of the chloride of iron which tends to form by the liberation of chlorine from the chlorides of sodium, potassium, and calcium.

The Galvano-Cautery.—When a large quantity of electricity is forced through the resistance offered to its passage by a platinum wire or a strip of platinum (usually bent into the form of a knife), the heat produced causes the platinum to rapidly approach redness or whiteness. Such an arrangement is known as a "cautery loop" or a "cautery knife." The battery which is employed to generate electricity in sufficient quantity to accomplish such a result is known as a "cautery battery." In cautery batteries the plates are large and near together; hence unusual precautions have to be taken to prevent "polarization," which takes place very rapidly on account of decomposition of the fluid in which the elements are immersed.

Of all the devices which have been suggested to overcome this difficulty, I prefer that of Dr. Piffard. In the battery devised by him the zinc plates are perforated, so that the fluid can be forced through them upon the platinum plates by means of a rocking motion when the battery is in action. The assistant who operates the battery can produce any degree of heat required by making the plates move slowly or rapidly

through the fluid. The key-board of the battery is so connected by means of large thumb-screws that the elements can be connected for either quantity or intensity, as the operator may desire. The rheophores are composed of large copper wire, heavily insulated with rubber.

I have made several improvements upon the original Piffard battery of late, which, in my opinion, will increase the ease of working the instrument. They are not, as yet, fully perfected.

It may be advisable to again impress upon you the fact that batteries designed for ordinary medical purposes are totally unfit for heating a cautery loop or producing an electric light. A battery designed for cautery purposes is also totally unfit for other purposes in medicine.

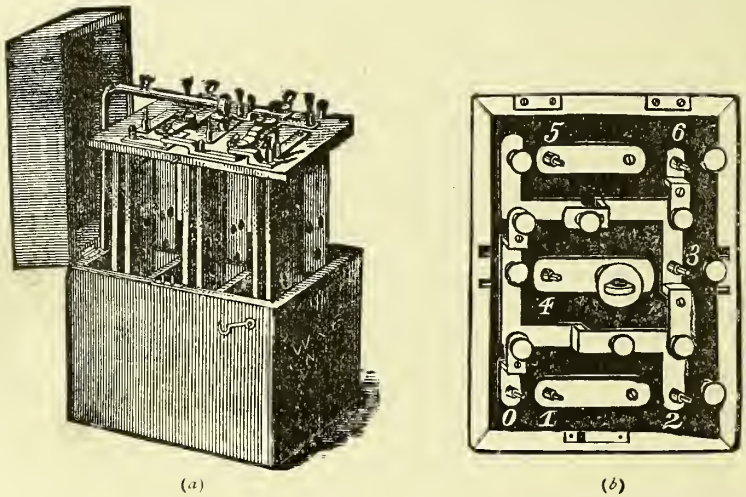


FIG. 188—PIFFARD'S CAUTERY BATTERY.—(*a*) The battery as suspended when not in action. (*b*) Arrangement of the top of this battery, showing the screws which regulate the connections between the different cells. The battery is rocked during its action to prevent extreme polarization. By making the movement slow or rapid, the heat of the loop or knife may be regulated at will.

In operations upon the tongue, nose, pharynx, uterus, vagina, rectum, and in some other regions, the galvano-cautery seems destined to supersede the scalpel and écraseur. No blood need be lost in amputations of considerable magnitude, provided the operation is skilfully performed. If the loop is employed, it is slipped when cold over the part to be removed. It can be adjusted, therefore, with every precaution against accident. After the current is turned on, the heat of the wire even can be regulated with great precision. Care should be exercised against drawing the wire too closely to the handle, and in selecting a wire which will not burn off or prove too large for the battery employed. As in all surgical procedures, this instrument should be handled by an expert. It is well for a novice to practice upon pieces of meat or bone until he

familiarizes himself with the details of its use, in case he meditates performing an operation upon a human subject. When operations are to be performed within the mucous cavities of the body, the patient has frequently to be trained to tolerate the necessary manipulation. A dull-red heat is preferable to a white heat in dividing vascular tissues, and it is very important that the division be slowly performed. When the skin is to be embraced within the loop, it is well to divide it first with a cautery knife, and subsequently to adjust the wire.

The cautery knife has been successfully used in removing cancerous growths within mucous cavities, in tubal pregnancy, in tracheotomy, in extirpation of the breast, and many other similar procedures.

An attachment to the cautery battery, known as the "dome cautery," consists of a coil of platinum wire over a cone of porcelain. These may be of any size, and the porcelain cone may be omitted if deemed necessary. It may be employed in destroying hypertrophied tonsils, hæmorrhoids, polypi, nævi, epithelioma, etc.

The great advantage which the galvano-cautery has over the use of the knife is the absence of hemorrhage and of great pain. The platinum knife can be made of any form desired. There is no limit to special forms of attachments which may be devised to simplify its use in different regions of the body.

In operating upon the tongue, needles may be passed through the organ in front of the site selected for the loop, so as to prevent slipping of the wire. Bryant, who has had an extensive experience in this operation, recommends a twisted wire rather than a large one. There is some reason to believe that the heat tends, moreover, to destroy (in the case of cancerous growths) the germs of the disease which might elude the knife.

GENERAL RULES GOVERNING ELECTRO-THERAPEUTICS.

Before we pass to the consideration of special methods of employing electricity in the treatment of disease, it seems to me advisable to suggest a few rules which may possibly aid you in deciding where and how to direct your treatment in any special case. There are, of course, some exceptions to each of these rules; but they are, nevertheless, sufficiently accurate to be used as guides in your practice:—

1. Soak your electrodes in a weak solution of table-salt, not in simple water. This diminishes the resistance afforded by the skin at least fifty per cent.

2. Always press your electrode firmly and evenly against the part which it touches. This renders the current employed an even one to the patient and assists in its conduction.

3. Put a milliampère-meter or a galvanometer, as well as the body of your patient, into circuit, and record all your observations, respecting

the current-strength employed, from its scale. It is neither scientific nor accurate to simply record the number of cells employed. Cells grow weak by long-continued use, by polarization, and other causes. In case a faradaic instrument is employed, a galvanometer is useless; hence you should record the number either of centimetres or inches of the primary or secondary coil employed.

4. Always endeavor to apply one of the poles to the part which is diseased. The plates of Ziemssen which I incorporate in this volume indicate the situation of the "motor points" of the head, trunk, and extremities. Such plates will enable you to direct your treatment to any special nerve or muscle.

5. Acquire, by frequent experimentation upon yourself, a knowledge of the effects of different current-strengths, the situation of most of the more important nerve-trunks, the formulæ of contraction of healthy nerve and muscle, and all other information necessary to the use of electricity in medicine.

6. Never use too strong a current upon a patient at the first sitting. It may frighten him, and he may never return. It is always best to begin with weak currents; in the majority of cases weak currents are indicated rather than strong ones.

7. If you have no galvanometer, the intensity of a galvanic current can be approximately determined by the burning sensation produced in the skin by the electrodes when they are applied to it.

8. The "polar method" is more painful when the faradaic current is employed than when the galvanic current is used. It is not well to separate the poles of a faradaic machine too widely; pain is intensified, and no special benefit is gained by so doing. Remember that the faradaic current has no fixed polarity. A galvanometer will record the difference between the current produced by the "make" and "break" of the circuit only; hence it is of no value in determining the intensity of the faradaic current actually administered to a patient.

9. The "polar method" is absolutely requisite to electro-diagnosis when the galvanic reactions of nerve or muscle are being tested. It constitutes the best method also of administering the galvanic current for therapeutical purposes, because it is usually important that the anode or cathode exert its special influence upon the part diseased. The farther apart you place the poles, the less is the effect of the indifferent or neutral pole upon the part which you wish chiefly to influence.

Although clinical experience seems to prove that we obtain different results in the majority of cases by employing the anode or cathode upon the part to be influenced, I am inclined to question the correctness of the view that those effects are in any way dependent upon the direction of the transmitted current. We know that it is not possible to confine an

electric current to any one channel by means of animal tissues. Every current becomes diffused to a greater or less extent, as is illustrated in diagrams prepared by Erb and other authors upon electro-therapeutics. It is probably more correct to view the special effects obtained by employing the positive and negative poles of a galvanic battery as the effects of the poles themselves, rather than the result of the direction of the current.

10. Remember that the anode or positive pole of a galvanic battery is the sedative pole, and the cathode or negative pole is the stimulating or irritating pole. When the cathode is made the indifferent pole, it is well to use a very large electrode.

11. Do not change the polarity of a current during its application to a patient any oftener than circumstances demand. As a rule, it is unnecessary to do so at all. It causes unnecessary irritation, which should always be avoided. In the treatment of neuralgia, diseased conditions of the brain or spinal cord, and painful points, it should never be done without some special reason. It is positively contra-indicated when catalytic effects are desired.

12. When galvanic currents to the head are indicated (especially if the current is to be sent through the brain), employ only those of moderate intensity (save in exceptional cases), and do not reverse the current unless there is good reason for so doing. When you read about thirty-cell currents being sent through the brain, it is safe to suppose that the battery was not of the most active kind, or that the ability of the patient to endure such a current was very exceptional. It is rare to meet with a patient who can tolerate a current of more than from three to six milliampères through the brain, and it is not safe to break currents of high intensity when employed about the head.

13. Static electricity will sometimes produce muscular contractions when faradaic currents will not. In hysterical conditions, some of the spasmodic diseases, sciatica, and organic spinal affections, it is well to try this form of electricity when galvanism fails to afford relief.

14. Respecting the duration of individual applications of electricity in its various forms, my experience teaches me that short sittings accomplish as much, and often more, than long ones. I seldom exceed five or six minutes, unless I am endeavoring to induce catalytic action, to benefit chronic articular rheumatism, etc.; or when I am employing general faradization, general galvanization, central galvanization, electrolysis, the galvano-cautery, or other procedures which require a longer sitting. Frequently, thirty seconds to two minutes is all that is required when some particular part of the body is alone to be galvanized or faradized.

15. It is impossible to lay down any rule which will guide you in determining the frequency of the applications required by any individual

case. It is seldom necessary to employ this agent oftener than every day, and three sittings a week will suffice in the majority of cases. If the disease is of a chronic type, it is often advisable to occasionally discontinue treatment for a few weeks, and then to renew it with vigor. Experience has taught me that the effects of electricity are more vigorous after such intermissions. It is often well to change from galvanic to faradaic, and again to static currents, from time to time, in the treatment of obstinate diseases which fail to progress satisfactorily.

16. I would advise you to be persistent in employing this agent when your judgment tells you that it is advisable to begin it. Many of the chronic forms of cerebral and spinal diseases are materially benefited and often completely cured by a proper course of electrical treatment which has been followed, with occasional intermissions, for some months during each year for several years.

17. As adjuncts to a course of electrical treatment, you will find massage, baths of various kinds, a change of climate, enforced rest in bed, and judicious internal medication, indicated in special cases. Delicate subjects, who suffer from neurasthenia, hysteria, persistent neuralgias, mental depression, sleeplessness, morbid fears, excessive "nervousness," rapid or extreme emaciation, profuse and persistent sweating of the palms or feet, dyspeptic symptoms, and the thousand other manifestations of debility, are especially benefited by these adjuncts to a judicious use of electricity.

18. When simple excitation of motor or sensory nerves is demanded, the faradaic or static current is the best one to employ.

19. As a counter-irritant, and in the treatment of anæsthesia, dry faradization with a wire brush excels all other electrical applications, unless it be the use of static electricity.

20. In spasmodic diseases, in neuralgia, and other like conditions, galvanism and static electricity are alone of material service.

21. Interrupted galvanic currents are of service when muscular contractions of a forcible character are desired. When degeneration of a nerve exists, these cannot be produced by the faradaic current.

22. The size of the electrodes modifies the density of the current directly. When large, the current is less dense because it is more diffused. The cathode should, as a rule, be larger than the anode when electrical applications are being made.

STATICAL ELECTRO-THERAPEUTICS.

We have now discussed at some length in this section (1) the physics of this form of electricity; (2) the improvements made from time to time in machines designed to generate it; and (3) the various methods of application of this agent.

We are now prepared to consider more intelligently the various diseased conditions of the human body which static electricity has been shown either to ameliorate or arrest. In this connection I take the liberty of first quoting from an admirable paper of my friend, Dr. W. J. Morton, published some years ago upon this subject, certain paragraphs which relate to the rise and fall of static electricity as a therapeutical agent. He says:—

"In 1730 Mr. Stephen Gray, of London, first insulated and electrified a human subject, and in 1734 the Abbé Nollet received the first spark drawn from a body thus insulated. From this incident undoubtedly sprung the modern idea of electro-therapeutical science, for Nollet pursued electrical investigations to great lengths, and as early as 1746 was treating paralytics by insulation, sparks, and shocks. About this time, also, Professor Kruger, of Helmstadt, and Kratzenstin, his pupil, cured paralysis by electricity, and Klyn cured by means of sparks a paralyzed arm. These cases were the first strivings of modern electro-therapeutics, but they produced little effect on medical practice.

"It was a publication in 1748 by Jallabert, professor at Geneva, that first drew the earnest attention of the medical world to the real curative power of electricity. Jallabert restored to perfect motion and sensation in two months a locksmith's arm which had been paralyzed during fifteen years. In the meanwhile the invention and perfecting of the electric machine and Leyden jar paved the immediate way to the practical use of electricity as a remedial agent, and soon, following the success of Jallabert, the whole medical world was awake on the subject of medical electricity. At Montpelier, under the auspices of Sauvages, president of the Academy of Medicine, the people flocked in multitudes to have their ailments relieved, and so great was the number of successful treatments that the physicians were obliged to appeal to the priests to protect them from the charge of witchcraft. Deshais, in 1749, wrote a dissertation upon the Montpelier experiences. Quelmalz, Linnæus, and Zetzel followed him, and from this period onward, up to the year 1800, works* on the subject multiplied in all countries.

"It is interesting to recall that Franklin, in 1752, treated paralytics at Philadelphia by statical electricity.

"It is evident, then, that statical electro-therapeutics was already, at the end of the last century, entering upon a marked career of service to medicine when galvanism and the voltaic pile, in 1800, extinguished it at the very height of its progress. It is not improbable that its abandonment was a loss to medical science.

*Besides those mentioned, the most important are by De Haen, Watson, Franklin, Priestley, Gardane, Sigaud de la Fond, Bertholon, Cavallo, Wilkinson, and Manduyt; the latter is particularly valuable to the student of medico-statical electricity.

"Up to comparatively recent times frictional electricity for medical purposes was produced from a single glass wheel. Its *tension was low* and its *quantity small.* But the invention of Holtz, in 1865, marked out for modern statical electricity the possibilities of a new career. In the Holtz machine we have an apparatus simple and durable in construction and capable of furnishing electricity of high tension and in great quantity. And by means of the Leyden-jar condensers, and of the possibility of increasing the number of wheels, both tension and quantity are within the control of the operator. At a given length of spark or tension, every additional wheel adds only to the quantity, and Holtz machines with as many as twenty revolving wheels have been constructed, in which the quantity, of course, was very great. This very fact of a greatly increased working quantity of statical electricity justifies the expectation that modern electro-statical therapeutics will take a step greatly in advance of its past.

"Statical electricity, as we have already seen, has never had fair play in modern medicine. The older practitioners (1740 to 1800) have left us glowing records of its value—records embodied in a period of literature still full of fruitful suggestion in other branches of medicine, though in none more advanced than in the treatment by electricity. The physicist of to-day cannot neglect the work of Franklin, of Symmer, of Du Faye, of Cavendish, and the long line of the men of their time who unrolled to view the mysteries of a new science. No more can the physician neglect, from a medical point of view, De Haen, Boze, Bertholon, Nollet, Wilkinson, Cavallo, Manduyt, and a dozen others. True, the mantle of their labors decked in a degree the new galvanism and the newer faradaism, while in the act statical electricity dropped from sight. It found conscientious revivers in Sir William Gull, Golding Bird, and Wilks in 1850, and thereabouts, and it is gratifying to note in their writings the highest appreciation of its merits. When at last it fell from their hands again abandoned, it was only and simply because of the inconvenience of administering it. The machine of their day refused to work in the damp of London fogs, and it was necessary in the electrical room of Guy's Hospital to keep a large fire constantly burning to dry the air; and even to-day, in Paris, one may visit the rooms of a practitioner heated summer and winter.

"But these disadvantages have now been removed. Statical electricity was again revived, and with great success, by Professors Clemens in Germany and Sewanda in Austria. In France, its revival has already received a notable impetus emanating from the famous clinic at Salpêtrière, where the presiding care of Professor Charcot and the efficient labors of Dr. Vigouroux have each contributed to place statical electrotherapeutics on a scientific modern basis."

The remarks of Dr. Morton which I have quoted are peculiarly significant in this connection. During the years in which I have personally been engaged in experimenting with various modifications and improvements upon the original model of Holtz's induction machine which have suggested themselves from time to time to my mind, I have had ample opportunities to observe, in the daily routine of my practical office work, the effects of static electricity upon many patients afflicted with diversified diseases. In preparing this article I have carefully searched through the records of quite a large number of cases where it has been most successfully employed by me. I have been struck in many instances with the rapidity with which it effected an apparent cure; in other cases, with the permanency of its beneficial results; and in all, with the simplicity and ease of its application. To a lady, for example, it is a matter of no small moment that she is freed from the necessity of divesting herself of any garments worn, and that almost any part of the body can be treated without exposure or annoyance. To the busy practitioner, also, to whom time is valuable, it is not unimportant that several patients can be treated simultaneously; and that no delays are caused by waiting for each one to remove and replace their clothing.

Again, the application of "static insulation" is far more agreeable than "general faradization" or "general galvanization;" and, in my experience, it is fully as efficient in many cases in its remedial action as either of the methods referred to. The inconvenience to the patient of having to disrobe almost completely, and the distaste which many naturally exhibit to having a wet electrode or the operator's wet hand rubbed over the skin for from ten to twenty minutes is entirely obviated. With a sufficiently large insulated platform several patients can, if desired, be given a static bath in the physician's consulting-room in the same period of time as would be consumed in administering general faradization to one patient, and be spared the annoyances mentioned.

I do not mean to infer that some cases do not require the use of faradaic or galvanic treatment; nor would I be construed as casting any reflection upon the therapeutical value of the methods which were first suggested and employed by Drs. Beard and Rockwell, of New York. The question at issue is simply one of convenience to the patient and the physician; provided that the indications of the case justify the trial of the static bath as a substitute for "general faradization" or "general galvanization."

In the second place, I think it has been justly claimed for static electricity that some of its therapeutical effects are more certainly and rapidly obtained than by means of any other form of electrical application.

As examples of such effects, I prominently mention (1) the *relief of contractured muscles;* (2) the *relief of certain forms of pain;*

(3) the *production of muscular contraction and the restoration of muscular power after the "reaction of degeneration"* has shown itself; (4) the *improvement of muscular power and general sensibility in certain organic spinal diseases;* and (5) the *stimulation of the skin in certain trophic neuroses.*

Respecting this statement, I take the liberty of again quoting certain paragraphs from the writings of my friend, Dr. Morton. He says:—

"We may now ask the special question, *Why, above and beyond other forms of electricity, does static electricity cure?* I will offer two explanations, and these are, first, simple mechanical disturbances, followed by a local alteration of nutrition; and, secondly, reflex action from irritation of the peripheral distribution of nerves.

"With regard to the first, when the electric discharge in the form of a spark takes place in a resisting medium like the various parts of the human body which are submitted to it, a very great mechanical disturbance in the tissue at the point of discharge must inevitably result. A piece of paper, for instance, held between the electrode and the skin is perforated by the spark. A parallel to the mechanical action referred to, though in a less localized and less powerful degree, is to be found in ordinary physical exercise or in massage. From this point of view, static electricity by the method of sparks has, in a special degree, owing to its high tension, great advantages. The spark strikes a sharp, incisive, and penetrating, though scarcely painful blow, and, often repeated in a given region, creates, by simple disturbance, a great alteration in the nutrition of the part. This, at least, is the only way in which I can account for the almost instantaneous relief and cure, after a few applications, of a large class of pains seated in deep and superficial fasciæ, and due to subacute and chronic rheumatism. Neither blisters, other violent counter-irritation, nor medicine will dissipate these pains, while, on the other hand, static electricity will subdue them at once.

"The contraction of muscles is also often due to the same mechanical effect of the spark, just as muscles of the thigh may be made to contract by a snap of the finger or sharp blow from a percussion-hammer.

"With regard to the second explanation,—that by reflex action following a peripheral irritation of the terminal sensory filaments and endings,—a very intricate question is opened, which we can no more than glance at here.

"How can simple electrification by insulation and the drawing of sparks, it is asked, produce the decided effects that are claimed for it? Static electricity, it is said, owing to its high tension accumulates merely on the surface of the body, and does not penetrate into the deeper organs, while the spark is merely the briefest kind of current.

"Recent investigations on the irritative action of applications to the skin have thrown a new light upon this question, and show that, though previously unexplained, the effects of the great accumulation of electricity on the surface and the sharp blow of the spark were, in truth, effects based upon a true physiological principle,—the principle named by Brown-Séquard, its recent expounder, "the phenomena of inhibition." A few drops of chloroform applied to the neck of a guinea-pig produced, on some occasions, an epileptic attack; on others, the nerves and muscles became highly excitable to stimulation.

"But the most notable effect of irritating applications of chloroform, as well as other substances, was a general anæsthesia; reflex symptoms were inhibited and muscular excitability lost.

"An interesting element has entered into our physiological and therapeutical studies,—that of the reflex phenomena of peripheral irritation. And we may at once place under this single heading a large number of facts long familiar.

"External irritant applications, in one form or another, have always formed an important element in medical treatment. And most of these applications have been used to relieve pain, or in some way modify the general sensibility, either in contiguous or remote parts.

"Familiar examples are blisters, sinapisms, cupping, the actual cautery, ammonia, the moxa, aqua and acupuncture, and in later days the magnet, the tuning-fork, and hypodermic injections of water into the thoracic walls for the purpose of allaying the cough of phthisis. The latest novelty in this direction is the electric percutor of Baudet, consisting of a tuning-fork kept in vibration by electricity, and communicating to any desired nerve or part, by means of a slender rod, the mechanical vibrations originated in the fork.

"Charcot, after cautious experimentation, has given his adherence to the statement that metals (metallotherapy) do produce effects contiguous and remote when applied to the skin,—that the magnet also produces similar effects, both upon general sensibility and muscular power. Vigouroux has pointed out that the vibrations of a tuning-fork, either alone or communicated to a sounding-board, provoke similar phenomena.

"Here, then, in this collection of well-known facts, and in the broad generalization of Brown-Séquard, drawn from his recent experiments, we have at last, it seems to me, found the law which governs the results produced and to be expected from statical electrification, as well as from some other uses of electricity. This law is the effect produced upon remote parts by affecting the peripheral distribution of the sensory nerves, and the effect produced is most commonly relief of pain or spasm in a remote part; and in this principle of inhibition from peripheral

application may doubtless be found the explanation of many of the definite and hitherto inexplicable effects of static electricity.

"The 'insulation' alone holds the entire sensory peripheral distribution of the skin in its grasp. Every nerve-filament is vibrating, is polarized, or affected, whatever term we choose to use, by the tense layer of electricity or electrical influence collected on the surface, there bound by the natural laws of physics, and only waiting to be drawn off by a spark or diffuse itself gradually into the atmosphere, while in the spark itself is found a still more potent and localized stimulating agent."

I have quoted the preceding paragraphs from the pen of Dr. Morton, because they coincide in the main with my own views; the possible exceptions being a few technical points in which I might not fully agree with that author.

From conversations held with him I am led to infer that his experiences and my own have been generally in accord respecting the therapeutical effects of each of the several methods of statical applications already described by me in preceding pages.

It is extremely difficult to formulate general deductions respecting any therapeutical agent. Such attempts necessarily tend to evoke criticism, because exceptions to every general statement may be brought forward as evidences of their unreliability. I am, however, inclined to offer the following general deductions respecting static electrical applications for the benefit of the reader, with the *proviso* that they may not apply to every case, and that they be not construed too literally:—

First.—My experience has not confirmed the view (heretofore advanced by some authors) that the *positive pole* of a static machine has a "tonic" and the *negative pole* a "depressant" action.

I have found, after repeated experimentation, that either pole seems to answer equally well upon most patients. I commonly employ in my office the positive pole, however, because it happens to be the most conveniently connected with the patient.

Second.—As a curative agent, *I regard static electricity as of great value.*

While galvanism must always hold a preëminent place in electrical therapeutics, because of the chemical effects so obtained, there are certain diseased conditions in which static electricity is unquestionably superior to faradaism and galvanism.

Third.—It has been shown in preceding pages that the *static induced current fulfills all the known indications of faradaism.*

It has, moreover, two great advantages over the faradaic instrument, namely, that a constant polarity is obtained and a much greater electromotive force. It is also less painful than the faradaic current.

Fourth.—Static electricity possesses a decided advantage in some cases where faradization or galvanization have either given negative results or have apparently lost their remedial power after their use has been too long continued.

It is a common expedient with medical electricians to shift from one form of current to another from time to time whenever the progress of the case seems unsatisfactory. Under such circumstances franklinism forms another link to the chain, and greatly aids us when faradaism and galvanism have both proven inefficient.

Fifth.—I have found *heavy static sparks to surpass any other form of electrical application for the relief of contractured muscles.*

The sparks are withdrawn from the part so affected in rapid succession for about five minutes.

Post-paralytic contracture, old deformities from preternaturally shortened muscles, and the various forms of obstinated and protracted tonic muscular spasm often yield like magic to the influence of heavy sparks.

Sixth.—It is well known that *certain forms of pain often disappear at once after static applications.*

The most marked type of pain so relieved is the so-called "*rheumatic muscular pain*," or that observed in genuine *muscular rheumatism.* I have seen many such cases where one application of heavy sparks to the seat of pain for a few minutes has caused permanent relief.

Again, *neuralgias* of a distressing kind are often dissipated after a few applications of heavy indirect static sparks for from five to ten minutes at a sitting.

Finally, I know of no other agent which exerts so marked an effect of a happy kind upon the "*lightning pains*" observed in locomotor ataxia as does the heavy static sparks.

Seventh.—The application of the spark, both by the direct and indirect methods (Figs. 176 and 177) *excites powerful muscular contractions.*

This effect is often desired in the treatment of *hemiplegia* and *other forms of motor paralysis.*

Some authors recommend the employment of "static shock" (Fig. 178) for cases of paralysis of long standing; but, personally, I am inclined to regard this form of application as too severe for most patients.

I have often obtained a complete restoration of muscular power in special nerve-trunks by static sparks alone after the "reaction of degeneration" was fully developed and all faradaic excitability had ceased.

Eighth.—Cases which exhibit a marked *impairment of sensation* (whether of touch, pain, or temperature) are generally improved, in my experience, by the use of static sparks over the anæsthetic area more rapidly than by the faradaic or galvanic currents.

I have encountered several very striking cases which illustrate this point admirably, but a lack of space precludes the insertion of their histories.

Ninth.—Remarkable effects of static sparks upon that *form of baldness* known as the so-called "*ivory spots*," or *alopecia arieta*, have been observed by myself through the courtesy of my friend Dr. F. B. Carpenter, of New York. I have seen several of his cases where he has wrought a wonderful change in the appearance of the scalp after several months of treatment of the bald spots by the "direct" spark. The growth of the hair, which had apparently been totally destroyed over the affected regions, is attributable probably to the rekindling of the circulatory and nutritive conditions of the affected area upon the scalp.

Tenth.—As a *general tonic* and also as a *stimulant to depressed nervous functions*, "*static insulation*" (Fig. 180) *seems to be particularly of service.*

I employ static electricity constantly by this method in *neurasthenia*, with marked benefit.

I have observed also remarkable improvement in *disturbed visceral functions* (such, for example, as dyspepsia, habitual constipation, diabetes, vertigo, asthma, etc.) after the use of static insulation for from ten to twenty minutes at a sitting.

Many such cases have expressed to me the greatest delight at the beneficial effects which such an application invariably produced. For the past three years, I have used my static machine almost exclusively as a means of *improving the "general nervous tone" of patients*, in preference to my faradaic or galvanic apparatus. It is much more satisfactory to patients because of its ease of application; and, as far as I have observed, equally effective as a tonic.

Eleventh.—I am inclined to think that those authors who have written upon static electricity as a therapeutical agent in a lukewarm spirit have probably been supplied with an apparatus which has been ineffective because it generated too slowly or imperfectly.

The *size and number of the revolving plates* and their thorough *protection from atmospheric changes* are factors of the greatest importance.

As I have already said, many of the static machines sold to the profession are hardly more than mere toys. Any machine which gives a thin spark (even if a long one) lacks one essential factor to success as a therapeutical agent, namely, QUANTITY.

Twelfth.—I have used static insulation and sparks with satisfaction in the treatment of *chronic inflammatory and spasmodic diseases;* such, for example, as influenza, phthisis, bronchitis, asthma, laryngitis, neuritis, synovitis, etc.

Three cases of chronic synovitis of the knee-joint of an intractable form recovered completely under my care within a month, under the daily administration of static sparks to the affected joint.

Many cases of bronchitis and asthma have been greatly benefited by insulation and sparks to the chest.

I have used static insulation (followed by the withdrawal of sparks from the spine and abdomen) upon subjects afflicted with dyspepsia, flatulency, and constipation. In many instances this form of electrical treatment gave very marked relief.

The influence of this agent upon visceral derangement is, however, a field for future investigation. It gives promise of happy results. As yet, my personal experience is too limited to justify me in formulating any positive conclusions respecting the method which is best employed in individual cases.

Thirteenth.—Static electricity is of value in the treatment of *hysterical states* and *other allied conditions.*

Charcot has long been an advocate of this therapeutical agent in such cases. My own experience teaches me that it is of great service as an aid to recovery; although I believe that in a very large proportion of these subjects reflex irritation from "eye-strain" has to be combated by the relief of anomalies in the eye or its muscles before a perfect restoration of health can be attained. I have discussed this field elsewhere. (See *New York Medical Journal*, February, 1886, and April, 1887.)

In closing, I would state that the length of this article (already too prolonged, perhaps) precludes the insertion of the histories of many typical cases which would be of great interest in this connection. To fairly illustrate the subject in its many ramifications a very large number of lengthy records would, however, be required.

Medical literature within the past ten years, and antiquated works, also, fairly teem with cases reported by leading men in the profession here and abroad, which illustrate their concurrence and firm belief in the views expressed in preceding pages.

Static electricity is to-day, for the second time, generally recognized by the profession as a valuable therapeutical agent. Most of the leading neurologists have now a Holtz induction machine as a part of their office equipment.

The later editions of recognized works upon electricity as applied to medicine show almost without exception that this variety of electricity is deemed worthy of more attention than it received in the earlier editions. It has passed through its stage of neglect and distrust safely. It is steadily regaining the popularity it so justly achieved in the eighteenth century.

SPECIAL ELECTRO-THERAPEUTICS.

We have thus far discussed the various methods of employing electricity in a general way, and there remains for us to consider how we shall proceed in employing this agent when special organs are diseased. I would preface my remarks upon this field with the statement that the curative properties of electricity must, of necessity, be modified by the pathological conditions which exist in each individual case. The prognosis is naturally more grave in some conditions than in others.

For example, a patient who has motor paralysis which is due to *degenerative changes* in the cells of the anterior horns of the spinal gray matter will not usually recover the power of motion completely, while he may do so if the paralysis be due to a cerebral or spinal lesion which is not accompanied by degenerative nerve-changes. Again, all forms of functional nervous derangements are more amenable to electrical treatment (if judiciously employed) than are the graver results of organic disease of the nerve-centres. A muscle which has atrophied from disuse can usually be restored, while one which has wasted from imperfect nutrition (resulting from a degenerated nerve) may possibly withstand all efforts to improve it. The therapeutical use of electricity is subject to the same influences as that of any other remedial agent, and the prognostic conditions are not always the same even among cases of the same nature.

In previous pages I have given many hints relating to the differential diagnosis which you will be called upon to make in nervous diseases, and enough has been said in reference to the anatomy and physiology of the nervous system to assist you in properly interpreting abnormal nervous phenomena. I shall therefore give, in closing, directions only as to how to employ electric currents upon different parts of the body without entering to any extent into the causation of the symptoms which you will be called upon to treat. Remember, however, that accuracy of diagnosis is the basis of cure in a large proportion of cases.

ELECTRICITY IN CEREBRAL AFFECTIONS.

Experiment has shown beyond dispute that galvanic currents can be made to pass through the substance of the brain when inclosed within the skull. It is much less certain whether the same may be said of faradaic or static currents. The beneficial results which are obtained by the two latter (and possibly many of the effects of galvanism as well) upon cerebral diseases are to be attributed, in my opinion, chiefly to the alterations produced in the blood-supply of the brain. Some of the most remarkable results obtained by neurologists from the employment of electricity upon the head itself or the cervical ganglia of the sympathetic

are unquestionably due to an alteration produced in the calibre of the cerebral vessels. I have never been convinced that *organic* lesions of the brain can be cured by the direct use of this agent on that organ. On the other hand, I am fully satisfied that the symptoms of cerebral hyperæmia and anæmia are directly influenced by galvanism and static electricity. I believe that any unprejudiced mind can be readily convinced of the scientific accuracy of this conclusion. I have treated many patients (who gave undisputable evidences of basilar hyperæmia by the deflections of the needle of a calorimeter), and have brought them to a state of perfect health within the space of a few weeks by galvanism of the head. The calorimeter confirmed the cure in these cases by the absence of deflection, which existed before treatment was commenced. In some instances of this condition, static electricity proves a very valuable adjunct to galvanism. I will give in detail a few of the methods which, in my experience, may be employed in cerebral diseases with a prospect of great benefit to the patient.

Cerebral Hyperæmia.—First ascertain by means of a calorimeter the situation and extent of the congestion. Test all parts of the head. When necessary, do so by separating the hair and bringing the poles as closely as possible in contact with the scalp. It is not necessary, as a rule, to shave the head. In case very accurate observations are demanded, this step may have to be taken,—as, for example, when a cerebral tumor is suspected to exist.

At the nape of the neck, over the mastoid processes, upon the temples, and over the forehead, no hair exists to interfere with the determination of the relative temperament of the two sides, or of different regions of the corresponding side. The calorimeter will aid you in diagnosis and treatment; if properly used, it is sometimes invaluable.

The following are the steps in treatment most generally useful:—

(1) Apply the cathode to the nape of the neck, close to the skull, and the anode over the forehead. Make stabile applications for one or two minutes to each side of the forehead, the cathode remaining stabile. (2) Make labile anodal applications to the forehead transversely for one minute. (3) Move the cathode to the mastoid region of each side, place the anode centrally on the forehead, and continue each stabile application for from thirty seconds to one minute. This may make the patient dizzy. (4) Do not use a current which produces pain to the patient, but have as great intensity as he can comfortably bear. (5) Never reverse the current when the poles are on the head.

These applications may daily be alternated with "*insulation*" and the "*electric head-bath*," if you possess a static machine. The sittings should occur daily until the symptoms are cured and the calorimeter ceases to show its previous deflection.

It is sometimes well to *stimulate the superior cervical ganglion* by placing a small anode in the fossa behind the angle of the jaw, and the cathode on the seventh cervical spine, and to slowly interrupt the current. Caution must be exercised against employing too strong currents.

Finally, active *faradization of the limbs* is sometimes necessary, in order to draw the blood to the limbs. It is not well to employ this step if it causes an elevation of temperature.

The effects of this treatment should be to relieve the pain or sense of fullness in the head, the vertigo on rising, the mental confusion or distress, the insomnia, and the many other symptoms peculiar to this condition; and to steadily reduce the calorimeter deflections when the poles are in contact with homologous parts.

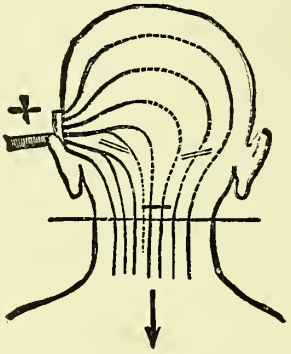


FIG. 189.—A SCHEMATIC REPRESENTATION OF THE DISTRIBUTION OF AN ELECTRIC CURRENT APPLIED UNILATERALLY THROUGH THE HEAD. (After Erb.) The anode (+) rests above the ear of the left side. The cathode (—) is supposed to be at the nape of the neck, and to exert its influence as far as the line drawn horizontally across the neck.

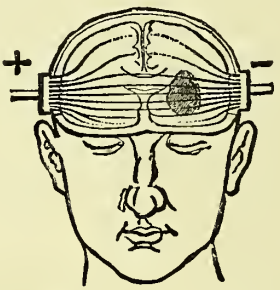


FIG. 190.—A SCHEMATIC REPRESENTATION OF THE COURSE OF ELECTRIC CURRENTS SENT TRANSVERSELY THROUGH THE HEAD. (After Erb.) The cathode (—) is represented as placed on the same side as the lesion.

CEREBRAL ANÆMIA.—I should advise you to begin the use of very weak galvanic currents after an attack of embolism. I believe that currents of this kind sent transversely through the head from the temples, and occasionally in the antero-median plane, assist in absorbing the collateral œdema and cause a diminution of the collateral hyperæmia. I prefer to use the cathode on the side of the embolic obstruction when transverse currents are employed. In my opinion, it tends to promote absorption and to contract the vessels far more than the anode. The paralyzed muscles should be treated separately, by methods given in detail later.

Some four years ago Löwenfeld published some deductions relative to the action of galvanic currents upon the brain, based upon experimental researches. Although their accuracy has been justly called in question by authors of note, my own experience leads me to confirm them in part

and to attach some importance to them. These conclusions were as follow: (1) anode at forehead and cathode at neck cause contractions of the vessels of the pia; (2) anode at neck and cathode at forehead cause dilatation of the vessels of the pia; (3) when transverse currents are employed, the cathode causes contraction of adjacent vessels, and the anode dilatation.

When cerebral anæmia of a *general character* exists (as a manifestation of poverty of the blood, defective heart-power, etc.), general faradization, central galvanization, and static electricity by insulation are often of material benefit. The removal of the cause by judicious medication, etc., is, of course, vital to successful electrical treatment.

Hemiplegia of Cerebral Origin.—A very large proportion of patients with hemiplegia from cerebral causes owe the paralysis of their limbs to hemorrhage, softening, or embolism. The electrical treatment should be directed to both the brain and the muscles. It should not be commenced (save in the case of embolism or thrombosis) until a month has elapsed since the attack. Each patient's susceptibility to the agent should be carefully studied, and the strength of current employed should be modified accordingly. The muscles may be treated by faradization or galvanization, or by the static current (indirect sparks being drawn from the paralyzed limbs). The brain should be subjected to galvanization only, or to static insulation.

If the patient fails to show improvement within a month after the treatment has been daily applied, or if the improvement of the first few days is rapidly lost in spite of continued treatment, the prognosis, as regards marked amelioration of the paralysis by electrical applications, is grave.

Hemianæsthesia is best treated by the wire brush upon the dry skin in connection with the secondary faradaic current. I have also obtained some remarkable effects with the combined current (as before stated), and also with the static current, in cases where the faradaic current was ineffective.

Post-paralytic Rigidity (occurring late) is the result, in most cases, of secondary changes within the spinal cord. The supervention of pigmentation of the nails, œdema, a shiny skin, disease of the joints, and other evidences of trophic alterations, points to a serious and often permanent destruction of the nerve-centres.

Hints which have been given under the head of general electrotherapeutics will guide you in modifying the treatment according to the exigencies of each individual case. The remarkable improvement which some hemiplegics obtain through the instrumentality of electrical treatment should impress you with the necessity of employing it long enough to ascertain whether its continued use is indicated.

Monoplegia or Monospasm.—These conditions are particularly indicative of cortical disease. The muscles affected are a guide to the convolution attacked. I have covered this field in a previous section. The indication in such a case is to improve, if possible, the nutrition of the diseased part directly by galvanism, and also to stimulate the muscles functionally associated with it. I employ for this purpose a "medium" electrode over the diseased convolution, the indifferent electrode being placed over the centre of the sternum. It is my custom to employ both poles separately to the head for an interval of two minutes each at a sitting. The monoplegic limb may be treated by labile galvanic applications, the wire brush and faradization, or the indirect spark by means of a static machine.

Duchenne's Disease.—The morbid changes in the nuclei of the

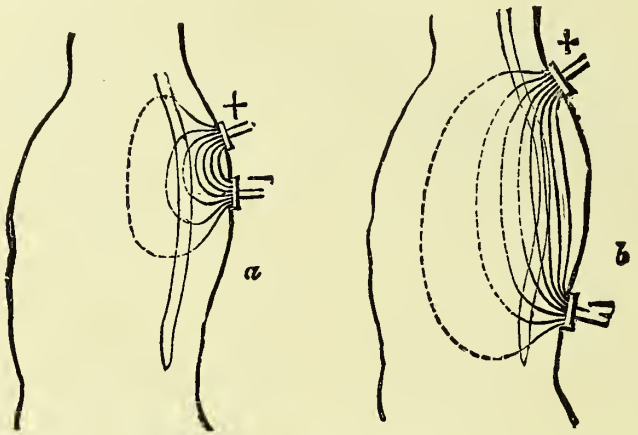


Fig 191.—A Schematic Representation of the Distribution and Density of the Threads of Current with regard to their Entrance into the Spinal Cord. (After Erb.) In *a* the poles are placed near each other. In *b* the poles are more widely separated. The size of the electrodes shown in the cut is the same for both the anode and cathode.

medulla which accompany bulbar paralysis may, in some cases, be held in check for a while and the symptoms markedly improved by placing the positive electrode (of large size) at the nape of the neck and as close as possible to the foramen magnum, and applying the negative electrode (covered with absorbent cotton and attached to a long handle) successively to the pharynx, fauces, tongue, cheeks, and lips. As strong a current as the patient can easily endure should be used. The duration of the sitting should not exceed five minutes. It is well to complete the sitting by passing transverse currents through the neck, so as to excite the muscles concerned in deglutition. Some authors recommend the employment of currents through the head, both longitudinally and transversely.

ELECTRICITY IN SPINAL AFFECTIONS.

There are various ways of bringing the spinal cord under the influence of electrical currents. The method of application selected in any individual case will depend somewhat upon the symptoms which the patient presents, and also upon the character and seat of the lesion. The diagrammatic cuts of Erb, which illustrate the diffusion of electrical currents, show in a graphic way the effects of close approximation and wide separation of the poles. We may also modify some of the morbid conditions of the spinal cord by electrization of the extremities when the indifferent pole is placed over spinous processes. It is well to increase the size of the electrodes proportionately to the strength of the current employed.

Fig. 191 illustrates the effect of separation of the poles when applications of electricity are made to the spinal column. Some of the threads of current depicted are rendered ineffective on account of their diffusion. This is made more apparent in Fig. 192.

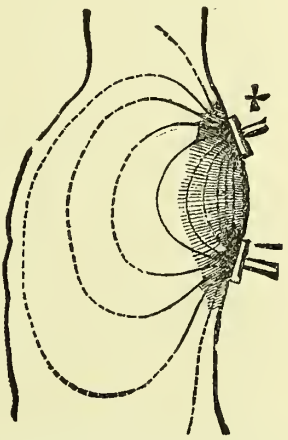


Fig. 192.—A Schematic Representation of the Density of the Current upon Application of the Electrodes to the Same Surface and in Close Relation to each other. (After Erb.) The dotted lines indicate the ineffective threads of current. The shaded portion represents the zone of greatest intensity.

ELECTRIZATION OF THE SPINAL CORD.

To treat properly of the various methods which may be used when the application of electrical currents as a therapeutical measure for the relief of spinal diseases seems indicated, it would be necessary for me to enter into greater detail regarding spinal diseases than the space allotted to these lectures will permit of. I am reluctantly forced, therefore, to summarize somewhat hastily the main points which my experience with this agent leads me to indorse. Most of my readers are probably already familiar with the pathological changes which exist in connection with the more common diseases of the cord; but, if any are not so, these changes should first be studied and thoroughly mastered before they can hope to successfully combat them.

Galvanic currents are of greater service in the treatment of spinal diseases than faradaic or, perhaps, the static,—chiefly on account of the depth of the tissues affected and the chemical and molecular changes which galvanic currents tend to induce.

Spinal electrodes should be of large size.

The applications may be either stabile or labile, the former being of the greatest benefit when the spinal lesion is circumscribed in extent,

and the latter when a larger part of the spinal cord is affected. If labile applications are indicated, the movements of the electrodes should be made somewhat slowly.

In directing galvanic currents to the *cervical* and *upper dorsal segments* of the cord, it is well to place one electrode of medium size behind and below the ear alternately on the two sides of the neck, while the other is applied to the spine.

Points of tenderness to pressure along the spine should be subjected to stabile applications of the anode. They should be sought for in each individual case with care and separately galvanized.

The *strength* of the currents employed should be modified in individuals by the condition which is presented for treatment. Weak currents of from two to five milliampères act best, as a rule, when excessive irritability of the organ exists; chronic pathological conditions respond better to currents of greater intensity. I often use eight to twelve milliampères of current in chronic cases.

It is advantageous, in some subjects, to make *electrical applications to the limbs* when the cord is affected. Stimulation of the peripheral nerves and the muscles connected with the segments of the cord involved should be particularly aimed at, although the electrization of the skeletal muscles and the skin should not be exclusively confined to the limits thus indicated. It is my custom to employ the "*combined current*" (previously described) when applications to the limbs are thus made. This form of current is particularly indicated when the muscles exhibit a tendency toward atrophy. The electrode which rests upon cervical or lumbar enlargements of the spine should be of large size, while that used upon the limbs should be of medium size, so as to direct the combined currents to the nerves or muscles affected.

If *galvanism alone is used upon the limbs* in spinal disease, it is often beneficial to the patient to break the current by an interrupting electrode, or to reverse its direction by means of the commutator.

Some authorities advocate *faradization of the vertebral region and of the limbs* in conjunction with galvanic applications. I have seen, in a few instances, some remarkable effects follow the employment of the wire brush alone in poliomyelitis of children, and I can see no reason to doubt its occasional efficacy in other forms of spinal disease.

In some unexplained way the excitation of muscular action and stimulation of the cutaneous nerves exert in many instances a remedial effect upon lesions of the spinal cord.

It is not always possible (as, for example, in poliomyelitis) to excite muscular action by faradaism alone. In these cases interrupted galvanic currents, or the "combined current" (galvano-faradaic), will accomplish the desired end, I have repeatedly observed beneficial effects of this

treatment in locomotor ataxia, and Rumpf has published some cases which sustain this view in which the wire brush was used upon the arms and legs daily for about five minutes.

In all acute *inflammatory disorders of the cord* I deprecate the use of electrical applications. When the acute stage has passed, or when the disease has assumed a chronic type, many of the effects of the disease (as, for example, muscular paralysis, rectal or vesical complications, incipient caries, anæsthesia, etc.) may often be greatly relieved by its judicious use. The current-strength employed in such cases usually varies from five to eight milliampères. The applications should be made daily. When possible, it is important that the reader localizes early the seat of the structural lesion and concentrates the treatment, for a while at least, upon the segments of the cord involved. The muscles, skin, bladder, rectum, etc., should be separately subjected to the influence of electricity in case they exhibit a loss of function.

ELECTRICITY IN PARALYSIS OR PARESIS.

Hypokinesis may be due to many different conditions; hence, its electrical treatment and prognosis must vary in accordance with the cause which excites it. You should remember that paralysis of a muscle is only symptomatic of other conditions,—such, for example, as lead-poisoning, diphtheria, hysteria, mechanical pressure upon a motor nerve, severance of a motor nerve, destructive processes or inflammation within the motor cells of the brain or spinal cord, and changes in the vessels. All of these tend to impair either the generating power of a motor centre, or the conducting power of a motor fibre.

Respecting the application of electricity to the seat of central lesions (*i.e.*, lesions of the brain or spinal cord) in cases of motor paralysis, De Watteville pertinently remarks as follows :—

"It is true that we have too often but a very imperfect idea of those processes in the nerve-centres upon which the symptom depends, and that we have no right to assume that the current has any specific curative influence upon any one of them; still, as a justification for central treatment in such cases, we may plead our very ignorance, we may urge the poverty of our therapeutical arsenal in arms wherewith to combat our enemy, and may also invoke the possibility of at least staying its progress by promoting nutrition of the surrounding portions of the nervous structures threatened by its invasion."

When the lesion directly affects the conductivity of a nerve, we have reason to believe that the direct influence of electrical currents upon the lesion tends to overcome the resistance offered to conduction by the disease-process, and facilitates the subsequent transmission of voluntary stimuli.

There are certain general rules that are applicable to the electrical treatment of paralysis of motility. These may be stated as follows:—

1. The treatment should not be alone confined to the region of the paralyzed muscles.

2. The seat of the exciting lesion should be ascertained early, if possible, and subjected to the influence of this therapeutical agent in an intelligent way.

3. If the motor paralysis is accompanied by anæsthesia, hyperæsthesia, or other sensory disturbances, or if the vaso-motor system of nerves be apparently implicated, the wire brush may often be used with advantage upon the skin in the vicinity of the lesion, and also over the muscles paralyzed.

4. Faradaic currents (provided they excite muscular action), or the cathode-pole of a galvanic battery (with interruptions of the current), are of use in exciting the conductivity of the nerve-tracts affected. Static electricity is also of great utility in inducing muscular contractions, and is generally less painful than strong faradaism or galvanic shocks.

5. The "combined current" (galvano-faradaic) is chiefly of service in overcoming trophic disturbances, which often manifest themselves in connection with motor paralysis.

6. I prefer labile applications to stabile in applying either faradaism or galvanism to the muscles. Stabile applications are preferable to labile when the brain, spinal cord, or peripheral nerve-trunks are to be influenced.

7. Never begin the use of electricity immediately after the onset of paralysis (when due to a central lesion). It is always best to wait until all danger of exciting a recurrence of the attack by stimulation of the nerve-centres has passed.

ELECTRICITY IN SPASMODIC AFFECTIONS.

Hyperkinesis is frequently encountered as one of the varied forms of external manifestation of irritative and destructive lesions of the central nervous system. For example, it is by no means uncommon to observe convulsions (of the clonic or tonic type), tremor, muscular rigidity and contracture, etc., in connection with morbid changes in the brain and spinal cord. By these symptoms we are often assisted in determining the seat of the lesion, although, as De Watteville remarks, "the pathogeny of spasm is one of the most obscure problems in neurology." On the other hand—as, for example, in many instances of chorea, epilepsy, hysteria, etc.—spasm may exist without any apparent structural changes in the nervous system; being excited by some source of reflex irritation, such as visual defect, phimosis, uterine displacement, insufficiency of the ocular muscles, etc. In tetanus the exciting cause is

generally found in one or more of the peripheral nerves. Sclerosis of the motor fibres of the lateral columns of the spinal cord is known to produce muscular contracture as a prominent symptom, probably because the inhibitory influence of the brain upon the reflex excitability of the spinal cord is arrested, or because the sclerosis directly excites the motor apparatus of the cord. The peculiar deformities produced by post-paralytic contracture, and the eccentricities of gait and posture exhibited by patients suffering from tetanoid paraplegia (lateral spinal sclerosis), are illustrative of the diagnostic value of tonic muscular spasm in the course of some spinal affections.

Respecting the effects of electrical treatment of spasm, I am convinced that in some cases many methods must be tried without benefit before the right one is discovered. I have occasionally had brilliant results follow some particular method, and subsequently I have been utterly disappointed when it was tried upon some other patient with identical symptoms.

I think that in this class of subjects more depends upon success in ascertaining and removing the cause than upon any electrical applications (valuable as they may be as adjuncts). The correction of an optical defect by glasses, the relief of ocular insufficiency by tenotomy or prisms, the operation of circumcision, the mechanical relief of a displaced womb, the removal of bad teeth, and many other such procedures, form the basis of an absolute cure in many cases which have been otherwise treated unsuccessfully. This fact is too often disregarded.

Electrical currents may be made to act upon these cases (1) as a sedative (chiefly the action of the anode and static insulation); (2) as a stimulant (the action of the cathode, the static spark, or faradaism); (3) as a counter-irritant; (4) as a check to the progress of some peripheral or central morbid state (catalytic action); and (5) as an agent for the destruction of some neoplasm, induration, etc. (electrolytic action), or as a cautery.

I have lately come to regard static electricity (franklinism) as more generally applicable to spasmodic conditions (hysteria, torticollis, blepharospasm, tremor, contracture, etc.) than either faradaism or galvanism. It seems, in my experience, to act more promptly, and to produce more lasting results than the methods more commonly recommended by authors. I would advise those who decide to purchase a static machine to try the effects of insulation, the "electric wind," and the indirect spark (as the circumstances may indicate) faithfully before they resort to galvanization or faradization. If good results are not obtained, they can easily substitute for it the other forms of treatment at a later date. I should never regard any case as hopeless until it had been thoroughly tried (after all reflex causes had been removed). I have cured

several severe cases of tonic spasm of the muscles of the neck in a few sittings by means of the indirect spark, and relieved many cases of suffering from other forms of spasm in a short time.

In EPILEPSY, the employment of galvanism alone has never, to my knowledge, resulted in a complete cure, although some decided benefits have been reported from its continued use. There is, to my mind, a close relationship in many cases *between epilepsy and ocular defect*, to which I have already called attention. This element in the causation of epilepsy certainly merits attention. When all defects in the visual apparatus have been corrected (in case such exist), or when other reflex causes (such as phimosis, for example) have been relieved, galvanism and static electricity may become valuable aids in controlling the subsequent convulsive attacks. Latent hyperopia, astigmatism, and insufficiency of any of the muscles of the eyeball may (and, in my opinion, often do) excite epileptic seizures. It is absurd to expect of electricity, or any other therapeutical agent, curative results when so important a source of irritation of the central nervous system is allowed to remain uncorrected.

Rockwell's method of employing "central galvanization" in epilepsy does not, to my mind, equal in beneficial effects the use of static insulation and the drawing of indirect sparks from the neck and back of the patient. It is my custom, however, in some cases to employ both of these procedures, each being used alone during alternate weeks for a period of two or three months with daily sittings.

In CHOREA I have obtained the best results with static insulation and sparks.

My previous remarks respecting the relationship between defects in the organ of sight and epilepsy apply with equal force to this disease and all other types of functional nervous derangements. I have discussed this subject more in detail when functional nervous diseases were being considered.

If galvanism is employed, it is best to subject the muscles affected with spasm to the action of the *anode*. The prognosis will depend somewhat upon the duration of the disease. The earlier you begin electrical treatment, the greater is the prospect of cure (provided all sources of reflex irritation have been successfully removed).

My experience with faradaism in the treatment of chorea has been somewhat limited; but the results obtained by me have not been so satisfactory as with static electricity.

In FACIAL SPASM (histrionic spasm) good results are occasionally obtained by following the plan of treatment suggested in connection with chorea; but treatment of the facial nerve alone is seldom satisfactory. I have one case at present under treatment, however, in which I have thus far had little, if any, success in my attempts to control the

spasm. It is a case of long standing, and is therefore more rebellious to treatment than if it were not chronic. The patient has an ocular defect which it is difficult to correct perfectly.

In these cases I have obtained the best results by subjecting both the cortical centres for facial movements and the nerve itself to stabile applications of the anode (the cathode being placed on the breast-bone), and by treating the nerve at intervals with static sparks drawn from the affected portions of the face. The electrode for the head should be large. The duration of each daily sitting should not exceed five minutes.

Nystagmus and blepharospasm belong to the choreic type of diseases, and are best treated by electrical currents, provided they are seen before the condition has become chronic. The prospect of a radical cure steadily becomes less as time elapses. If static currents are employed, wooden tips to the electrodes should be used. I usually treat these cases as if the seventh nerve were involved in all of its branches. Sometimes it is well to place the anode upon the mastoid process and the cathode upon the closed eyelid. The current should be very weak at first; should be gradually increased until faint flashes of light are perceived; finally, it should be again decreased to the faintest perceptible point.

Torticollis, or wry-neck, when subjected early to static sparks or strong faradization, may often be cured very rapidly. Interrupted galvanic currents are also of material benefit in some cases.

The spinal accessory nerve is usually the one which is at fault. A rheumatic origin may often be detected. If so, judicious medication will tend to hasten the cure.

Some cases of wry-neck are associated with symptoms of paresis. These have, as you might suspect, a more serious prognosis. Electrical treatment will prove, as a rule, only palliative. Too often organic changes have already occurred in the spinal accessory nerve, the spinal cord, or the vertebræ. The duration of treatment should extend over a period of months.

Spasmodic asthma may often be benefited by galvanism of the neck. I have previously described the steps of this procedure. Its beneficial effects are probably due to changes induced in the vagi. Drawing of indirect sparks (by means of the static machine) from the anterior and posterior surface of the chest has proved, in my experience, an admirable preventative against such attacks.

Some patients have assured me that they experienced a sense of great comfort after each sitting, and that the frequency of the paroxysms of asthma has been perceptibly modified by them. My experience in the electrical treatment of these cases is as yet somewhat limited; but I am inclined to believe that greater benefit can be derived from it than

from internal medication. Certainly it is worthy of a more extended trial as an adjunct, if deemed wise, to other remedial measures, or as a substitute for them.

In TETANUS (both of the traumatic and idiopathic varieties) two cases of cure have been reported by Mendel, of Berlin. He employed galvanization and subjected the muscles affected with spasm to the stabile influence of the anode, the cathode resting over the spinous processes of the vertebræ. The applications were continued for fifteen minutes, and the currents employed were mild ones. Bartholow suggests, when speaking of these cases, that the effect of these applications was probably due "to the influence of the currents upon the sensory nerves, thus lessening the intensity of the reflexes." The cures were complete in about ten days.

Personally, I have not as yet been able to test the effects of the different forms of electrical currents upon a case of tetanus. To my mind it would be very interesting, however, to observe the effect of static insulation and static sparks upon the spasms which occur paroxysmally in this disease. It is well known that this agent exerts a remarkable effect upon contracture of muscles. Thus far, to my knowledge, it has never been tried in tetanus.

SNEEZING, HICCOUGH, and COUGHING are spasmodic efforts of a reflex character. Occasionally they become distressing from their persistency. They may, in some instances, be relieved by faradization of the epigastrium, galvanization of the neck, and static electricity. De Watteville reports some curative effects of galvanization of the nasal mucous membrane in chronic cases of persistent sneezing.

ELECTRICITY IN DISORDERS AFFECTING SENSORY NERVE-TRACTS.

The discovery that different bundles of fibres which help to compose the substance of the spinal cord serve to convey sensory impulses only, and the later researches which have also been made respecting the paths of conduction specially prepared for sensations of pain, touch, temperature, pressure, the muscular sense, visceral sensations, etc., have a practical bearing upon both diagnosis and treatment.

Clinical observations go to show that, of the separate and distinct types of sensation enumerated, some may be partially or completely destroyed by diseased conditions without impairing the others. Thus, for example, a patient under certain conditions may be able to exercise his sense of touch with normal acuteness and yet be rendered absolutely insensible to pain; again, he may be unable to discriminate between degrees of heat or cold (provided the tests do not produce pain), although he retains unimpaired sensory faculties in all other respects. We are therefore forced to recognize a variety of types of anæsthesia as presenting themselves for diagnosis and treatment.

The sensory functions may be either increased (*hyperæsthesia*) or diminished (*anæsthesia*).

Either of these states may be of *organic origin* (by which we mean that structural changes in the nervous tissues accompany them), or of purely *functional origin*, in which case no structural changes can be shown to exist. Examples of the former are found in connection with central lesions (those of the brain or spinal cord), and with peripheral lesions of the sensory nerves or the organs of special sense, while examples of the latter are frequently encountered in connection with hysterical conditions, neurasthenia, cold, injury, imperfect capillary circulation, rheumatism, neuralgia, and many other morbid conditions.

In all forms of sensory disturbance the removal of the cause constitutes in many cases the basis of a cure, and the treatment will necessarily be modified by the causal indications.

Many suggestions which have previously been offered respecting electrical applications to the brain, spinal cord, and peripheral nerves are applicable alike to sensory as well as motor disorders when due to organic changes; hence, when this fact is borne in mind, it will be unnecessary to repeat what has already been given.

Anæsthesia.—In the treatment of this morbid condition nothing can surpass in its results the daily use of the *wire brush* for about ten minutes over the regions affected. This form of electrode should be applied dry and with the *secondary coil* of a faradaic machine. The stabile electrode should be well moistened and pressed closely in contact with some distant point.

If *trophic disturbances* coexist with anæsthesia, I have found the "combined current" (galvano-faradaic) to be more efficacious than faradaism alone.

Static sparks and static insulation often act wonderfully in functional nervous diseases.

Static *insulation* has been previously described. It should be administered daily for from ten to thirty minutes.

If the "*indirect spark*" is employed (see Fig. 176), the length of the spark should be sufficient to be perceptible to the patient, and the duration of the application should seldom exceed five minutes. It is well to administer a fusillade of sparks to the region of the spine after each insulation, in case the sensory disturbances are dependent upon hysteria or neurasthenia.

I seldom employ the "*direct spark*" (Fig. 177) except in the treatment of organic disturbances of sensation or motion. This form of administration should be used with extreme caution if the generating machine is a powerful one.

The "umbrella" electrode furnishes an agreeable and effective

method of concentrating static electricity to the head of the patient. The sensation is one which resembles that of a strong breeze circulating through the hair.

Hemianæsthesia (whether of cerebral or spinal origin) is often benefited by cutaneous faradization of limited portions of the area affected,—a point first observed by Vulpian, who employed this method with marked success.

Trophic disorders may occasionally manifest themselves, often in the skin, nails, hair, and muscles, when sensation is markedly affected. One such case (suffering from locomotor ataxia) was lately placed under my care. The fingers of both sides were almost destitute of sensibility to pain, and tactile sensation was impaired. The nails were thickened, loosened for half of their length, and deeply pigmented (as if stained with iodine). The terminal phalanges were "clubbed," the nails being bent almost in a semicircle. The skin was thickened and very hard under the loosened nails. The "combined current" (galvano-faradaic) with a wire-brush electrode caused decided improvement within a few weeks.

Neuralgia (when of idiopathic origin) is more successfully treated to-day by electricity than by any medicinal agent. In many instances it is cured in a few sittings.

It is well to bear in mind, however, the fact that neuralgic pains are very often symptomatic of causes more or less remote from the affected nerve, and that a permanent cure is impossible in many instances as long as that cause actively exists. Defective teeth, morbid processes in the bones, pressure upon a nerve, organic changes in the nerve itself, toxic diatheses, rheumatism, gout, reflex irritation from the eye, uterus, digestive tract, ovaries, etc., cardiac and pulmonary disorders, and many other morbid conditions, may be enumerated as among the exciting causes of neuralgia.

Respecting the electrical treatment of neuralgic pains (*per se*) the following deductions may prove of some advantage to you:—

1. If points of tenderness to pressure (*puncta dolorosa*) exist along the course of the affected nerve or its branches, it is well to subject them to stabile galvanic applications of the anode, the cathode being placed at a neutral point.

2. The anode should be made to cover as large an area as possible.

3. The duration of the sitting should not exceed five minutes, save in exceptional cases. The sittings may be repeated several times a day if necessary.

4. As a rule, it is unwise to break the current. In obstinate cases the current may occasionally be reversed, without changing the poles, by means of the commutator.

5. Faradization of the nerve and the use of the wire brush upon the skin have been recommended when galvanism proves unsuccessful in arresting the pain. It should not be used (in my opinion) until galvanism has been thoroughly applied.

6. It is well in obstinate cases to direct the applications of galvanism to the central origin of the affected nerve, as well as to its peripheral distribution.

7. Static electricity often produces marvelous results in neuralgia. I have more faith in it as a cure for sciatica than in any other remedial agent. It should be applied (by the "spark" method) over the affected nerve. One sitting has, in my experience, frequently arrested severe pain. It gives immediate relief, in most cases, to muscular rheumatism also, and to lumbago. Sufferers from muscular and neuralgic pains are perhaps as frequently encountered by the physician as any class, and static electricity should highly recommend itself to his confidence for such cases. The expenses of the outfit, and the fact that the machine is too large for transportation, will probably prevent its general use by the profession; but, until its effects upon a patient have been tried, I would caution against expressing an unfavorable opinion, even if galvanism, faradaism, and medicinal treatment have proved powerless to relieve the suffering.

8. The operation of electro-puncture of a nerve for the relief of neuralgia has proved of benefit in the hands of some neurologists; but it is an operation which, if injudiciously employed, will produce electrolysis, and serious results may follow its use.

9. The electrical treatment of various other forms of pain is similar to that of neuralgia. The judgment of the physician should be exercised regarding the position and size of the electrodes, the variety, strength, and duration of the current employed, and various other minor points suggested by the condition of the subject.

10. *Visceral neuralgias* (as, for example, the conditions known as hemicrania, migraine, gastralgia, enteralgia, hepatalgia, etc.) are often relieved by electricity, irrespective of the reflex or constitutional condition which induces the morbid state. The removal of the exciting cause, however, will greatly assist in making the cure a radical one. I have long taught in my lectures that I had yet to encounter a patient who had suffered for years from migraine who had not some defect in the eye or its muscles as an exciting cause. Experience leads me still to strongly assert this as my conviction. The same cause is very frequently manifested by paroxysms of spinal pain,—peculiarly so at two points, viz., between the scapulæ, and at the junction of the last lumbar vertebræ with the sacrum.

The currents which act best upon these cases are the galvanic and

static. I have in two instances employed faradaism in gastralgia with good results, but I regard it as inferior to galvanism or franklinism.

In treating the abdominal viscera by galvanic currents, one rheophore may often be attached with advantage to a rectal electrode, and the other to a large electrode placed over the organ to be influenced. I do not believe that polar effects are to be particularly aimed at. In some cases, an occasional substitution of the "combined current" (galvano-faradaic) for galvanism makes the improvement of the patient more rapid.

Static applications to the abdomen are best made by employing indirect sparks of about two inches in length. Long sparks are not borne well by sensitive subjects. If patients are subjected to static insulation only for twenty minutes daily, or to the electric spray over the abdomen, relief is often afforded and the application is painless. The clothing need not be removed in making applications of franklinism by either of these methods,—a point which renders the treatment particularly agreeable to ladies.

ELECTRICITY IN DISEASES OF THE CERVICAL SYMPATHETIC, THE VASO-MOTOR SYSTEM, AND ALLIED NEUROSES.

The CERVICAL SYMPATHETIC is undoubtedly, in rare cases, the seat of isolated morbid changes; but, as Erb remarks, these cases "constitute pathological curiosities." The morbid conditions which have been detected embrace inflammation, compression, traumatism, rheumatic conditions, etc. Such conditions may create either irritation of the sympathetic system or paralysis of its functions, or both simultaneously in different parts of the body.

Irritation of the cervical sympathetic produces pallor of the face and neck upon the affected side, with a sense of coldness in the parts. The pupils are dilated, the temporal arteries exhibit increased tension, the power of accommodation and the reaction of the pupil to light are both impaired, the eyeballs protrude slightly, and the secretion of sweat is diminished.

Paralysis of the cervical sympathetic induces the opposite conditions. The skin is red and hot, the patient suffers from a sense of heat in the skin, the pupils are contracted and exhibit normal reactions to light and accommodation of vision; the eyeball does not protrude, there are often headache and vertigo, the secretion of tears and sweat is increased, and the pulsation of the carotids is excessive.

In the electrical treatment of these opposed conditions Erb recommends stabile applications of the anode (with a strong current) until a change in the pupil is observed, if the condition of irritation exists. The same author suggests the use of the cathode with a feeble current, frequent interruptions, and occasional reversal of the poles, if the par-

alytic state is present. He places the "indifferent" electrode upon the spine. He also suggests applications of the wire brush, or labile galvanic currents, to the skin of the face and neck.

To the views of this author I would urge the advantage of trying the effects of static insulation and sparks directed to the neck and face.

Angioneuroses of the skin may assume one of two forms, viz., *spasm* or *paralysis*. They are most frequently observed in connection with neurasthenia and in hysterical patients. The abnormal contraction or relaxation of the vessels may cause (1) modifications in the color and the general "feel" and sensibility of the skin; (2) subjective sensations of heat, tingling, formication, etc.; (3) disturbances of perspiration; (4) awkwardness of movement of the part (especially in the hands); and (5) many reflex symptoms referable to the viscera.

Unnatural conditions of the vessels of the skin (spasm or paralysis) are most frequently observed in the upper limb, less frequently in the lower limb, and least often in the face and neck. They may be excited by a variety of causes,—such as fatigue, excitement, menstrual disturbances, malaria, exposure to cold, the effects of poisons, and direct irritation of the skin itself.

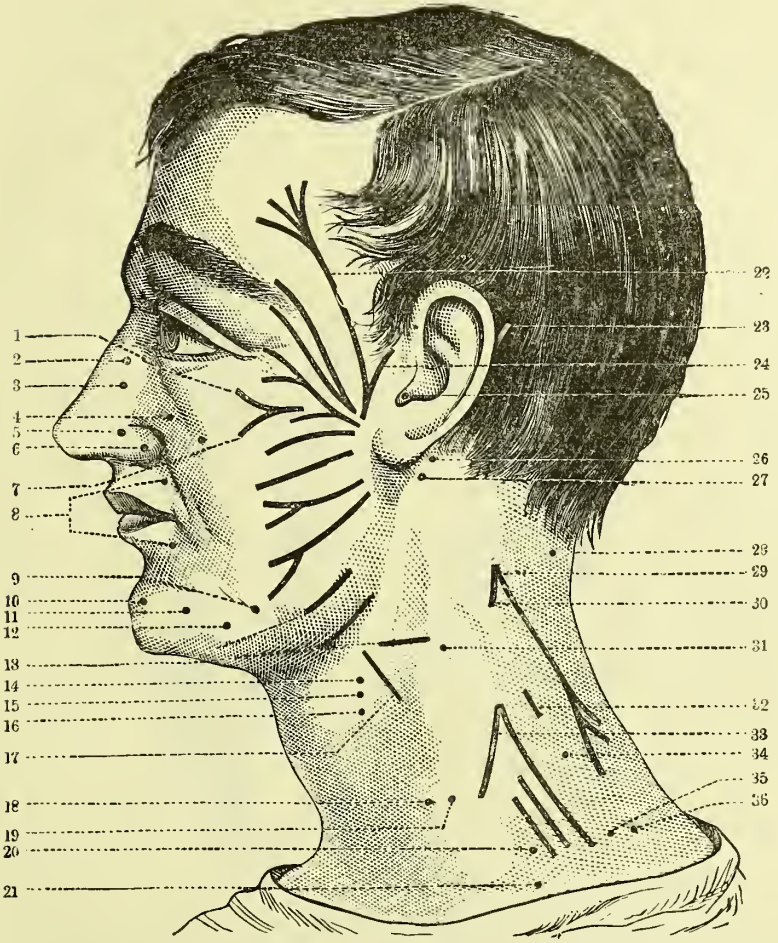
I have seen the skin (especially of the fingers) made as white as chalk in some cases, and in others rendered cyanotic, by *spasm of the vessels*. The muscles of the papillæ of the skin may participate in the spasm and produce the so-called "goose-flesh" appearance. Pain, tingling, formication, partial anæsthesia, and other disturbances of the sensory apparatus may occur as sequelæ to the vascular spasm.

Paralysis of the cutaneous vessels leads to directly opposite conditions. The skin may be made intermittently or permanently red, and feel unnaturally hot and extremely sensitive. Subjects so afflicted frequently suffer from insomnia, headache, disturbed heart-action, excessive perspiration, vertigo, and other visceral manifestations of irritability.

Respecting the electrical treatment of angiospasm and angioparalysis, the general rule may be given that weak or moderate applications of faradaism or galvanism to the affected part act best upon dilated vessels, and stronger currents upon those affected with spasm.

Applications of static electricity are often very beneficial to neurasthenic and hysterical subjects. Personally, I believe this method of treatment surpasses any other in its effects upon this class, although it is well to alternate with galvanism and faradaism when a case proves obstinate to treatment.

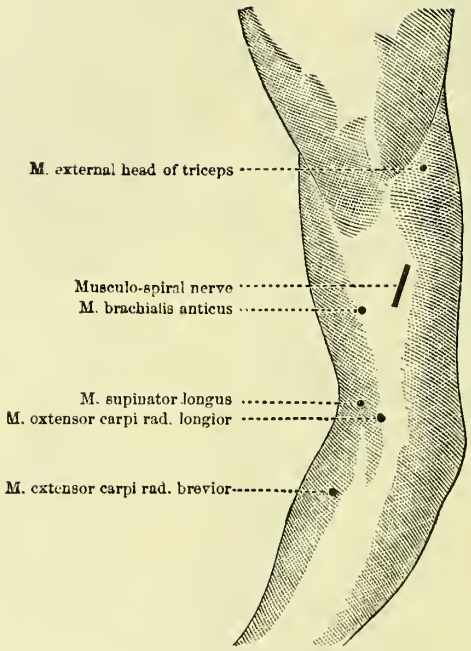
When any of the methods suggested are employed, it is well to subject both the vaso-motor centres and the nerve-trunks which supply the affected regions (as well as the parts directly) to the influence of electrical currents.



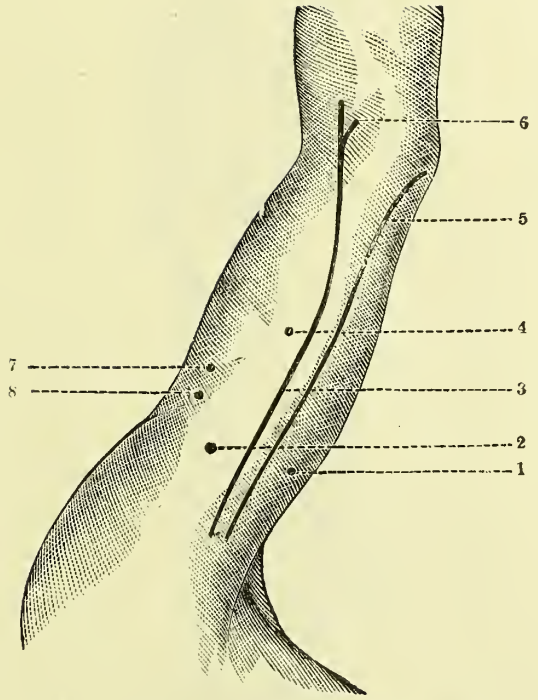
A Diagram of the Motor Points of the Face, showing the Position of the Electrodes during Electrization of Special Muscles and Nerves. The Anode is supposed to be placed in the Mastoid Fossa, and the Cathode upon the Part indicated in the Diagram.

1, m. orbicularis palpebrarum; 2, m. pyramidalis nasi; 3, m. lev. lab. sup. et nasi; 4, m. lev. lab. sup. propr.; 5, 6, m. dilator naris; 7, m. zygomatic major; 8, m. orbicularis oris; 9, n. branch for levator menti; 10, m. levator menti; 11, m. quadratus menti; 12, m. triangularis menti; 13, nerves, subcutaneous, of neck; 14, m. sterno-hyoid; 15, m. omo-hyoid; 16, m. sterno-thyroid; 17, n. branch for platysma; 18, m. sterno-hyoid; 19, m. omo-hyoid; 20, 21, nerves to pectoral muscles; 22, m. occipito-frontalis (ant. belly); 23, m. occipito-frontalis (post. belly); 24, m. retrahens and attollens aurem; 25, nerve—facial; 26, m. stylo-hyoid; 27, m. digastric; 28, m. splenius capitis; 29, nerve—external branch of spinal accessory; 30, m. sterno-mastoid; 31, m. sterno-mastoid; 32, m. levator anguli scapulæ; 33, nerve—phrenic; 34, nerve—posterior thoracic; 35, m. serratus magnus; 36, nerves of the axillary space. In this text m. = muscle; n. = nerve.

PLATE II.



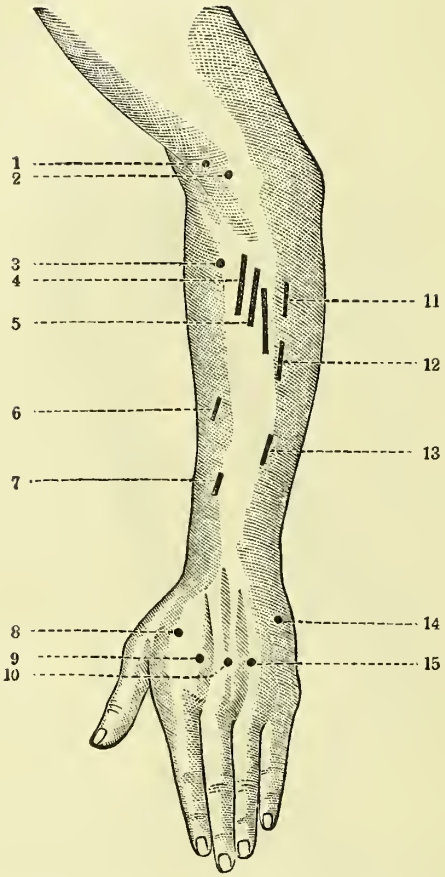
The Motor Points on the Outer Aspect of the Arm.



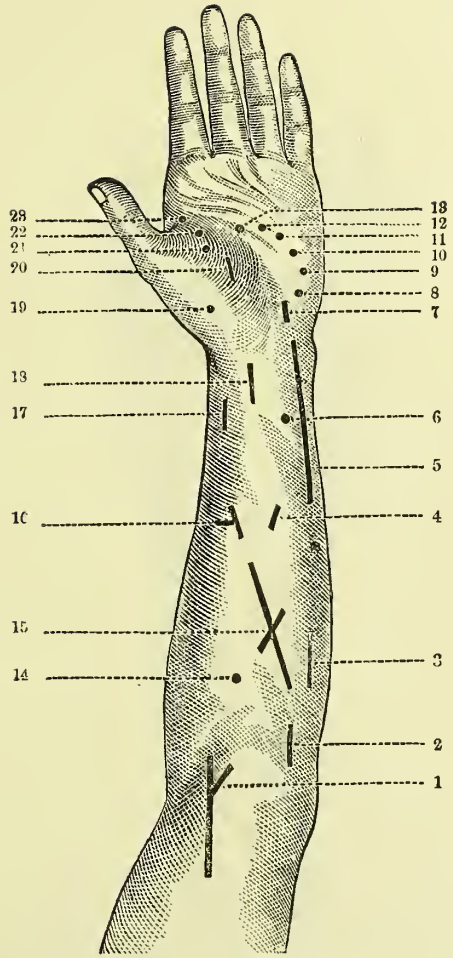
THE MOTOR POINTS ON THE INNER SIDE OF THE ARM.

1, m. internal head of triceps; 2, musculo-cutaneous nerve; 3, median nerve; 4, m. coraco-brachialis; 5, ulnar nerve; 6, branch of median nerve for pronator radii teres; 7, musculo-cutaneous nerve; 8, m. biceps flexor cubiti.

PLATE IV.



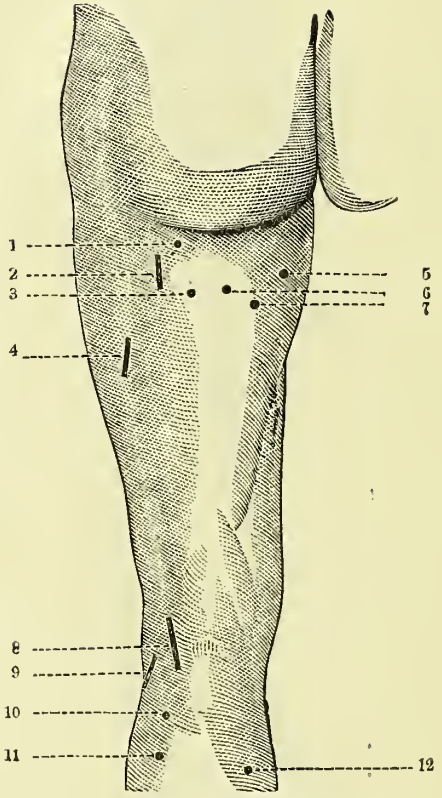
THE MOTOR POINTS ON THE EXTENSOR (POSTERIOR) ASPECT OF THE FOREARM.
1, m. supinator longus; 2, m. extensor carpi rad. longior; 3, m. extensor carpi rad. brevior; 4, 5, m. extensor communis digitorum; 6, m. extensor ossis. met. pol.; 7, m. extensor primi. internod. pol.; 8, m. first dorsal interosseous; 9, m. second dorsal interosseous; 10, m. third dorsal interosseous; 11, m. extensor carpi ulnaris; 12, m. extensor min. digiti; 13, m. extensor secund. internod. pol.; 14, m abduct. min. digiti; 15, m. fourth dorsal interosseous.



THE MOTOR POINTS ON THE FLEXOR (ANTERIOR) ASPECT OF THE FOREARM.

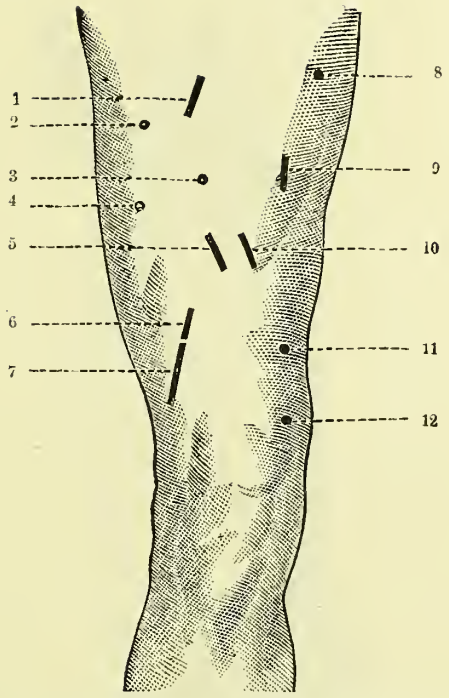
1, median nerve and branch to m. pronator radii teres; 2, m. palmaris longus; 3, m. flexor carpi ulnaris; 4, m. flexor sublim. digit.; 5, ulnar nerve; 6, m. flex. sublim. dig.; 7, volar branch of the ulnar nerve; 8, m. palmaris brevis; 9, m. abductor min. digit.; 10, m. flexor min. digit.; 11, m. opponens min. digit.; 12, 13, m. lumbricales; 14, m. flexor carpi radialis; 15, m. flexor profund. digitorum; 16, m. flexor sublim. digitorum; 17, m. flex. longus pollicis; 18, median nerve; 19, m. opponens pollicis; 20, m. abductor pollicis; 21, m. flexor brevis pollicis; 22, m. adductor pollicis; 23, m. first lumbricalis.

PLATE VI.



THE MOTOR POINTS ON THE POSTERIOR ASPECT OF THE THIGH.

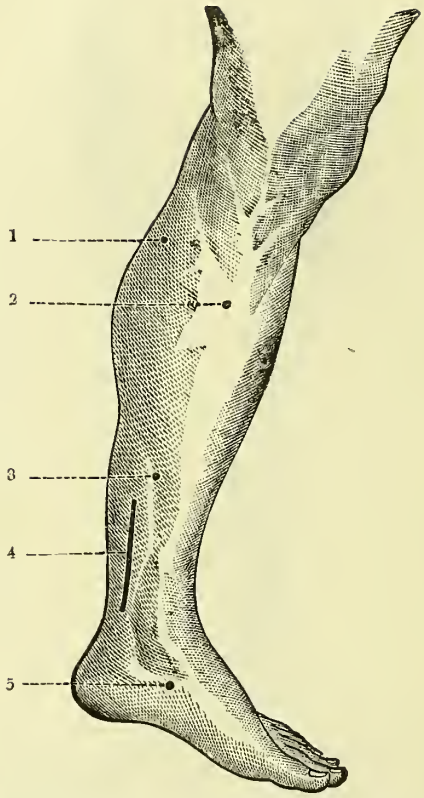
1, branch of the inferior gluteal nerve to the gluteus maximus muscle; 2, sciatic nerve; 3, long head of biceps muscle; 4, short head of biceps muscle; 5, adductor magnus muscle; 6, semi-tendinosus muscle; 7, semi-membranosus muscle; 8, tibial nerve; 9, peroneal nerve; 10, external head of gastrocnemius muscle; 11, soleus muscle; 12, internal head of gastrocnemius muscle.



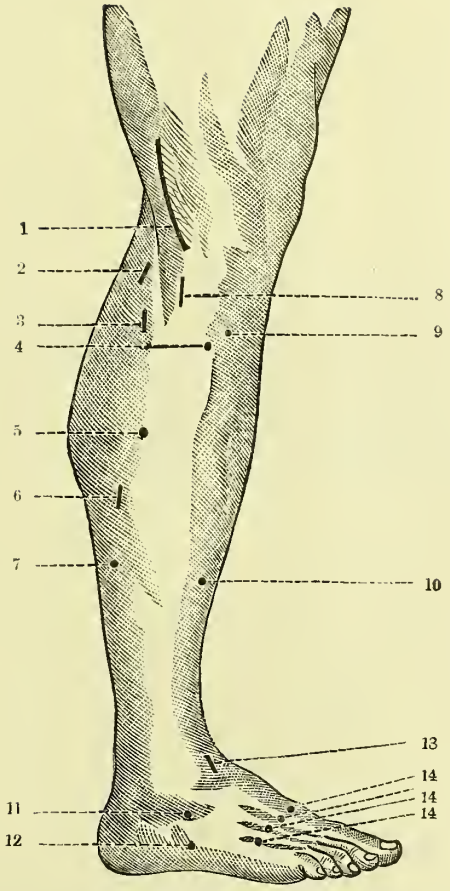
THE MOTOR POINTS ON THE ANTERIOR ASPECT OF THE THIGH.

1, erural nerve; 2, obturator nerve; 3, sartorius muscle; 4, adductor longus muscle; 5, branch of the anterior crural nerve for the quadriceps extensor muscle; 6, the quadriceps muscle; 7, branch of anterior crural nerve to the vastus internus muscle; 8, tensor vaginæ femoris muscle (supplied by the superior gluteal nerve); 9, external cutaneous branch of anterior crural nerve; 10, rectus femoris muscle; 11, 12, vastus externus muscle.

PLATE VIII.



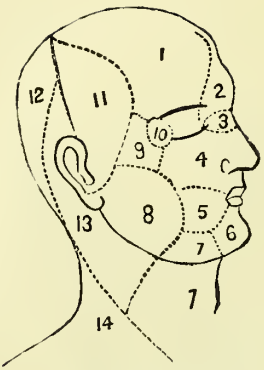
THE MOTOR POINTS ON THE INNER ASPECT OF THE LEG.
1, internal head of gastrocnemius muscle; 2, soleus muscle; 3, flexor communis digitorum muscle; 4, posterior tibial nerve; 5, abductor pollicis muscle.



THE MOTOR POINTS ON THE OUTER ASPECT OF THE LEG.

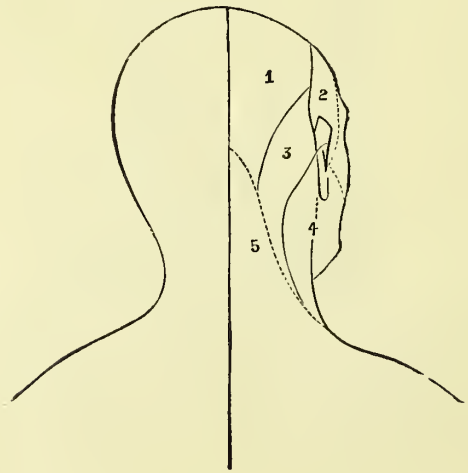
1, peroneal nerve; 2, external head of gastrocnemius muscle; 3, solcus muscle; 4, extensor communis digitorum muscle; 5, peroneus brevis muscle; 6, soleus muscle; 7, flexor longus pollicis; 8, peroneus longus muscle; 9, tibialis anticus muscle; 10, extensor longus pollicis muscle; 11, extensor brevis digitorum muscle; 12, abductor minimi digiti muscle; 13, deep branch of the peroneal nerve to the extensor brevis digitorum muscle; 14, 14, 14, dorsal interossei muscles.

PLATE X.



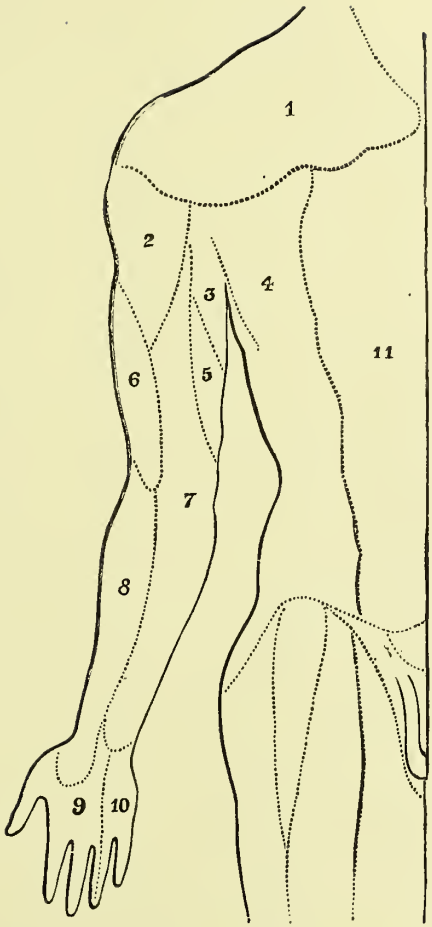
THE NERVOUS DISTRIBUTION OF THE SKIN OF THE HEAD. (After Flower, but slightly modified.)

1, region supplied by the *supra-orbital* branch of the fifth nerve; 2, region supplied by the *supra-trochlear* branch of the fifth nerve; 3, region supplied by the *infra-trochlear* branch of the fifth nerve; 4, region supplied by the *infra-orbital* branch of the fifth nerve; 5, region supplied by the *buccal* branch of the fifth nerve; 6, region supplied by the *mental* branch of the fifth nerve; 7, region supplied by the *superficial cervical* from the cervical plexus; 8, region supplied by the *great auricular* from the cervical plexus; 9, region supplied by the *temporo-malar* branch of the fifth nerve; 10, region supplied by the *lachrymal* branch of the fifth nerve; 11, region supplied by the *auriculo-temporal* branch of the fifth nerve; 12, region supplied by the *great occipital* (a spinal nerve); 13, region supplied by the *small occipital* from the cervical plexus; 14, region supplied by the *supra-clavicular* from the cervical plexus.



THE CUTANEOUS NERVE SUPPLY OF THE POSTERIOR PORTION OF HEAD AND NECK. (Modified from Flower.)

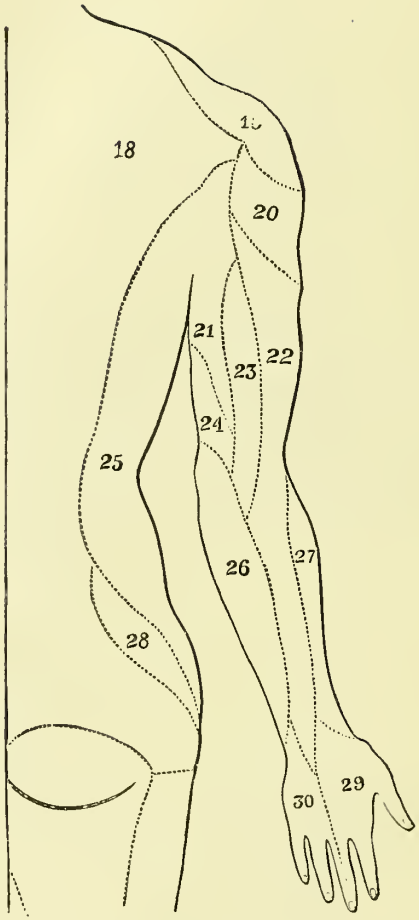
1, region supplied by the great occipital nerve; 2, region supplied by the auriculo-temporal nerve; 3, region supplied by the small occipital nerve; 4, region supplied by the great auricular nerve; 5, region supplied by the third cervical nerve.



A Diagram of the Regions of Cutaneous Nerve Distribution in the Anterior Surface of the Upper Extremity and Trunk. (Modified from Flower.)

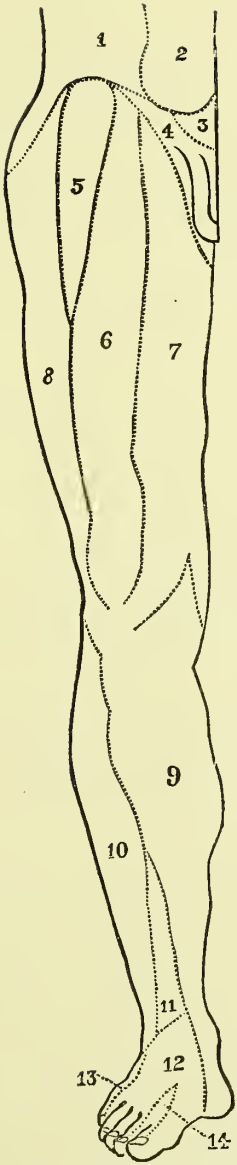
1, region supplied by the supra-clavicular nerve (branch of the cervical plexus); 2, region supplied by the circumflex nerve; 3, region supplied by the intercosto-humeral nerve; 4, region supplied by the intercostal nerve (lateral branch); 5, region supplied by the lesser internal cutaneous nerve (nerve of Wrisberg); 6, region supplied by the musculo-spiral nerve (external cutaneous branch); 7, region supplied by the internal cutaneous nerve; 8, region supplied by the musculo-cutaneous nerve; 9, region supplied by the median nerve; 10, region supplied by the ulnar nerve; 11, region supplied by the intercostal nerve (anterior branch).

PLATE XII.



A Diagram of the Regions of Cutaneous Nerve Distribution on the Posterior Surface of the Upper Extremity and Trunk. (Modified from Flower.)

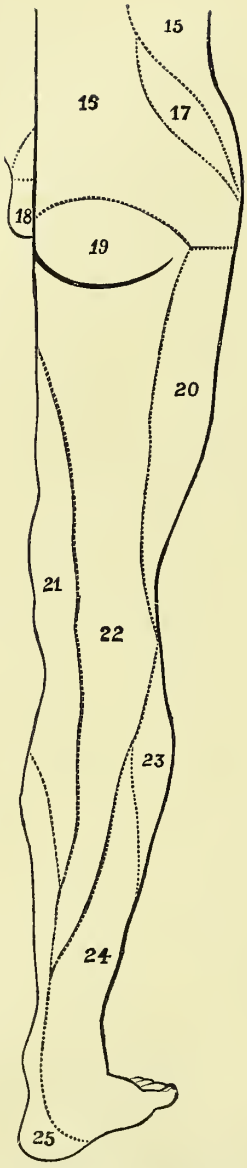
18, region supplied by the *second dorsal* nerve; 19, region supplied by the *supra-scapular* nerve; 20, region supplied by the *circumflex* nerve; 21, region supplied by the *intercosto-humeral* nerve; 22, region supplied by the *external cutaneous* nerve; 23, region supplied by the *internal cutaneous branch* of the *musculo-spiral* nerve; 24, region supplied by the "*nerve of Wrisberg*;" 25, region supplied by the *lateral branches* of the *intercostal* nerves; 26, region supplied by the *internal cutaneous* nerve; 27, region supplied by the *musculo-cutaneous* nerve; 28, region supplied by the *iliac branch* of the *ilio-inguinal* nerve; 29, region supplied by the *radial* nerve; 30, region supplied by the *ulnar* nerve.



A Diagram of the Cutaneous Supply of the Anterior Aspect of the Lower Extremity. (Modified from Flower.)

1, region supplied by the lateral branches of the intercostal nerves; 2, region supplied by the anterior branches of the intercostal nerves; 3, region supplied by the ilio-hypogastric nerve; 4, region supplied by the ilio-inguinal nerve; 5, region supplied by the genito-crural nerve; 6, region supplied by the middle cutaneous branch of the anterior crural nerve; 7, region supplied by the internal cutaneous branch of the anterior crural nerve and partly by the obturator nerve; 8, region supplied by the external cutaneous nerve; 9, region supplied by the long saphenous branch of the anterior crural nerve; 10, region supplied by the branches of the external popliteal nerve; 11, region supplied by the musculo-cutaneous nerve; 12, region supplied by the terminal filaments of the musculo-cutaneous nerve; 13, region supplied by the external saphenous nerve; 14, region supplied by the anterior tibial nerve.

PLATE XIV.



A Diagram of the Cutaneous Supply of the Posterior Aspect of the Lower Extremities. (Modified from Flower.)

15, region supplied by the lateral branches of the intercostal nerves; 16, region supplied by the posterior branches of the lumbar nerves; 17, region supplied by the iliac branch of the ilio-hypogastric nerve; 18, region supplied by the pudic nerve; 19, region supplied by the inferior gluteal branch of the small sciatic nerve; 20, region supplied by the external cutaneous nerve; 21, region supplied by the internal cutaneous branch of the anterior crural nerve; 22, region supplied by the small and great sciatic nerves; 23, region supplied by branches from the external popliteal nerve; 24, region supplied by the external saphenous nerve; 25, region supplied by the posterior tibial nerve.

GLOSSARY.

ACCOMMODATION OF VISION. The adjustment of the crystalline lens of the eye for the clear perception of objects within a radius of twenty feet from the eye. It is usually estimated, however, at about fourteen inches from the eye (Fig. 39).

ÆSTHESIOMETER. An instrument to estimate the acuteness of the tactile sense in any given part.

ÆSTHESODIC. Pertaining to the appreciation and conduction of sensations of various kinds. Centripetal.

AGEUSIA. A loss of taste.

AGRAPHIA. A loss of the ability to write or copy familiar characters correctly.

AKINESIA. A loss of voluntary motion.

ALEXIA. A loss of the power of reading.

AMBLYOPIA. Indistinct vision from structural changes accompanying disease of the eye or optic nerve.

AMIMIA. A loss of the power of making appropriate gestures.

AMNESIA. A loss of memory.

AMYGDALA. It literally signifies the "almond." The term is applied to a mass of gray matter seen after a section made through the tail of the caudate nucleus of the cerebrum (N C′ in Fig. 9).

AMYOTROPHIC. A term which signifies defective nutrition of muscles.

ANALGESIA. The abolition of sensibility to pain in any part.

ANARTHRIA. A peculiar form of imperfect speech due to interference with the speech-tract (Fig. 24).

ANODE. The positive pole of a galvanic battery.

ANOSMIA. The abolition of the sense of smell in one or both nostrils.

APHASIA. A morbid condition where speech, reading, or writing become impaired, either from an inability to properly coördinate the muscles, or from a defective interpretation of sounds or visual impressions.

APRAXIA. A loss of the power to recognize common objects and often to appreciate their ordinary uses.

ASEMIA. See *Asymbolia*.

ASSOCIATING TRACTS. Fibres which serve to unite the various physiological centres of each side of the brain, and also of the cord, with each other, so as to allow of a harmony of action and the proper performance of complicated mental and physical processes (Fig. 6).

ASTHENOPIA. An inability to use the eyes without great discomfort. It is usually dependent upon a disturbance of equilibrium in the eye-muscles, with or without some coexisting error of refraction.

ASTIGMATISM. An abnormal condition of vision caused by irregularity in the curvature of the cornea or crystalline lens of the eye.

ASYMBOLIA. Loss of power by a patient of signing or duplicating his or her customary signature.

ATAXIA. A morbid condition, characterized by a loss of the normal power of performing coördinated movements.

ATHETOSIS. Constant and uncontrollable movements of the fingers or toes (Fig. 47).

BASAL GANGLIA. Collections of nerve-cells buried within the substance of each cerebral hemisphere near to its base. See *Corpus Striatum* and *Optic Thalamus* (Fig. 1).

BASIS CRURIS. See *Crusta Cruris* (Fig. 11).

BELL'S PARALYSIS. Unilateral facial palsy,

BETZ'S CELLS. The motor cells of the cerebral cortex. They are arranged in groups of five or six, called "Betz's nests." They are found only in the so-called "motor area."

BLIND SPOT. The point in the retina where the optic nerve enters,—about two lines to the nasal side of the macula lutea.

Boulimia. Insatiable hunger.

Brach-Bomberg Symptom. An inability to stand without swaying or staggering when the eyes are closed. It indicates anæsthesia of the soles of the feet, and is frequently observed in locomotor ataxia.

Bulbar Symptoms. A train of symptoms referable to the tongue, lips, palate, pharynx, and larynx, caused by a degeneration of those nuclei in the medulla (Fig. 16) which preside over movements of the respective parts. (*Duchenne's disease.*)

Burdach's Column. The postero-external column of the spinal cord (Fig. 29).

Calorimeter. An instrument for the actual estimation of heat, or the comparison of the relative temperature of differing parts.

Capsular Fibres. Two distinct masses of fibres which embrace and act as a capsule to the *lenticular nucleus* of the corpus striatum. They are designated, from their relationship to this body, as the "internal capsule" and "external capsule" (Fig. 9). They are prolonged dorsad into the crus and pass cephalad into the corona radiata.

Cardialgia. Neuralgic paroxysms in the region of the heart.

Catalepsy. A morbid condition, characterized by coma and the so-called "waxy flexibility" of the muscles.

Cathode. The negative pole of a galvanic battery.

Central Convolutions. The ascending frontal and ascending parietal gyri of the cerebrum (Figs. 4 and 5).

Centre. A term commonly employed to designate some special subdivision of any collection of nerve-cells which has diversified physiological functions. It is often used synonymously with the term "nucleus."

Cheirospasm. Writer's cramp.

Cheyne-Stokes Respiration. An abnormal form of respiration in which the rhythm undergoes frequent and regular modifications.

Choked Disk. The condition of the retina observed in connection with neuro-retinitis (Fig. 87).

Cincture-feeling. A synonym for the so-called "girdle-pain." A sense of painful constriction of some part.

Clarke's Column. A column of cells in the spinal gray matter, probably associated with the transmission of visceral sensations to the cerebellum by means of the direct cerebellar column (Fig. 32).

Clavate Nucleus. A collection of cells which are structurally related to the fibres of the column of Goll, and in which they probably end. See also *Triangular Nucleus.*

Commissural Tracts. Fibres which tend to unite the cells of homologous parts of the cerebral hemispheres, chiefly of the cerebral cortex (Fig. 6).

Contracture. A permanent rigidity and shortening of a muscle. It is commonly observed in connection with lesions involving the crossed pyramidal tracts of the spinal cord.

Corona Radiata. This term embraces all the fibres which pass from the cerebral cortex toward the region of the crus (peduncular group). Some are structurally associated with the basal ganglia, while others constitute the so-called "capsular fibres" of the lenticular nucleus. See *Capsular Fibres.*

Corpus Striatum. This term literally signifies the "striped body." A ganglion of the cerebrum, consisting of two nuclei (the *caudate* and the *lenticular*). It is one of the so-called "*basal ganglia*" of the cerebrum (Figs 1 and 9).

Cortex. A term that literally signifies "rind" or "external covering." It is applied, therefore, to the gray matter of the convolutions of the cerebrum and cerebellum

Crossed Paralysis. A condition where right or left hemiplegia coexists with a paralysis of some cranial nerve of the opposed side. It varies in type, according to the cranial nerve impaired.

Crusta Cruris. The anterior or motor part of the crus cerebri (Fig. 11).

Cuneate Nucleus. See *Triangular Nucleus.*

Cuneus. A part of the cortex of the occipital lobe of the cerebrum, lesions of which tend to cause homonymous hemianopsia.

Delayed Sensation. A peculiar retardation of the transmission of tactile pain or temperature sensations to the seat of consciousness of such sensations within the cerebral cortex.

DIPLOPIA. Double vision. This may be *habitual*, as exists with strabismus; or *transient*, as observed in many cases of heterophoria. This distinction is of great clinical importance.

DUCHENNE'S PALSY. See *Bulbar Symptoms.*

DYNAMOMETER. An instrument to determine the relative muscular power in the hand or foot of the two sides.

DYSPHAGIA. Difficulty in swallowing. A frequent symptom of Duchenne's disease.

DYSPNŒA. Difficult respiration.

ECLAMPSIA. Acute attacks of epileptiform spasms.

ELECTRODES. The terminal attachments to an electrical instrument, by means of which electrical currents are applied to a patient.

EMBOLISM. Plugging of a blood-vessel by a clot or foreign body which has been transported to the seat of its lodgment by means of the blood-current.

EMMETROPIA. The power of vision with an eye whose axes are normal (Fig. 38).

ENCEPHALORRHAGIA. Cerebral hemorrhage. Cerebral apoplexy.

EPENDYMA. The gray lining of the ventricular cavities of the brain.

ESOPHORIA. A tendency on the part of the visual lines to deviate inward.

EXOPHORIA. A tendency of the visual lines to deviate outward.

FARADAISM. The employment of the interrupted current generated by the magnetizing and demagnetizing of a soft-iron core within a helix.

FASCICULIS TERETES. See *Round Bundle.*

FIBRILLARY TWITCHINGS. Slight muscular contractions of a fibrillary character. These may be observed after tapping or faradizing the skin, blowing on the skin, or exposure of the skin after disrobing. They are most frequently met with as a symptom of progressive muscular atrophy.

FILLET. See *Lemniscus Tract.*

FISSURE. A prominent and distinct demarcation between component parts of the brain, the spinal cord, etc. See *Sulcus.*

FOOT-CLONUS. A morbid reflex phenomenon observed at the ankle, in connection with spinal diseases.

FORMICATION. A feeling "as if ants were creeping over a part." One of the many subjective phenomena caused by organic and functional disease of the nerve-centres.

FRANKLINISM. The employment of electricity generated from glass plates by means of friction. The so-called "induction machines" are now generally employed for medical purposes.

FRIEDREICH'S DISEASE. The so-called "hereditary ataxia" or "generic ataxia."

GALVANISM. The employment of a current generated by means of a chemical action.

GALVANOMETER. An instrument for measuring the current-strength of a galvanic battery.

GANGLION. Any isolated collection of nerve-cells; usually possessing considerable magnitude and distinctly marked boundaries.

GENERIC ATAXIA. See *Friedreich's Disease.*

GIRDLE-PAIN. See *Cincture-feeling.*

GLIEDER'S MEMBERS. Subdivisions of the lenticular nucleus. See Figs. 6 and 9.

GLOSSOPLEGIA. Paralysis of the hypoglossal nerve, causing a loss of control of the tongue.

GOLL'S COLUMN. The postero-median column of the spinal cord (Fig. 29).

GRAPHOSPASM. Writer's cramp.

GUBLER'S LINE. An imaginary line connecting the points of apparent origin of the trigeminal nerve-roots (Fig. 26).

GYRUS. A synonym for "convolution."

HÆMATOMYELIA. Spinal apoplexy.

HÆMATORRHACHIS. Meningeal spinal apoplexy.

HEMIANÆSTHESIA. Impairment of tactile sensibility in one lateral half of the body.

HEMIANOPSIA. Blindness of one lateral half of the retina. Three varieties are to be clinically recognized,—the *homonymous*, the *bi-nasal*, and the *bi-temporal.*

HEMICHOREA. Convulsive twitchings of the right or left half of the body.

HEMIOPIC PUPILLARY REFLEX. A response of one lateral half of the pupil only to a concentrated pencil of light. It is advisable to throw the beam upon the iris at an obtuse angle.

HEMIPARAPLEGIA. Paralysis of one lower limb.

HEMIPLEGIA. Paralysis of the lateral half of the body,—right or left arm and leg.

HETEROPHORIA. A disturbance of the normal state of equilibrium in the muscles which move the eyes.

Hydromyelia. Cavities within the substance of the spinal cord.
Hydruria. An abnormal secretion of sugar by the kidneys.
Hyperæsthesia. An abnormal acuteness of sensibility in a part.
Hypergeusia. Abnormal sensitiveness of the taste-apparatus. Often accompanied by parageusia.
Hyperkinesis. Spasmodic disease.
Hypermetropia. A defect in vision caused by an abnormal shallowness of the eye (Fig. 38). Its existence is commonly masked by abnormal ciliary action. It is revealed after the full effects of atropine.
Hyperosmia. Abnormal acuteness of smell,—often associated with parosmia.
Hyperphoria. A tendency of the visual lines to assume different vertical planes.
Hypoglossal Tract. A bundle of fibres which serves to connect the cortical centre for the tongue with the hypoglossal nucleus of the medulla oblongata.
Internal Capsule. One of the paths of those peduncular fibres that are probably unassociated structurally with the cells of the basal ganglia of the cerebrum. See *Capsular Fibres*. These fibres constitute an internal capsule, as it were, to the *lenticular nucleus* (Fig. 9).
Inter-olivary Tract. A part of the lemniscus tract, lying between the olivary bodies of the medulla oblongata (Fig. 27). It probably constitutes a part of the so-called "fillet."
Kinesodic. Pertaining to the power of motility. Centrifugal.
Kussmaul-Landry's Paralysis. The so-called "acute ascending spinal paralysis."
Lemniscus Tract. A term used synonymously with the so-called "fillet." A bundle of fibres in the pons and medulla, which is probably associated with coördination of movement, and possibly also with our dependence upon the visual sense as an aid to coördination (Figs. 12, 36, 37).
Macropsia. An apparent magnifying of visual images by the eye,—due to paresis of the external rectus muscle. Objects appear abnormally large to the patient.
Macula Lutea. The retinal area for distinct visual perceptions.
Malum Cotunnii. Neuralgia of the sciatic nerve. Sciatica.
Mastodynia. Neuralgia of the breast.
Megalopsia. An apparent exaggeration of the size of objects when viewed by the eye,—due to paresis of the external rectus.
Micropsia. An apparent diminution of the size of familiar objects when viewed by the eye,—due to paresis of the internal rectus.
Migraine. Hemicrania. Sick-headache.
Milliampère. The unit of current-strength employed in medical treatment of disease by galvanism.
Milliampère-meter. A medical galvanometer for estimating the current-strength during galvanic applications.
Mogigraphia. Writer's cramp.
Monoanæsthesia. Impairment of tactile sensibility in some distinctly localized part.
Monoparæsthesiæ. Subjective sensory phenomena confined to some special part,—such as tingling, numbness, formication, etc.
Monoplegia. Paralysis of some special group of muscles not, as a rule, supplied by one nerve. It differs from paralysis of a spinal nerve-trunk in the latter respect.
Monospasm. Uncontrollable spasmodic movements of some special group of muscles not, as a rule, supplied by one nerve.
Motor Tracts. Those fibres which are functionally associated with voluntary motion. They arise from the cells of the "motor area" of the cortex of each hemisphere of the cerebrum. The "pyramidal tracts" are the paths for these fibres within the spinal cord. The motor fibres traverse, in order to reach the spinal cord, the following parts successively: The corona radiata, the internal capsule, the crusta cruris, the pons, and the anterior pyramids of the medulla oblongata (Fig. 12).
Muscular Sense. The power of analyzing any muscular effort being exerted by the patient. The relative position of the limbs is also thus estimated, the aid of the visual sense being excluded.
Mydriasis. Preternatural dilatation of the pupil.
Myopia. A defect in vision due to an increase of the antero-posterior axis of the eye over the normal standard (Fig. 38).
Myosis. Preternatural contraction of the pupil.
Myotonia Congenita. See *Thomsen's Disease*.

Myxœdema. A morbid state, characterized by a swelling and distortion of the outlines of the features, from a deposit of mucin beneath the skin.

Necrotic Softening. Death of a part from a sudden arrest of its blood-supply.

Neutral Pole. The pole whose effects are not being observed during a galvanic application by the polar method.

Nuclei of Stilling. The so-called "red nuclei" situated in the tegmentum cruris. See *Red Nuclei* (Fig. 11).

Nucleus. Any collection of nerve-cells, possessing some special physiological function; but often without distinct limits of demarcation from neighboring cells.

Nystagmus. Oscillatory movements of the eyeballs.

Œdema. Transudation of the sero-albuminous elements of the blood, without a rupture of the vessels.

Ophthalmoplegia. Paralysis of the muscles which move the eyeball, causing strabismus of varying types.

Optic Thalamus. This term literally signifies the "bed" of the optic fibres. It is one of the "*basal ganglia*" of each hemisphere of the cerebrum (Fig. 1).

Orthophoria. The normal state of the equilibrium in muscles which move the eyes.

Parageusia. Abnormal subjective symptoms referable to taste. This condition is usually observed in hysterical and insane subjects. These sensations are apt to be nauseous and perverse.

Paraphasia. The substitution of wrong words in conversation; an incorrect use of numerals, etc.

Paraplegia. Paralysis of the lower half of the body,—both legs and possibly the pelvic organs.

Paresis. Incomplete or partial paralysis.

Parkinson's Disease. Shaking palsy. Paralysis agitans.

Parosmia. Unpleasant subjective sensations of smell, or a perversion of the appreciation of the odors commonly encountered. This condition is usually observed in hysteria and insanity; and also during an epileptic aura, or from organic lesions of the olfactory nerve or brain.

Peduncle. A term commonly applied by neurologists to bundles of fibres which connect the hemispheres of the cerebrum and cerebellum with adjacent parts,—as, for example, the cerebral peduncles (crura), and the three pair of cerebellar peduncles.

Perimeter. An instrument for the determination and registration of the visual field of a patient.

Piesmeter. An instrument for the estimation of the "pressure-sense" in any given part.

Polar Method. The form of application of galvanism made with special reference to the determination of the individual effects of the postitive or negative pole.

Polar Reactions. The effect of the closure or opening of the galvanic current when either pole is applied to a nerve or muscle. In health, the reactions so obtained follow a recognized sequence, according as different degrees of current strength are employed.

Poliomyelitis Anterior. Inflammation of the cells of the anterior horns of the spinal gray matter. The so-called "infantile paralysis."

Polydipsia. Insatiable thirst.

Polyneuritis. Disseminated neuritis. Multiple neuritis.

Polyphagia. Voracity. Excessive hunger.

Posterior Longitudinal Bundle. A bundle of fibres supposed by Spitzka to assist in bringing into harmony the corpora quadrigemina, the nuclei of the fourth and sixth nerves, and those which govern muscles of the neck (Fig. 11).

Postero-external Column. See *Goll's Column.*

Postero-median Column. A term applied by Gowers to the column of Burdach. See *Burdach's Column.*

Post-paralytic Rigidity. A state of contracture which often develops in muscles after an attack of paralysis.

Presbyopia. Failure of the power of accommodation due to age.

Projection Systems. A classification of the various bundles of fibres which serve to unite component parts of the nervous system.

Prosopalgia. Facial neuralgia.

Prosopodysmorphia. Progressive facial hemiatrophy.

Psychical Blindness. An inability to properly interpret visual perceptions, due to a lesion of the cortex of the occipital lobes of the cerebrum.

Ptosis. Inability to prevent a falling of the upper lid over the eyeball. Paralysis of the levator palpebræ superioris muscle.

Pulvinar. The posterior tubercle of the optic thalamus. It is believed to be functionally associated with the optic fibres.

Puncta Dolorosa. Points of tenderness clinically observed along the course of nerve-trunks affected with neuralgia. They were first described by Valleix.

Pupillary Reflex. The contraction of the pupil caused by an excess of light entering the eye. It is to be distinguished from the contraction of the pupil observed when near objects are focused upon the retina.

Purkinje's Cells. A form of cell peculiar to the cerebellar cortex.

Pyramidal Tracts. Fibres which are so called because they pass through and compose the *anterior pyramids* of the medulla oblongata. They are probably functionally associated exclusively with motor impulses (Fig. 29).

Quintus Tract. The ascending root of the trigeminus nerve (Fig. 11).

Railway Paralysis. Spinal concussion. Erichsen's "railway spinal affection."

Raphania. Ergot poisoning.

Reaction of Degeneration. Abnormal electrical formulæ of the so-called "muscular reactions to galvanic currents." This is also associated with an impairment or a total loss of faradaic excitability of nerve and muscle.

Red Nuclei. Two collections of cells in the "tegmentum cruris," which are structurally associated with the superior cerebellar peduncles (Fig. 11).

Reflex Action. A sensory impression transformed into a motor impulse.

Respiratory Bundle. See *Round Bundle.*

Restiform Body. The inferior peduncle of the cerebellum. The so-called "processus e cerebello ad medullam."

Reticular Ganglion. A term applied by Spitzka to the gray matter of the "formatio reticularis" of the pons and medulla oblongata (Fig. 27).

Rheophores. The cords of an electrical battery which are employed to connect the poles with the electrodes during an electrical application.

Robertson's Pupil. A failure of the pupil to react to light without any perceptible impairment of its normal reaction during the accommodation of vision for near objects.

Rolando's Fissure. The fissure which separates the frontal lobe from the parietal lobe of the cerebrum (Fig. 4).

Round Bundle. The so-called "respiratory bundle" of Krause and the "trineural bundle" of Spitzka. A bundle of fibres within the medulla probably associated with ninth, tenth, and eleventh nerves.

Sömmering's Yellow Spot. The point where most distinct vision is afforded by the retina, situated about two lines to the outer side of the entrance of the optic nerve.

Saltatory Spasms. A dancing or hopping of the body, caused by uncontrollable spasms of the muscles of the lower limbs, back, neck, or upper limbs.

Sclerosis. A morbid condition dependent upon an increase in the connective-tissue elements of an organ or other structures.

Speech Tract. A bundle of fibres which serves to join the cortical coördinating centre of speech (Broca's centre) with those nuclei of the medulla oblongata that preside over the various movements associated with articulate speech (Fig. 24).

Spider-cells of Deiters. A form of cell which belong to the connective-tissue formation. (Neuroglia.)

Spinal Segment. A disk of the cord, with a pair of the spinal nerves attached to it,—one on either side (Fig. 30).

Status Epilepticus. A state characterized by continued epileptic convulsions with scarcely perceptible intermissions.

Strabismus. A condition of abnormal deviation of the visual axes, resulting in habitual diplopia, of which the patient may often be unconscious.

Subjective Symptoms. Any morbid nervous phenomenon of which the patient is himself conscious.

Subependymal. Situated beneath the ependyma,—the lining of the ventricles.

Sulcus. A line of demarcation between convolutions of the cerebral cortex. Less deep and more subject to variations than the so-called cerebral "fissures."

Sylvian Fissure. The fissure containing the middle cerebral artery and separating the parietal from the temporal lobes of the cerebrum (Fig. 4).

Syringomyelia. Cavities within the substance of the spinal cord.

Tabes Dorsalis. Locomotor ataxia. Posterior spinal sclerosis.

Tegmentum Cruris. The posterior or sensory part of the crus cerebri (Fig. 11).

Tetanoid Paraplegia. A type of paraplegia associated with a peculiar rigidity of the muscles and exaggeration of the spinal reflexes.

Tetanus Neonatorum. Infantile tetanus, following umbilical irritation, fæcal stasis, puerperal infection, etc.

Tetany. Paroxysmal tonic muscular spasms. They may be artificially induced by pressure on a nerve-trunk or a main artery. (Trousseau's test.)

Thomsen's Disease. Primary spinal muscular spasm. Attempts at voluntary movements are suddenly hindered or interrupted. A hereditary affection.

Thrombosis. Occlusion of a blood-vessel by a coagulum formed at the seat of occlusion.

Tic-convulsif. Diffuse clonic facial spasm.

Tic-douloureux. Neuralgia of the facial nerve.

Triangular Nucleus. A collection of cells which are structurally related to the fibres of Burdach's columns, and in which they probably end. This nucleus (together with the clavate nucleus) probably gives origin to some of the fibres of the fillet tract (Fig. 12).

Trineural Bundle. A term applied to the round bundle of the medulla by Spitzka. See *Round Bundle.*

Trochlear. Relating to a pulley. The superior oblique muscle of the eye is so called because it works through a pulley.

Turck's Column. The so-called "direct pyramidal column" of the spinal cord (Fig. 29).

Will-tract. A term employed by Spitzka as synonymous with the pyramidal tracts. See *Pyramidal Tracts*, and Fig. 12.

Word-blindness. A loss of ability to properly interpret ordinary sight-perceptions,—chiefly the meaning of printed and written characters. It must not be confounded with actual blindness.

Word-deafness. A loss of ability to properly interpret ordinary sound-perceptions, such as the meaning of spoken language, etc. It must not be confounded with deafness, where sounds are not perceived on account of some defect in the apparatus of audition.

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