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BRAIN.

PARTS I. & II., 1897.

Original Articles and Clinical Cases.

HEREDITY AND NEUROSIS.

(*Being the Presidential Address to the Neurological Society for* 1897.)

BY GEO. H. SAVAGE, M.D., F.R.C.P.

GENTLEMEN, by your request I find myself in the most honourable position of President of the Neurological Society, and I feel in accepting this post and before entering upon my first duty, which is the giving of the Address, that I must refer to the death of Dr. Hack Tuke, which has thus placed me as the representative of the Psychiatric Branch of this Society. Dr. Hack Tuke was one of the best examples of the transmission of mental and moral characteristics from father to son. Coming of an earnest methodical family he developed, notwithstanding bad health, a power of work which was astonishing to all his friends. A seeker after truth, and a careful recorder of all he saw, he is hardly to be replaced in our branch of medicine, where the material so far exceeds the means for observing and arranging it. He has left us and I will do my best to fill his place, though I fear his mantle has not fallen on me.

The Address of last year was so masterly a demonstration of the work done by its author in the definite lines of anatomical research, that I feel my task in handling the

indefinite and little known must make my address a poor contrast to his, but my life's work has been with those expressions of nerve power which, though disordered, may yet be made as instructive to the medical observer as are the decay and denudations of rocks to the geologist. I cannot hope to bring before you any brilliant generalisations or any new discovery. The brain anatomist and physiologist are employed in examining the basis of mental factors, and therefore we alienists must be content to wait for their work before we can hope to do more than collect, arrange and speculate.

After much consideration, I thought that my special experience might best be utilised by comparing the relationships of the neuroses, and by tracing, as far as I am able, the lineal descent of these disorders of mental function: hence "Heredity and Neurosis" is the subject I have chosen.

The subject is an old one, but with each step in advance in science old subjects are found to have new aspects. Things have as many characteristics as the observer has points of observation; there is no finality, there is no definite or ultimate knowledge. With the development of the Darwinian idea and with the full recognition of the force of evolution, immense strides have been made in all branches of human knowledge, but the goal is not yet reached. More recently Weismann has called special attention to the problems of heredity, and still later Romanes and Archibald Reid, besides innumerable foreign authors, have taken up the subject. The present time is one rather for collecting and weighing evidence than for forming a final judgment, therefore I find myself in a natural but unsatisfactory state of doubt, and I can only give my experience and my opinions, which must after all be only provisional.

Before proceeding further I will lay down briefly my belief in the part played by heredity in the development of normal and abnormal man.

We take it for granted that evolution has taken place, and we believe that it is still in progress, and that the chief factors in evolution are selection or survival and heredity. Survival of the fittest, though in the past so important a

factor, is of little value in the present phase of human development, and whether there has been marked continuous development and advance in man is still a moot point. It is quite certain that advance in the higher animals must be very slow. We watch for the appearance on the earth of new species of animals and we watch in vain, and specific continuity and invariability are arguments against any visible evolution going on at present. In human intellect we have no real evidence of advance in quantity for many ages. From the lowest savage to the highest civilised being, doubtless, there is a great advance, but even here the increase in bulk of brain is not what some might expect it to be. Men advance and are advancing, not in the mass, but in the adaptability of their nervous systems. It is the surrounding conditions which are growing, and the work of the nervous system is to adapt itself to these rapidly changing conditions. The theory at present popular is that of Weismann, which theory is, that there is a continuity, an immortality of germ plasm, and that the only real changes are in the somatic case or surroundings of this substance, or depend directly on this.

The germ plasm is looked upon as quite different and more elemental than the germ cell, it is as it were the nucleus around which the soma grows. That, just as the nucleus takes unto itself matter and forms a germ cell, so the germ plasm as a whole takes to itself a body which is specifically suited to the surrounding of the future animal. The germ plasm is constant in each species and thus it forms the fixed point about which all variations occur and it is in that we must seek for any changes if there is a transmission of acquired qualities. Weismann is supported in his contention that the germ plasm is continuous and unaltered by the fact of specific fixity; that the characteristics of the animal, *i.e.*, the specific ones, do not alter whatever may be the external or internal variations in conditions; varieties may arise depending on these changing surroundings and pass away, but the species is constant. This is apparently true as far as limited periods are concerned, but unless we believe in the individual creation of species we

must accept the evolution of fresh species through a slow accumulation of changes which have been transmitted.

If evolution is true there must have been a gradual change in the parts which slowly aggregate to form the new species, and we have to admit that these changes, whether the result of one form of selection or another, have been sufficiently established to be passed on from parent to child. These characteristics seem to be definitely represented in some way in the reproductive elements. We no longer expect to see the oak tree in miniature in the acorn, nor the chick in the egg, we only look for a power in the seed which will enable it to grow like its parents in harmony with its surroundings. I do not intend here to consider the various theories which have been started to explain the method by which each part of an organism is reproduced in the ovum, for none of these theories are fully accepted, and none will be considered in the practical part of this paper. We accept as facts that the specific characteristics of beings are transmitted directly with a certain power of slight variation, and that the functional correlates of the transmitted organs also reappear in succeeding generations.

No one disputes that there is a direct transmission of the simpler organs of the simplest animals, nor is there any doubt about the transmission of the simpler functions such as the reflexes, and there also seems little doubt that there is a direct transmission of the parts concerned in the instincts, whether these instincts are congenitally active or only develop later with the enlarging of the surroundings. There is, therefore, little difficulty in comprehending how still more complex organic relations may be passed on, but when the connection between the organ and its function is less clear and definite, it is only natural that the mode of transmission should be more difficult to follow. It is now more fully recognised that there is no such thing as the inheritance of a fully developed function of the higher kind; the tissues and the organs reappear in succeeding generations, and from them the function may be evoked. The faculty of speech may be inherited, but the special language comes from the surrounding. Aptitude with the hands is passed on,

but the special form which the aptitude may take depends on the education. A predisposion is a transmitted, not a developed quality. It is noteworthy that among the functions which are transmitted in association with certain organs there may be temporary delay in their exhibition, and there may be a potential function in reserve which may be kept in abeyance for long periods, and yet may appear when the suitable stimulus is given. This will be seen later to have an important bearing upon certain nervous disorders, which appear only at certain stages of life, when in fact certain conditions act as the stimuli. For instance, the duckling may, in the absence of water, walk and fly and not swim, yet when the stimulus of water is present the highly evolved function of swimming is performed with exactitude and endurance.

I shall throughout take it for granted that there is no such thing as the inheritance of a function, but only the inheritance of the capacity or predisposition to react in certain definite ways under certain definite stimuli. The definite organ responds to definite stimuli, and the changed surroundings do not alter the specific character of the being.

When considering the neuroses it will be seen that the surrounding does cause their FORM to vary. This only means that in the neuroses the differences are usually not specific; they must be considered only as varieties, not species.

There is admitted to be a capacity for passing from parent to offspring special ability to react to certain stimuli, but the question which is being most debated at present is, whether it is possible to transmit directly anything which has been acquired by the individual during his lifetime. The general answer to this question is in the negative, but I think this reply has been given too definitely. For although there is no doubt that the great majority of mutilations are not inherited, yet it does not follow, because most of these coarse material effects are not passed on, that some other acquisitions or defects of a different nature may not be so transmitted.

I have already said if there is no power to vary, and no

power to transmit the power to vary, there can be no progress, no fresh definite varieties and certainly no new species. In most living things there is a recognised power to vary, though not in the mass itself yet in its relations. There is a tendency at first, connected with the survival of the fittest to vary more and more in harmony with the changing surrounding. The power to vary and the power to transmit the power to vary, is all that is required, and I think whatever may have been one's earlier ideas, one must now accept the theory that with the higher developing surroundings it is absolutely necessary for the good of the individual that a more rapid power of reacting to the varying surroundings must be transmitted. This is the point where the inheritance of neurosis comes in. If it is accepted that with developing complexity of life there is necessarily developing complexity of relations, with a corresponding tendency to go wrong, and if there is a transmission of capacity for proper self-adjustment, so may there equally be the opposite.

Having considered the bearings of heredity, I wish to define the use I make of the terms "neurotic" and "neurosis." With development, the relationships between the *self* and the *non-self* become more and complicated, and the means of communication between the two have to be more delicate, and more unstable. The movements of a gale of wind are easily noted by a wind gauge but to mark the movements of waves of light you need an extremely delicate mechanism. The finest human adjustments can only be made by extremely mobile tissues. These delicately mobile tissues are chemically and mechanically unstable, and are sometimes too delicate for their work. Disorder may depend, not on the fineness of the organ, but rather on the force of the excitant, and it will be at once recognised that in studying nervous disorder these two conditions have to be both well weighed.

The nervous systems which are too delicate for their surroundings, and which, as a consequence, tend to be constantly upset in various ways, are called "neurotic," and the instability itself is "neurosis." It will thus be seen that

I do not mean by the term neurosis a definite disease, only a more or less definite tendency, and in my opinion it is this tendency which may be transmitted.

The connection between the so-called "insanities" and the neuroses must be studied in detail. In many cases the same nervous weakness leads to insanity on the one hand, or neurosis on the other, the result varying with the surrounding conditions of the individual and the exciting causes of the disease. Nervous diseases are not properly studied by dividing them too definitely into classes or groups; Nature is not rigid and definite in the making of living things. I trust the experimenters in psychology will be able to give us useful knowledge in reference to the "reaction times" and acuity of sense perception in the neurotic, for though there seems little ground, according to them, for believing in *types* or temperaments, yet I am inclined to think that in some neurotics, at least, there will be found some common physical sensory disorder or defect.

The neurotic individual is the natural outcome of highly specialised modes of living, and we shall find among the unstable two distinct groups—those who react very rapidly and delicately to their surroundings, and are looked upon as geniuses; and others who are unstable without being brilliant, that is, who react destructively.

The next statement I would make is that the neuroses are allied, and that this is so, will be seen in the fact that similar persons, under similar conditions, break down in different ways, *i.e.*, exhibit the different forms of neurosis; also that the same person, from similar causes, at different times, may exhibit the various forms of neurosis; and I hope to show that there is a hereditary relationship, so that in the transmission of neurosis variation in form often occurs. Much still remains to be done in tracing the causes of difference between the neuroses, and there is a like difficulty in explaining the physical origin of the nervous instability. This latter may chiefly depend on increase of bodily refinement due to habits of life, climate, and food, or it may arise from delicacy or weakness due to exhaustion or degeneracy in the parents. One is in the habit of talking of the chain

of neuroses; the simile is wanting in complete exactness, for though there are many links of this chain they are hardly to be considered as forming any connected whole. The links are of different sizes and shapes, and, though forged out of the same metal, they greatly differ. The linking is best seen by noting what may be the possible development of the neurotic type in one family. In such there may be deficient *power* or mass of brain, associated with idiocy; there may be a tendency to unequal one-sided mental development, which may be associated with precocity on one side and moral defect and criminality on the other. There may be defects of *control*, leading on the one hand to convulsions, and on the other to chorea, or to impulsive acts; there may be tendency to irregular discharge of nerve force, this leading to epilepsy, insanity of critical periods of life, and to recurrent forms of insanity. There may also be morbid forms of mental association and morbid sensory states which lead to organised systems of delusions. Besides all these, which may be seen in the same family and which may represent one another in the same person at different times, there are many other forms of nervous disorder which I shall consider in detail as I proceed. Among these other conditions we shall have to note the bodily diseases which often have a nervous aspect, or which may replace nervous diseases—such are asthma, gout, megrim, and diabetes.

Seeing, then, the varieties of nervous disorder which may be represented in a neurotic family, the study of the starting point of neurosis is all-important in the present relationship. This nervous instability, I have no doubt, depends in the majority of cases upon some bodily defect or weakness in the parent. My belief is that in these cases there is a peculiarity existing in the parent which leads to the instability in the offspring—the tendency to decay in the parent showing itself in weakness in the child. The germ plasm may be continuous, but it must vary directly in some ways with the health of the body. The germ plasm of the young animal must be taken to have more initial vigour than that of the decaying one, and the physical state resulting from bodily illness must affect the whole body. There is no possibility of one organ saying to another, "I have no need of thee."

I have seen many examples of the children of old age being neurotic, whereas previous ones were healthy; and also I have been able to trace a connection between the children begotten soon after a serious illness and those begotten before it. I have clear evidence of the instability of some children born after the parent has had an attack of insanity, the children born before the attack having been stable enough.

To put my experience briefly: neurosis or unstable nervous disposition depends in many cases on disease, or causes of bodily weakness in the parent. It is noteworthy that the conditions of nervous weakness leading to transmission of neurosis do not always exist in the insane themselves, but that an insane parent may beget or bear a normal child. The old notion that all insanity was likely to be passed on is not correct. There are very well-marked examples in which insane mothers have had several normal children, though they were insane during the whole pregnancy and after these children were born.

My belief is that physical, rather than nervous instability, is transmitted; this does not, however, cover the whole field, for in certain families, for generation after generation, the nervous balance alone seems to be very unstable, the members of these families having plenty of vitality.

The physical tendency to neurosis is easily transmitted, and in some cases a direct constitutional tendency to break down under special stresses is transmitted. Neurosis is not, as a rule, started by consanguinity alone, but if there is a tendency to nervous weakness, this is increased greatly by intermarriage.

Though an insane parent may have a normal child, there is no doubt that the offspring of the insane show their weakness in various ways, and it is here that we see so well marked the transmission of the tendency, as contrasted with the transmission of the disease.

As a rule, the children of the insane and of certain classes of neurotics resemble one another. These children may have no deficiency of brain mass, and no evidence of initial lack of any faculty, but they are *restless;* they seem to react

unduly to all external and internal stimuli, so that there is no surplus of nervous power, no storage; memory is defective or absent, and there is no power to advance from experience. These children vary greatly in the degree of their restlessness, and also as to the time when it becomes manifest; the earlier this restlessness is marked the greater the risk of mental defect in the child.

The same defect of power of control of nervous force is seen in the various kinds of impulsive acts of these neurotic children. In some, "*rages*," or nocturnal terrors, represent undue reaction to outside stimuli. The stimuli, in some cases, are started from without the body, but in others from within, there being subjective sensory impressions or visceral impressions which, neglected by the normal child, may in the neurotic start various exaggerated reflexes. Interestingly related to these children are those who have an extreme dread of heights. Many persons, otherwise normal, have giddiness or dread on looking down great heights, but in my experience the offspring of the neurotic suffer much from this dread. Uncontrolled or exaggerated motor reflexes are seen in these children, also mimicry of a monkey-like type, and it is among such that we meet with the musical mimic, the weak-minded child with a phenomenal memory, and the calculating idiots. Such children not only react abnormally to outward stimuli, but convulsions are easily caused in them.

A skin eruption, a dental trouble, or gastro-intestinal irritation may set up fits. The occurrence of infantile convulsions does not mark every child as neurotic, but it is a very important element in the diagnoses. These convulsions occurring in neurotics may readily lead to a *habit* which becomes epilepsy, each recurring convulsion making the next more easily produced. Neurotic children exhibit various interesting disorders of expression, some of which need further study. Facial twitches are common, proceeding in some cases from mimicry, but in others from ill direction of nervous energy. Certain forms of stammering, too, are common in neurotics. Before leaving these cases, I should like to mention certain other defects of expression

(or peculiarities, I might say). In some there is a missing link between one of the sensory centres and the highest centres: thus, some cannot learn by sight, and others cannot learn by hearing, the former defect being the more common. I have seen half a dozen young patients who could not be taught to spell; they saw no relationship between alphabetical signs and sounds. Again, in studying mirror writing, I have met with idiots who did it at once when set to copy with their left hands, and I would go one step further and say that the neurotic learn to do this much more quickly than the normal; in this case, rapid power of adapting to changing conditions may mark a connection between the genius and the fool.

Defect of control seen in the neurotic may extend beyond the senses and the muscles, &c. In children belonging to nervous stock, the temperature, which is generally variable in childhood, is more erratic, the slightest bodily disorder being followed by rapid and sudden rise in temperature, which falls equally quickly. Allied to the instability of temperature in the neurotic is the facility with which delirium is started. The offspring of neurotic parents show their physical and mental instability in no way better than the ease with which they pass into delirious states. A very slight illness will be associated with nocturnal delirium, and with the simpler fevers of childhood, such as measles and the like, delirium will be present. I think this is of double interest in comparison with the easy production of convulsions under similar conditions, and the tendency in these patients later on, say at adolescence, to develop grave *delirium* or delirious mania, just as epilepsy may be the result of the convulsions.

I have met with one family in which one member had convulsions leading to epilepsy; another member died of delirious mania after measles; a third developed systematised delusional insanity, the links in the neurotic chain being easily traced to their origin, namely, a neurotic father.

The general idea that *youth* is in favour of the patient is markedly a mistake; with the neurotic, the younger the patient the greater the danger to the individual, the dangers

being either of permanent loss of power, or of permanent instability.

I hardly know where to place somnambulism in my series of derived neuroses, for, though it differs in origin in different patients, yet there is no doubt it is very common in the children of the insane and the neurotic. In some it is started during the restless state when there is undue reaction to outward stimuli; in others, it is associated with dreams, or with nocturnal hallucinations of a less pronounced type than nocturnal terrors. I have known it arise in restless, irritable children from vesical irritability. In one case which I saw recently, I have no doubt that unrecognised dreams played a chief part; for this girl recollected dreams occasionally, which were always of exactly the same type, so that the fright and reaction certainly depended on dreams in some cases, and, I believe, the similar acts were always, in this case, started by similar mental states. Automatic states will be seen to be linked with convulsive and epileptic conditions. Exaggerated irritability leads to another series of troubles—the constant wetting of the bed, and the development of precocious sexuality; in both instances I have been able to trace reflex acts as starting these morbid habits.

The descendants of neurotic parents exhibit their nervous weakness in instability of various kinds, but they also show very clearly want of power, and as might be expected in the evolution of the civilised states, the finest adjustments will be seen to fail first. In the individual the first sign of weakness as well as the first sign of decay may be a lack of adaptability; senility is often marked by rigidity and want of plasticity, and with those decaying or degenerating through defective heredity the same may be seen. In some cases the neurotic is too unstable to have definite power of adapting himself to his surroundings, and I wish now to consider such who fall under the description of the morally insane.

These classes now under consideration form the connecting links between the criminal and the insane, and a few examples will best convey my meaning. In some there is

either a want of memory or lack of power of comparing past experiences with present stimuli. Moral idiocy, moral imbecility, and moral insanity are all forms, or rather degrees, of the same thing, and it is again worthy of note that just as a previous attack of insanity may produce moral insanity in the individual, so previous insanity in the parent may leave moral insanity in the children. Of the moral defects which occur as symptoms, the most common are lying, theft, cruelty, destructiveness, and increasing precocious or perverted sexuality.

Lying may be an exaggeration of the common childish habit of "make believe." I have met with most astonishing romancers among the children of the neurotic. In these children there seemed no power to separate the imagined from the experienced, and the lying was rather a genius than a fault. On the other hand, the neurotic liar is generally malignant and cunning, and I have known such, who on the spur of the moment could invent the most elaborate lie which was hard to disprove. I daresay lying is not uncommon in its various forms among normal children, but it is necessary to consider it here in relationship to the abnormal. *Stealing* may be associated with lying (it generally is), or it may be met with alone. It again is of two markedly different types. It may be a kind of uncontrolled reflex, the mere taking anything which falls in the way of the child, or may be allied to the collectors' habit; it may be quite simple in the restless child, but may be developed by cunning through injudicious punishment. In some, punishment has no effect either in developing or checking the habit, there being either inability to remember, or what is more likely, to associate the act of stealing with the punishment. In some cases there is what one patient described to me as "lust of possession," there being recurring periods of possessive desire which might be compared to the recurring periods of sexual desire. These cases might be considered as cases of morbid impulse. The cases of destructiveness and cruelty, again, resemble those already considered in being of two classes, in the one the mere reflex desire to act leading to the destruction, the sight of fire

being quite enough to induce some of these unstable children to throw their most cherished toy into the flames. In other cases there is the exaggerated pleasure of power; this I fear, is not uncommon in the ordinary school boy, and may do little or no harm, or it may occur early in exaggerated degree in the neurotic child. The subject of sexual precocity is probably one of the most difficult to discuss fairly, yet I feel that to do it justice it is one which needs a paper to itself, and for this I am not here prepared, I have no doubt that the children of the neurotic have often very strong sexual passion developed prematurely. It is always hard to gauge the statements of sexual perverts; they are almost always anxious, like their connexions the hysterics, to make a parade of their symptoms, while they apologise for their weakness by lying, or by transferring the blame to others. I have evidence, however, that sexual self-abuse may arise in infants from local irritability—that an adherent prepuce may start a vicious habit, or that worms in the rectum, or some bladder trouble may similarly give rise to a habit which is hard to cure. This may occur in the healthy child, but is rarely so started in such. There is doubtless a very great danger to neurotic children in relation to the sexual functions, and here again we meet with the fact that similar symptoms of defective control over the desires may also occur as the result of previous attacks of insanity or of senile nervous decay. If I had to plead specially for the possibility of the direct transfer of mental symptoms from parent to child, I should certainly select cases which have been under my observation in which immoral parents have had immoral children, but in these cases, as in the alcoholic ones—in which, too, the transference of habits seems to be clear—we have many other factors to consider which show that the heredity is not clearly proved. Sexual precocity is to my mind one of the most common symptoms of neurotic origin; I have not very much experience of sexual inversion, but I believe that in these cases heredity is also supposed to play a very important part.

Already I have spent a very large amount of time on the subject of the forms of the neurosis which are met with in

the offspring of the nervous and insane; not that I expect to be able to point out the absolute physical tendency to transmission, but chiefly with the purpose of showing how a neurotic tendency shows itself in the offspring in various ways—that, in fact, there is only a transfer of a tendency which may develop in different ways according to the surroundings of the individual. Having done this, I shall now pass to other branches of my subject.

To complete the chain, heredity and the neuroses, it is necessary to refer to certain relationships which exist between bodily disease and neuroses. I cannot speak on this subject from any large personal experience, but I am inclined to think that hereditary nervous instability leads to a special predisposition to contract some contagious diseases. Sir W. Jenner pointed out that in some families there is a remarkable susceptibility; he specially referred to our royal family and its proclivity for typhoid fever. I believe that members of highly neurotic families not only take such diseases as scarlet fever, measles, typhoid, &c., readily, but that they are more liable than others to have a second attack of the same fever. If my idea is borne out and found to be true, it is only another example of the transference of a *predisposition* to unstable vitality. Micro-organisms of a dangerous nature are probably very generally present, but fortunately the soil required for their growth is not so common. I believe, however, that the neurotic subjects provide a fertile soil. If this be true of fevers, the same may hold good with phthisis. It is worth noting that this disease is commonly met with in the insane, and I believe it is still admitted that the tendency to develop phthisis is readily transmitted. Nowadays no one looks for the direct inheritance of either consumption or of cancer, but they recognise that certain families seem to be more liable to them than others. My opinion was, until quite recently, that no connection could be traced between cancer and the neuroses, but I feel now that my judgment should be reserved until I have observed further.

Next, as to the transmission of insanity, as such, from parent to child. I must make it clear that in my opinion

there are certain neurotic conditions which are more easily passed on than others. It does not follow that every neurotic must become insane, in fact there are certain forms of neurosis, such as hypochondriasis, which rarely pass beyond the border line of sanity. I think it will be best for me now to consider the part which heredity seems to play in some of the *forms* of mental disorder which are commonly recognised, not as species of disease, but as convenient groupings of symptoms. Much has been written which I have not time to discuss as to the special power of transmission of insanity which belongs to one sex or the other; doubtless the laws underlying the transmission of bodily and mental characteristics have to be studied much longer before any general rules can be formulated.

One-third of the insane are said to have neurotic heredity, but here we at once meet with a difficulty, for this makes all kinds of mental disorder of equal value in transmission. This is not so, and though I cannot pretend to weigh the exact value of each form of mental disorder as to its power of being passed on, I shall give an approximate value to each.

Mania.—In my experience, some forms of maniacal excitement are very likely to occur in the neurotic subjects, but ordinary (emotional) mania is not one of these forms. Hysteria of a grave type (with various forms of hysterical paralysis, refusal of food, malingering and the like), occurs in such persons, yet the development of these symptoms in these patients into organised mania is not common. On the other hand, I believe that neurotic subjects are specially liable to acute delirious mania, from comparatively slight causes; thus just as the neurotic child may readily pass into delirium, the neurotic adult or adolescent will easily pass into the higher grade of the same disease, *i.e.*, delirious mania. In these cases the cause is often some moral shock, some fright or sudden grief, a disappointment or an illness, which last may have started with fever and passed gradually into delirium and to acute delirious mania. It is interesting that this form of mental disorder which, in my opinion, is so frequently met with in highly neurotic families, is

characterised by extreme physical alterations, in fact the tendency is to rapid dissolution, the basis of mind being in a very unstable state.

Melancholia.—I find it much more easy, as a rule, to trace a melancholic inheritance in a family than I do to trace any other form of family neurosis, and this is interesting, for it is with melancholia that one is most accustomed to find a co-related disorder of the bodily functions. With mania you may have every bodily function apparently in order, but in melancholia you generally find all the vital processes imperfectly performed. It is, therefore, not very surprising that a bodily tendency which has a mental aspect should often be transmitted. I might go further and say that commonly many bodily ailments tend to be represented by morbid self-consciousness of a depressed or melancholic type. Visceral action when unconsciously performed is healthy action; unhealthily performed it tends to cause gloom. There is, therefore, a natural relationship between the transmission of certain unhealthy bodily conditions, and a melancholic interpretation of the morbid feelings.

I have already pointed out that in the passing on of instinct it is common to see a potentiality transmitted which may never be called into action by the surroundings; and so it is in certain families. Melancholia occurs in the members of these families when they attain advanced age, or when they are by disease brought to a premature old age. I have known such families in which, generation after generation, the members pass into melancholia, though the lives of the individuals in many instances have varied widely; some being active, some passive, some having borne the burden and heat of the day, while others rested in the shade of easy and happy circumstances, yet one and all have broken down similarly in old age. In some of the families where there has been conspicuous ability in managing affairs and in the steady pursuit of literary distinction, the end has been, in one generation after another, a melancholic shadowing of the great mental powers. I think it right also to point out here that in some of these families the characteristic instability of the neurotic is well marked.

The brilliant ones have even during their times of work periodically to pass into the *wilderness* like the prophet of old. There are periods of deep depression alternating with periods of restless energy, and in many cases these persons end in chronic mental depression; such persons often transmit similar tendencies to recurring times of brightness and despair.

Dementia.—I need say little on this subject, but I must briefly refer to it. The natural tendency of all mental disorders is supposed to be dementia. To accept this as true one has to modify the term dementia, or use the expression "forms of mental weakness," which includes not only defect of faculties but defect of mental stability weak-mindedness and chronic and recurrent insanities being placed together. The tendency of insanities, then, is to dementia, but this is more evident in some instances than in others. The neurotic individual, though liable to insanity, is more likely to recover once or more often. Instability means tendency to go wrong, but power to be set right. The neurotic, by inheritance, then, tends to recurrent insanities with a greater chance of some recoveries, and to ultimate weak-mindedness or permanent instability. I have seen a fair number of cases in which the family tendency was always to fail first in their highest mental abilities and then slowly to pass into permanent and general dementia. Here the bodily health is often maintained to a great age, and we have the difficult task of explaining the relationship between the transmission of a healthy body with a mind tending to decay or to disorder. All I can say is that such cases are not infrequent. In dementia the lines of degeneration may follow those of development, and in generation after generation similar lines of decay may be followed. It is noteworthy that in such families one meets with tendency to apoplexy, senile epilepsy, &c., or to some visceral degeneration such as Bright's disease; one member of the family dying of one and another of some other of the diseases named.

The next subject I wish to refer to is systematised Delusional insanity, Monomania, &c. These groups are to

my mind the most interesting of all classes of insanity, and need much careful study and some generalisation to place the chaotic mass of observed facts in some sort of order. I would say the chief characteristics of the disorder are the frequency of a history of insanity in the progenitors of the sufferers, and the constancy with which most of the mental functions, apart from the delusion which dominates the mental life, remain unimpaired. It is well to recognise that in some cases of delusional insanity there is no history of insanity in the parents, but there is history or previous mental illness in the patient himself, thus once more showing the import of insanity in the individual, and its relationship to hereditarily derived neurosis.

Among the chief characteristics of the neurotic are instability or susceptibility to the influence of surroundings and the facility of forming habits, and in delusional insanity it is these characteristics which are most marked. I have met with much difficulty in explaining the apparently direct transmission in some of these cases of imperative ideas.

From the time of Darwin it has been noticed that trivial acts performed by a parent may apparently, without education, re-appear in the child—that in fact a bodily something may be transmitted which has a special mode of expression which will be transferred. The trifling, and one might say useless, variations which may occur in one generation after another may be seen in the extra digit and certain types of nose or mouth peculiar to some families. I have seen several patients whose history I have been able to examine carefully, in whom mental tricks have been transmitted from one generation to another. In one case a daughter was brought to me with the *folie de doute* and the *folie de toucher*, and I heard her father had been subject to both these peculiarities for many years. In one such case I was told the parent and child had been separated from the earliest years.

The power of transmission of muscular tricks and other peculiarities is, I think, beyond dispute. Though it is very difficult to exclude all risk of imitation, as for example, with

the history of the transmission of special musical ability, I have met with "musical prodigies," the children of musical parents, but have never been able to decide how much was direct inheritance and how much the result of imitation; but allowing that much was due to the latter, yet there seemed to be a transmission of a greatly increased aptitude or tendency which is all one is contending for. Among the inherited tricks I should place imperative ideas which may follow neurotic heredity or neurotic illness. I have not any statistical evidence as to the inheritance of the special senses; we all recognise that deafness or shortness of sight may occur in parents and children. I am also inclined to think that I have evidence of transmission of unusual acuity of the sensory perceptions, and it is possible that with special nervous constitutions and special sensory acuity there may readily occur systematised delusional insanity. I have described elsewhere certain forms of this disorder which depend on deafness, and, therefore, I am prepared to meet with similar forms of organised delusion depending on other congenital or acquired sensory defects. Cases of delusional insanity are chiefly characterised by suspicion, doubt, ideas of plots, persecutions and the like, and there are frequently hallucinations of the senses. The memory and other faculties may be quite normal; there is little, or perhaps one should say, only a slight tendency to dementia, though there is great tendency to chronicity. I believe few of the sufferers from these organised forms of mental disorder have been free from strongly neurotic heredity, that they have been the children of old or degenerate parents, or have themselves had other attacks of acute insanity. The time at my disposal will only allow me to make this bald statement, of fact and not to enter into any more interesting points in relation to the direct or indirect transmission of sensory mental traits.

Certain other hereditary relationships deserve notice, but I fear I can only mention them in rapidly passing to my conclusion. We recognise the transmission of a tendency to develop gout, and we recognise that the disease produced by the individual himself differs little from that which may have been inherited. We recognise that

such a disease as gout may re-appear in the next generation in another form, and what is more, it may assume a neurotic character, gout replacing insanity, or insanity gout. This is not only true of gout, but of many other bodily diseases.

There is, then, a solidarity in the human body with its functions, which makes it almost a fault to speak of neurosis as a thing apart from bodily states and conditions.

I have endeavoured to trace the links existing between the bodily states and the mental expressions, and so I must leave the subject by summing up shortly as to my belief in the force of heredity and the influence of the developing surroundings in the production of nervous instability.

I have pointed out the links in the neurotic chain as seen in the offspring of the insane and of the highly neurotic.

I have shown how similar mental states may result from neurosis in a parent or from neurosis in the individual.

I have pointed out my experience as to the relative danger of transmission of the various forms of insanity; and now, gentlemen, I have completed my task, I leave it with regret, knowing how imperfectly I have performed it. Yet I leave it with the hope that, at least, I have shown you that I do not look upon heredity as a tyrant which, with inflexible power, drives the offspring to destruction. I do not think because the fathers have eaten sour grapes that all the children should have their teeth set on edge. I have striven to show that, though there is, in my opinion, power to transmit acquired peculiarities, yet the tendency is to transmit a predisposition which may be modified by surroundings.

That, in fact, it is not with the Fates we have to struggle, but with flexible powers, this justifies our treatment and encourages us to hope that we are doing our service to mankind with a prospect of doing good; hopefully we may proceed in the quest of truth, not trusting to attaining perfect truth, but having the pleasure of the quest which, like the search for the Grail, will hallow our work and consecrate our lives.

ON HUNTINGTON'S CHOREA.

BY J. MICHELL CLARKE, M.A., M.D., F.R.C.P.

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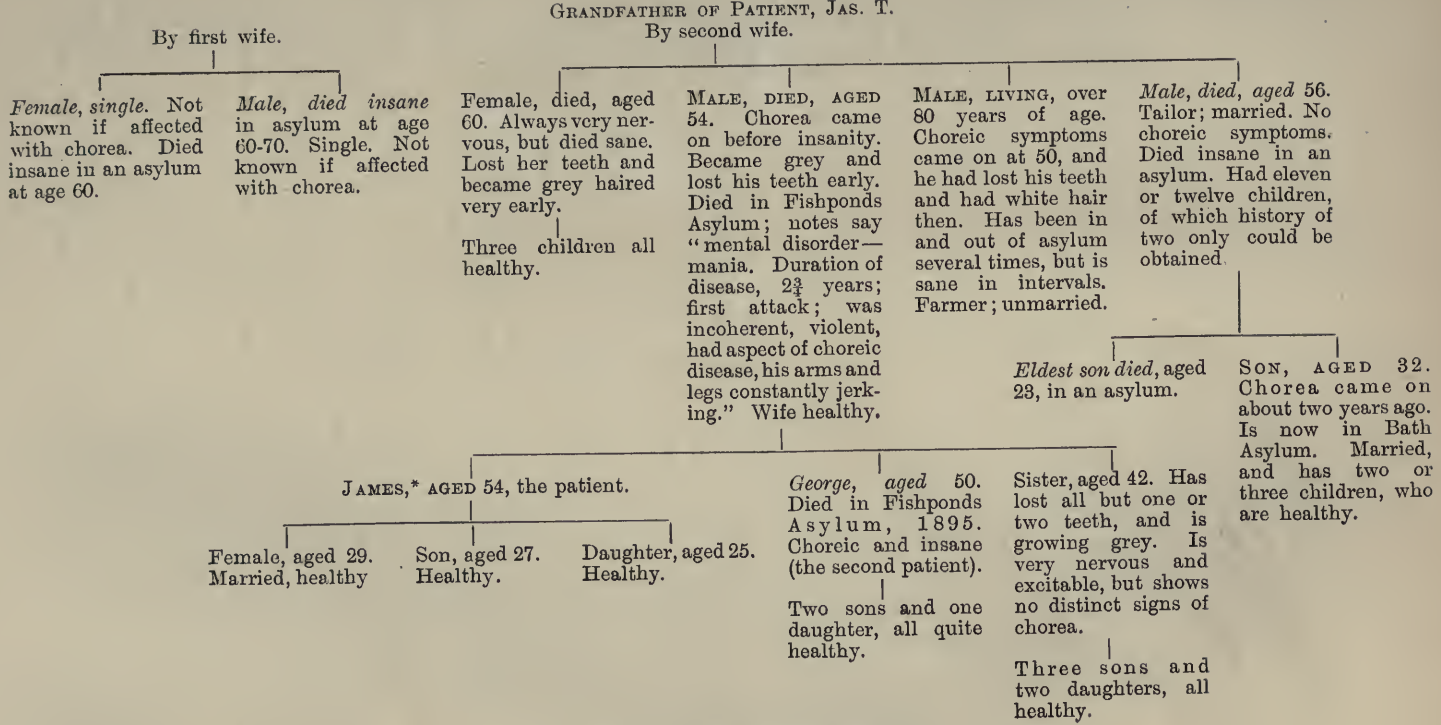
THIS form of Chorea, characterised by its appearing first in adult or late adult life, its tendency to affect several members of a family, to be inherited, and to end in insanity, is now well-known, but cases are rarely met with in ordinary practice, and there have been few opportunities for *post-mortem* investigation of the morbid changes.

James T., a painter, aged 54, was admitted into the Bristol General Hospital for chorea and weakness, in September, 1895. The family history is given in the accompanying scheme. We could get no further back than the patient's grandfather, and no very definite account of him. The disease, however, appears to have come into the family through him, and not through either of his wives. On the male side there is also a history of rheumatism running through the family, and in both males and females a tendency to premature senility as shown by early greyness of the hair, and loss of the teeth is noticeable. Both the patients seen had the arcus senilis and well-marked degeneration of the arteries.

The patient was, like his brother, a small, thin man, looking older than his years, with marked arcus senilis and thickened, tortuous arteries. He had always worked hard, lived a regular and temperate life, had had no venereal disease, nor any bad illness. There was no history of any fright or great mental emotion, nor of any kind of fit or apoplectiform attack. There was no evidence of any past or present lead-poisoning. According to his wife the first symptom noticed was involuntarily stamping of the right foot five years previously; two or three months later, twitchings came on in the right and then in the left arm; soon all four limbs were affected. The patient himself said that he first noticed the twitchings on waking up one morning five years previously, and that they had persisted ever since,



* A twin brother died when two days old.

gradually growing worse. During the last six months he had been less intelligent, his memory had been failing, and his manner was changed. For two nights before he was admitted his wife had to sit up all night with him, as he was in a very excited state, looking wild, and talking constantly and incoherently. After admission he was quite quiet.

State on Admission.—Abdominal and thoracic viscera normal. No cardiac murmurs; heart sounds being normal. Urine normal. Constant choreic movements affect all the muscles of the trunk and limbs; walking is difficult on account of them, but though the knees are often suddenly flexed, the spasms are not strong enough to throw him down. The facial muscles are affected, the patient making grimaces from time to time; the tongue can be protruded, but is jerked backwards and forwards. The ocular muscles are unaffected. During voluntary movement the chorea becomes worse, and the hands and arms especially are agitated by spasmodic jerking movements when he tries to do anything with them. He can, however, feed himself if his food is cut up for him. Though voluntary actions are thus interfered with they are not distinctly ataxic. Muscular power rather weak; dynamometer, 22 kilos., both for right and left hands. The muscles are small but show no wasting. Electrical reactions normal. The choreiform movements are constant during the waking hours, but cease entirely when he is asleep.

Eyes.—Pupils equal, of normal size, react well to light and accommodation. There was marked error of refraction; on ophthalmoscopic examination the right optic disc was pale, with mottling of choroid on nasal side (Guttate choroiditis). Left optic disc not so pale, choroidal changes were the same as in the right eye.

Superficial reflexes active, a well-marked "goose-skin reflex" is obtained over the skin of the chest and abdomen. Knee-jerks normal; no ankle clonus; front tap contraction present; no muscular rigidity. Sensation to touch and pain normal, and power of localising sensations fairly good, but very slightly deficient over the hands. No affection of smell, taste or hearing. The functions of micturition and defæcation are natural. His face is somewhat expressionless, and he does not succeed in frowning or expressing surprise. He answers simple questions well, but if they are at all complicated he answers irrationally. He often repeats his statements. He has no delusions or hallucinations. His memory is defective and inaccurate. He sleeps

well and has a good appetite. When in bed he can move his feet and legs well, and to any desired position, and there is no loss of the sense of position. The muscular sense appeared to be quite normal. He improved in nutrition during his stay in the hospital, but otherwise his condition was unchanged. He was given liq. arsenicalis in gradually increasing doses.

The younger brother of the above patient, George T., was in the Bristol Lunatic Asylum at the same time. I am greatly indebted to Dr. J. V. Blachford, Assistant Medical Officer to the Asylum, for notes of the case and of the *post-mortem* examination, and for the opportunity of seeing the patient and making the microscopical examination of the central nervous system. He was a small, thin, grey-haired man, who had for several years suffered from choreic movements affecting the limbs and trunk. He had never had syphilis. The diaphragm was unaffected, and he had no difficulty in swallowing. For three months previous to admission into the Asylum on July 1, 1893, he was irritable and dangerous, and for twelve months his memory had been failing. He was demented, and had the delusion that the attendants were keeping back money that had been sent to him, but apart from this delusion he was quiet, and showed some amount of intelligence and power of memory. He answered questions as to his past life readily and correctly. The thoracic and abdominal viscera were normal; the heart being unaffected. The skin showed a condition of mild ichthyosis, best marked over the abdomen and thighs. The muscles though small showed no decided wasting. The knee-jerks were somewhat exaggerated, but there was no ankle-clonus nor muscular rigidity. The pupils were equal and reacted well, the ocular movements were unaffected by spasm. He protruded his tongue spasmodically, and after much effort, and he walked with some difficulty on account of the muscular spasm. It is unnecessary to describe the choreiform movements, which were constant, and affected the muscles of the face, trunk and limbs, as they were precisely similar to those above described in the elder brother's case. They ceased entirely during sleep. In both cases the spasmodic movements were exactly like those of ordinary chorea. This patient died in the Asylum, after a few days' illness, of pneumonia, in November, 1895. The *post-mortem examination* was made twenty-seven hours after death by Dr. J. V. Blachford. There was lordosis of the lumbar spine. The *skull cap* showed a slight degree of transparency all over with some thinning at bregma. Pacchionian depressions well-marked. Diploë dense and scanty.

Measurements:

Average thickness		45 cm.
Diameters (Internal)	Antero-posterior	17 cm.
	Transverse ...	13 cm.
Circumferences ...	Antero-posterior	31·2 cm.
	Transverse ...	31·2 cm.
Horizontal		52 cm.

Except for some injection of the pia-arachnoid the membranes were normal, and the pia-arachnoid stripped easily, and was not adherent to the cortex. The gyri appeared normal. The grey matter over the motor area was well differentiated into a light intermediate band between two darker bands. The ventricles contained some excess of cerebro-spinal fluid. No abnormal changes were visible to the naked eye in the basal ganglia, pons, medulla, cerebellum, or spinal cord.

The whole brain weighed 44 oz., the pons, medulla and cerebellum, 6 oz. The xiphoid cartilage of the sternum was bifid. The *right lung* weighed 44½ oz.; the lower lobe was in a state of red hepatization, portions sinking in water, the rest of the lung was intensely congested and œdematous. The *left lung* presented some fibrous contractions at the apex; there was some general congestion and œdema. The pleural cavities were obliterated by general adhesions. The other thoracic and abdominal viscera presented no morbid changes. The brain and spinal cord were hardened in Müller's fluid.

The following table gives the average of numerous measurements of the grey matter taken from the convexity of the convolutions in the several lobes of both cerebral hemispheres, after hardening about three months in Müller's fluid, together with those of a case reported by Dr. Charles L. Dana, and of the normal grey matter as given by him in his paper.[1]

The measurements are in millimetres.

	This case		Dana's case	Normal
Frontal lobe ...	... 2·50		2·85	3·1
Motor convolutions ...	2·56	Superior central ...	2·2	2·75
		Parietal lobule ...	2·2	
		Third central ...	1·9	
		Lower central ...	2·2	3·0
Temporal lobe ...	2·75		3·2 and 3·1	3·1
Occipital lobe ...	2·58		2·1	2·6 to 2·5

[1] *Journal of Nervous and Mental Disease*, vol. xx., p. 577.

Sections of the fresh cerebral cortex were cut and stained by Bevan Lewis's method; and of the hardened brain from all parts of the cerebral cortex and from the cerebellum, after embedding in celloidin, were stained in hæmatoxylin and eosin, methylene blue, aniline blue-black, hæmatoxylin and picro-fuchsin (Van Giesen), and by Weigert's method.

Pia-mater.—Vessels full, especially over the frontal and motor convolutions, their walls were, as a rule, normal, but, in a few instances, slightly thickened.

Convolutions of frontal lobe; vessels prominent, their walls healthy, and the perivascular spaces distended; in superficial layers of the cortex there were a few microscopic hæmorrhagic extravasations. The layer of small granule cells was narrow and stained badly. The small pyramidal cells showed morbid changes, many of them appearing shrunken, others deeply pigmented, their nuclei obscure and processes stunted. The large pyramidal cells and branched spindle or multipolar cells of the fifth layer appeared healthy.

In the motor convolutions the vessel-walls were healthy, but here and there a few red blood cells had escaped from the vessels. In a very few places the perivascular sheaths contained numerous leucocytes, which were passing into surrounding tissues. The small pyramidal cells showed the same changes as in the frontal region; a few of the large pyramidal cells contained much pigment obscuring the nucleus, but they were mostly normal. Cells of fifth layer normal. In both frontal and motor regions the number of small branched glia or interstitial cells seemed to be excessive.

In the occipital and temporo-sphenoidal lobes similar microscopic changes, but in less degree, were observed; in the former the glia cells were especially conspicuous, and in a few places wandering cells were present in the pericellular spaces around the nerve cells. Sections of the cerebellum, prepared by the above methods, showed injection of the small vessels of the cerebellar cortex, but otherwise presented no abnormal changes. The fibres in the white core of the convolutions, the cells of Purkinje and of the granule layer, appeared healthy. Unfortunately the nitrate of silver process was for some reason, a failure in the cerebellum.

Sections were also prepared according to Dr. Berkeley's modification of Golgi's nitrate of silver method (BRAIN, vol. xviii., p. 473). This gave good results, with the drawback that in some cases in cutting the sections the cell processes became broken across by the microtome knife, and also, like all the nitrate

of silver processes, the staining is very capricious, only a proportion of the cells being stained; larger or smaller, as the case may be. Further, we do not know whether normal or abnormal cells best take the stain; so that so far as proportion of normal to abnormal cells is concerned, no conclusions can be drawn from the sections, but it was quite evident that there were a large number of morbid cells, and this stain also brought out the fact, observed in sections stained by the other methods, that the change is a partial one, healthy cells lying side by side with diseased ones. The most superficial layer of the cortex was healthy, the second layer stained very badly in all sections, and appeared narrow; those cells that were stained mostly appeared normal. The small pyramidal cells, as in the other sections, showed the most marked changes, many being shrunken, irregular in shape, their processes stunted with small nodose swellings upon them, and disappearance of the little "gemmulæ" in the affected processes. No such swellings were seen, however, upon the axis cylinder processes, when these were visible. The large pyramidal cells, and the cells of the fifth layer, showed much less marked changes, being, where stained, with some few exceptions, healthy. The nerve fibres of the white and grey matter and collaterals stained well and appeared healthy, both by the nitrate of silver and by Weigert's method. The glia cells were conspicuous in all parts of the cortex, especially in the frontal and motor regions, from their great number and large size, and appeared in many instances to be attached to pyramidal nerve cells, lying in immediate proximity to them. The occipital cortex was also rich in large, fine and richly-branched glia cells in its deep layer and subjacent stratum of white matter. In the parts of the cortex most affected by the morbid changes, the processes of the glia cells showed little nodose enlargements upon them, which I have not before observed. The pons and medulla appeared to be healthy. Spinal cord: grey matter in all parts normal; nerve cells in all parts healthy. The pia matter was thickened, and there was a slight increase of the processes running into the cord from it, and of the neuroglia throughout the cord. No system-degenerations were present, the white matter being otherwise healthy.

It should have been mentioned above that the fibres in the white centre of the cerebral convolutions appeared healthy, the vessels in parts, but not universally, much injected. The glia cells were conspicuous and abundant throughout all sections. No further changes were noted in the white matter.

To sum up, the morbid change consisted in a widespread but partial degeneration of the cells of the cerebral cortex, especially the cells of the second and third layer, most marked in the frontal and motor convolutions, together with an increased amount of interstitial tissue and number of neuroglia cells.

In this family males have been chiefly affected. It will be noticed that the first patient showed evidence of mental affection; and that in the case of the sons of the patient's youngest uncle, both insanity and chorea have come on at an earlier age than in the others. According to Diller and Suckling, the disease comes at an earlier age in each generation, but this is by no means always the case. Wharton Sinkler (*Journal of Nervous and Mental Disease*, 1889) states that the disease may skip a generation.

Another case of this disease was under my care in the Bristol Hospital in 1893. In this patient, a compositor, 46 years of age, the family history could not be fully obtained, but so far as could be ascertained, the tendency to insanity was less marked. His father was affected with choreiform movements, which came on two or three years before his death at 42 years of age; he was a temperate man, and was killed by falling from the roof of a house; the spasmodic affection of the muscles was said to have been the cause of the fall. The patient had two brothers and two sisters alive and well; two other sisters are affected with choreiform movements, one of the latter is in the workhouse, and is said to be crazy: her children are mentally defective, the Board-school teachers finding it impossible to teach them. There is no other case of insanity in the family. The patient himself used to drink at one time, but had been a teetotaller for twelve years. The illness began with loss of power in the legs, then the twitchings came on, and soon extended to all parts of the body. He also suffered from dyspepsia, and occasionally from attacks of frequent micturition. He was grey, poorly nourished, and looked ten years older than his age. The thoracic and abdominal organs were healthy. Heart of normal size, and the sounds normal. His face was rather expressionless; he spoke slowly and indistinctly, with a

somewhat slurred utterance. Intelligence defective; memory very bad, and inaccurate; he showed a marked want of power of sustained attention, and answered questions badly, wandering off to other subjects.

The muscular system generally was only moderately well developed; there was no wasting, the muscles being well nourished, but flabby and weak. No rigidity, nor excessive muscular irritability to percussion. The dynamometer gave 21 kilos. right, and the same left hand. No fibrillary tremor. No paralysis. The muscles of head, neck, arms, and legs, and to less extent those of trunk, were agitated by constant spasms, in their general characters exactly resembling those of ordinary chorea, but rather more sudden and shock-like. These choreiform movements ceased during sleep, were aggravated by movement, or by talking; when lying down his head was occasionally suddenly raised up from the pillow. The movements interfere with walking, and have caused him to fall down. The facial muscles are affected, and those of the eyelids, but not of the eyeball. He protrudes the tongue well, and has no difficulty in swallowing. The respiratory movements were occasionally noted to be a little irregular. There is no in-cordination of movement.

The special senses were unaffected; sensation of all forms was normal, except that there was some loss of the temperature sense over the feet. Knee-jerks normal. Superficial reflexes; cremasteric and right abdominal not obtained, others present, plantar exaggerated. Pupils normal, equal in size, react well to light and accommodation. The temperature was constantly sub-normal, 97° to 97·5°. The bowels acted regularly.

This patient's daughter, aged 14, was said by him to be suffering from the same disease; but I saw her, and found that she was affected with diplegia and athetosis, most marked on the right side of the body, and the result of injury to the brain during birth. This bears out an observation in the *Lancet* (December 21, 1895), "that this is almost invariably the actual condition in cases of so-called congenital chorea, so that the term is probably a misnomer, and the movements would be more correctly described as those of athetosis."

In a paper by Dr. E. S. Reynolds (*Med. Chron.*, vol. xvi., p. 21) will be found an excellent account of several cases, with references to the history and literature of the subject.

Important contributions to the study of the pathological anatomy of the disease have been made recently. Menzies (*Jour. Mental Science*, October, 1892, and January, 1893) describes two families: in the first, 25 persons out of 100 traced were affected, the average age of onset was 27·6 years, and of death 43·7 years. In the second family: 13 persons out of 74 suffered, the average age of onset being 37·2 years, and of death 54 years. He concludes that the disease may descend from either parent to either sex, but that males are more commonly affected.

The *post-mortem* changes in the brain were slight coarseness of neuroglia, thickening of vessels, cell-degeneration, and, in hardened specimens, a few spider cells; in the cord, a diminution in the cells of Clarke's column, slight sclerosis of the ascending antero-lateral tract in all parts, and sclerosis of the posterior columns in the cervical region.

Oppenheim (*Centralb. f. innere Medicin*, 1894, p. 918) reports two cases: (1) a man who died of apoplexy, aged 75, in whom the disease had lasted sixteen years; and (2) a woman, who died of influenza, aged 56, in whom the duration was five years. There was a history of heredity, but I find no mention of insanity. *Post-mortem*, the heart was healthy in each case. In the brain the gyri were narrowed, and the sulci broadened in the motor, parietal and occipital regions; in these regions there were foci of hæmorrhagic infiltration in recent, fibrillar in later, stages in the sub-cortical layer. In the cortex, the small round cells next below the uppermost layer were deficient in number. The pyramidal (? large) cells were normal. The basal ganglia were normal. In the cord there was increase in the neuroglia cells and fibres, chiefly in the lateral columns; these changes he does not regard as secondary to the cerebral lesions, as they are not systematic, and further, affect only the neuroglia and vessels. The peripheral nerves examined were found degenerated; and this he attributes in the first case to old age, in the second to influenza. He thinks the essential morbid con-

dition to be a miliary disseminated encephalitis, cortical and sub-cortical, followed by atrophy of the cortex.

Dr. Charles L. Dana's (*Jour. of Nervous and Mental Disease*, vol. xx., p. 565) patient was a man in whom symptoms first appeared at the age of 33, and who died of typhoid fever at 37. The case constituted a transmission through females to the fifth generation. The convolutions of the brain showed anomalies in an interruption of the fissure of Rolando, and absence of the superior "*pli de passage.*" There was a general thinning of the grey matter, most marked over the central convolutions ; areas of cell-degeneration, the angular and small pyramidal layers being most affected, the cell-defect being primary, the vascular changes secondary. He considers the disease belongs to teratology, being an innate defect in cell structure.

Kronthal and Kalischer (Virchow's *Archiv.*, Bd., cxxxix., Abs. in *Neurol. Centralb.*, May 15, 1895), on the basis of the examination of three cases of chronic hereditary progressive chorea, conclude that the morbid process in Huntington's chorea consists in diffuse, rarely circumscribed, changes in the cortex of the brain ; these are essentially disease of the vessel walls, increase of nuclei, cell accumulations, small hæmorrhages, and increase of the interstitial framework ; whilst the nervous elements are only slightly affected.

There seems then to be no doubt that the cerebral cortex, especially of the motor convolutions, is the seat of disease ; the chief difference of opinion being as to whether the primary change is in the nerve cells themselves or in the supporting tissue. So far as my own sections go they point to a degeneration of the nerve cells, with a concomitant increase of the neuroglia.

Changes of a similar nature to those above described, but less marked, have been observed in cases of severe chorea of the ordinary type. Severe cases of ordinary chorea occasionally end in mania. In a girl, who suffered from an acute attack of extremely severe chorea, which became complicated with acute mania, and who died from exhaustion, I found, *post-mortem*, intense congestion of the small arteries and capillaries of the cerebral cortex. The bright injection

of the vessels was obvious to the naked eye, and, under the microscope, sections of the cortex showed injected capillaries in extraordinary abundance, with some minute hæmorrhagic extravasations and cell-exudation, most marked over the motor area. The vessels of the basal ganglia and pons were also injected, but in much less degree.

The identical character of the spasms in cases of Huntington's chorea, and of ordinary chorea, points to affection of the same part or parts of the central nervous system in both cases. The severity, long duration and intractability of the symptoms in the former should render the recognition of the underlying lesion more easy, because the morbid changes would naturally be more extensive. The *post-mortem* changes above described indicate the cerebral cortex, especially of the motor convolutions, as the seat of the morbid change. The conclusion seems to be that all forms of chorea are due to disturbance in this region of the brain, although in each form the exciting cause of this disturbance is probably of a different nature, and the precise nature of the resulting alterations and capability of recovery vary accordingly.

In conclusion, I must express my best thanks to Mr. James Taylor for his kindness in making many excellent photographs of my sections, and to my clinical clerk, Mr. C. P. Mackie, for taking much trouble in investigating the history of the family above recorded.

EXPLANATION OF PHOTOGRAPHS.

FIG. 1.—Frontal cortex, low-power magnification; shows nearly whole thickness.

Degenerated and healthy cells in second and third layers; there are several degenerated cells to the left of the figure. Glia cells.

FIG. 2.—Motor cortex, low power. The surface of cortex lies to the left and above.

In the pyramidal cells of the second and third layers, some are healthy, others swollen, rounded, and irregular in outline. In uppermost part of figure is seen a glia cell attached to degenerating small pyramidal cell (*a*); above this, again, is a healthy pyramidal cell.

(In some cases the main process of the cell is artificially separated with a sharp fracture by the microtome knife.)

FIG. 3.—Motor cortex, high power. Degenerating pyramidal cells; the one (*a*) on the left hand shows swellings on main processes, and absence of "gemmulæ," and the cell body is deformed. (The process itself shows fractures caused by the knife.)

FIG. 4.—Motor cortex, high power. One or two healthy and other degenerate pyramidal cells. (Processes of some cells have again been broken in cutting.) (*a*) Healthy cell with neuron.

FIG. 5.—Motor cortex, high power. Glia cells, with small nodosities on their branches.

FIG. 6.—Occipital cortex, high power. Group of large, richly-branched glia cells in subcortical layer.

NOTE.—*The first two photographs should be viewed with a hand-lens.*

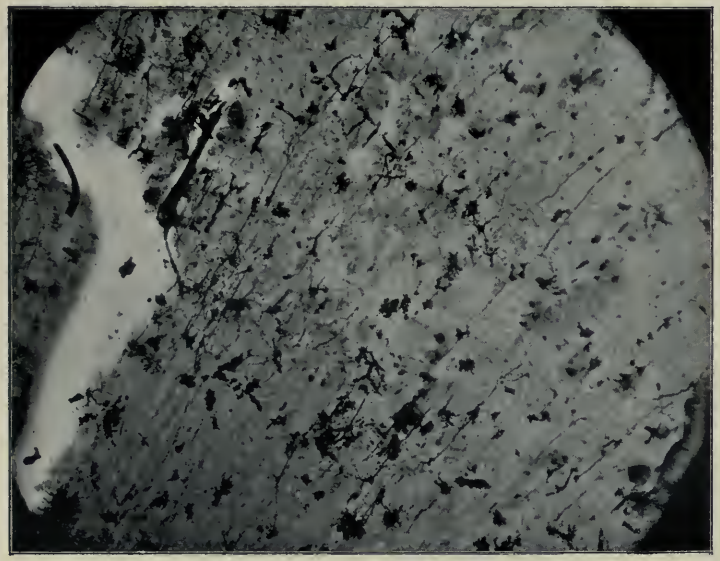


FIG. 1.

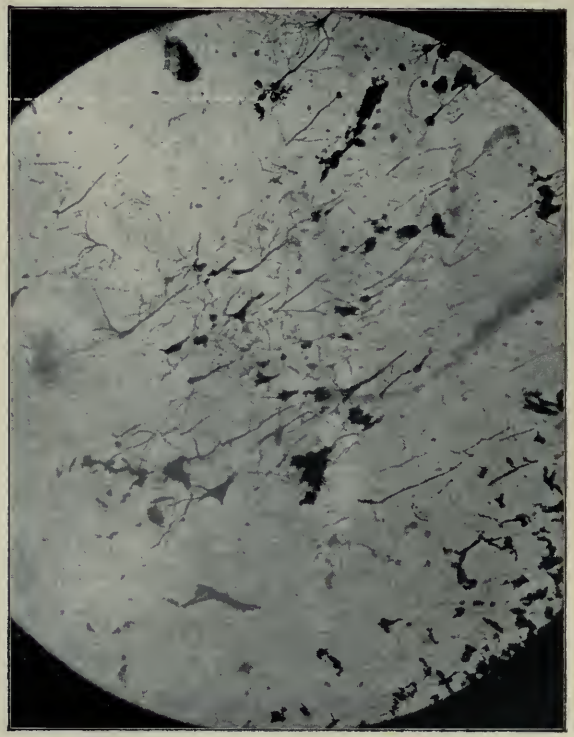


FIG. 2.

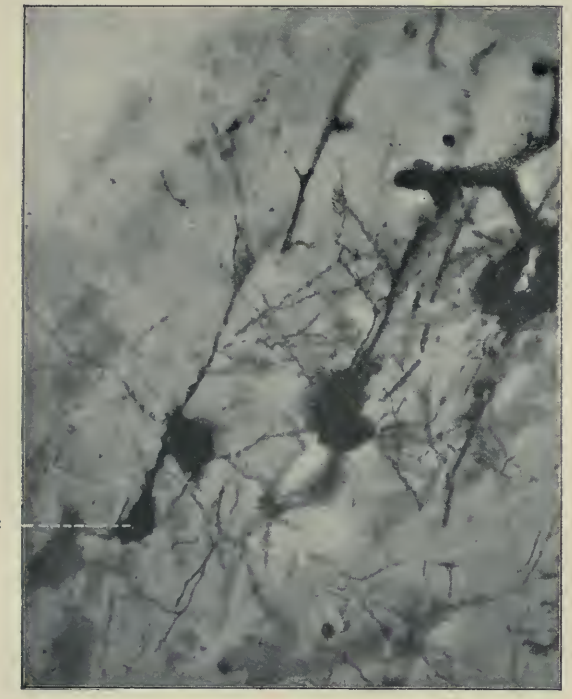


FIG. 3.

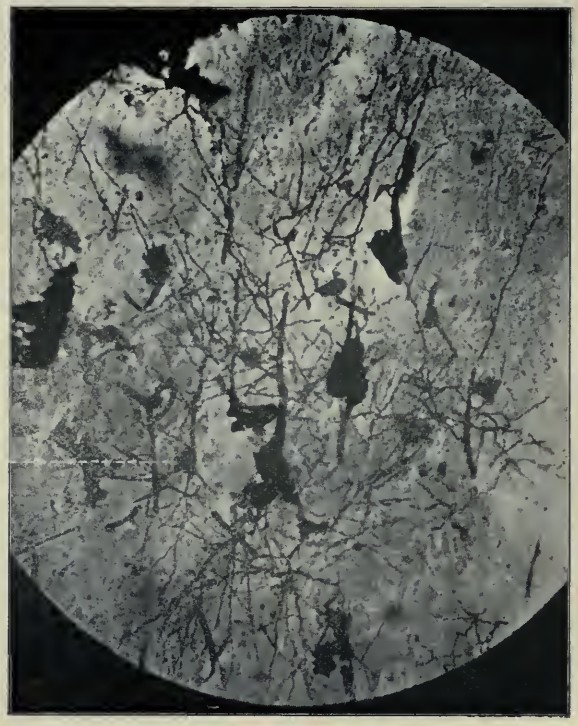


FIG. 4.

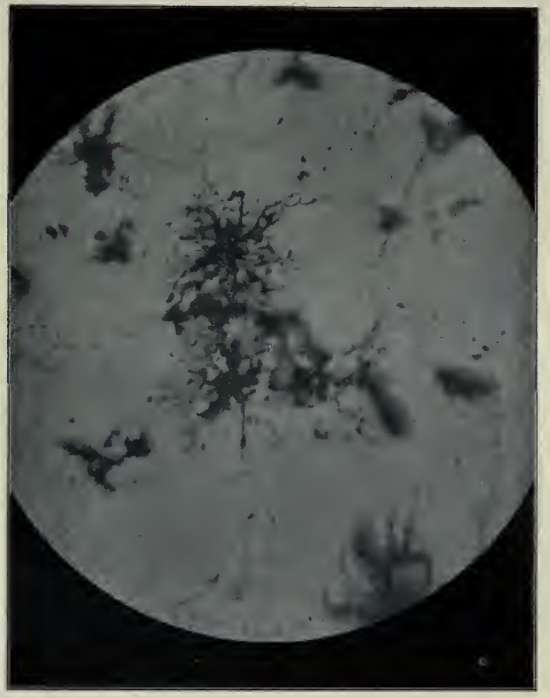


FIG. 5.

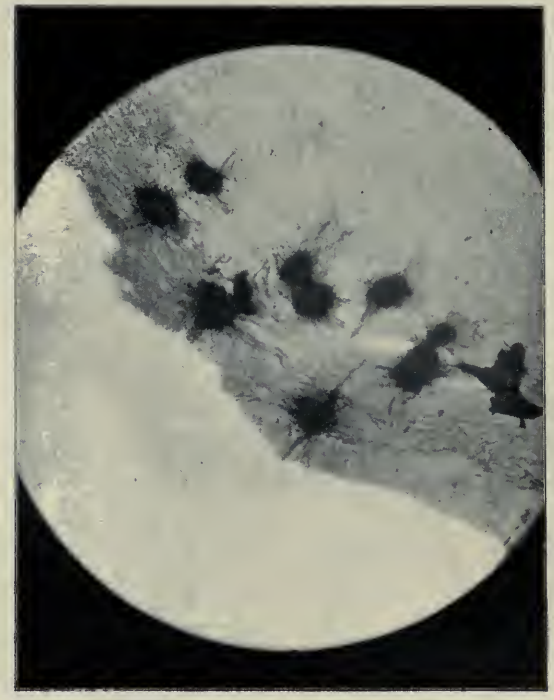


FIG. 6.

AN EXPERIMENTAL INVESTIGATION OF THE CERVICAL AND THORACIC NERVE ROOTS IN RELATION TO THE SUBJECT OF WRY-NECK.

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Introduction.

I am indebted to Professor Victor Horsley for allowing me to carry out the majority of the experiments on which this paper is based in the pathological laboratory of University College, London, when it was under his directorship. I have also to thank Professor Vaughan Harley for enabling me to perform a few supplementary experiments in the Laboratory for Pathological Chemistry. To Dr. Max Lowenthal my sincere thanks are due for his kind assistance in some of the latter experiments; his help was especially valuable to me when I was checking the results which I obtained in connection with the anterior neck muscles.

There is an abundance of evidence to show that in a large proportion of cases of spasmodic wry-neck, surgical measures, such as division of muscles and stretching of the spinal accessory nerve, are useless, and that there must be few cases in which even excision of a portion of the spinal accessory nerve can be expected to lead to permanently good results, in that, in the majority of cases which come under observation, muscles beyond the control of this nerve take part in the spasm. Further, in that the sterno-mastoid has a double nerve supply, the spinal accessory and the posterior branches of certain cervical spinal nerve roots, even in those cases in which the spasm appears to be limited to this

muscle, it scarcely seems reasonable to suppose that severance of only one of its paths of connection with the central nervous system is likely to do more than lessen the spasm in the muscle, while another path by which impulses can reach it from the nerve centres is still intact. Complete section of both of these paths of connection with the central nervous system can alone be expected to result in complete arrest of spasm in the muscle. If this be true with regard to the cases in which the sterno-mastoid alone appears to be involved, how much more unlikely is it that any such surgical measures directed to the spinal accessory nerve can influence the spasm in muscles over which this nerve has no control, and which are obviously involved in so many cases of wry-neck.

The only surgical measure which offers a reasonable prospect of permanent relief in such cases is division (with excision of portions) of the posterior branches of the upper cervical spinal nerve roots, an operation devised by Gardner in Melbourne and Keen in America, and which has been practised by Horsley in this country. This being the case, it seems of no small importance that we should accurately determine by experiment: (1) The exact number of nerve roots, electrical excitation of which results in contraction of muscles which bring about movements of the head on the trunk; (2) the precise position in which the head is placed by contraction of the group of muscles supplied by each individual nerve root; (3) in how many roots a given muscle is represented; and (4) the muscles represented in each nerve root.

The necessity for such an investigation will be obvious if the results which I have obtained in the present research are compared with the statements made in the standard text books on anatomy in this and other countries; and, in order to emphasize this, I have drawn up two tables in which my results are compared with the statements made in the last edition of Quain's "Anatomy."

While many observers have investigated the nerve roots which innervate the limbs, with a view to ascertaining the movements and muscles represented in each root as far as I

am aware, no similar attempt has been made to determine the movements or muscles represented in the upper cervical roots; roots concerned with the movements of the head on the trunk. The only exception to this statement is to be found in an investigation carried out by Beevor and Horsley,[1] which had as its object, the determination of the function of certain cranial nerves, in addition to which the three upper cervical nerve roots were stimulated with a view to ascertain the root supply of the sterno-hyoid, sterno-thyroid, and omo-hyoid muscles.

The experiments on which the present paper is based have been conducted in monkeys, *Macacus rhesus* chiefly, and also a few *Macacus sinicus*. The animals were always anæsthetised by means of ether administered by inhalation; they were kept under the influence of this anæsthetic throughout the course of the experiment, and killed by an overdose of it at its termination. During the operative procedure necessary to expose the cervical spines and neural arches, great care was taken to keep to the middle line as far as possible, and not to sever more of the attachments of the muscles than was absolutely necessary to allow the nerve roots to be satisfactorily exposed in the neural canal by removal of the cervical spines and arches. After effecting an entrance into the neural canal the dura mater was opened, and all the posterior roots of the cervical series were divided on one side, together with a varying number of the thoracic posterior roots, in different experiments, and also the roots of the spinal accessory nerve.

Owing to the fact that the intra neural portions of the upper cervical motor roots are so short, they were left intact for excitation, and were not divided, except for purposes of control in some instances. As this plan is open to the objection that many of the results obtained might be due to spread of current along the cord, &c., to roots other than that excited at any given instant, every possible means was taken to avoid error from this source by comparing the results of excitation of contiguous roots, of intermediate portions of

[1] Beevor and Horsley, "Proc. Roy. Soc.," 1888, vol. 44, p. 269.

the spinal cord, of the dura mater contiguous to the root excited, and so on. But, of course, the most reliable control experiments were those in which these motor roots were severed from the spinal cord before their peripheral ends were stimulated.

Excitation was effected by fine, closely approximated platinum electrodes attached to the secondary coil of a Du Bois Raymond's inductorium, supplied by a single bichromate cell. The strength of current employed was 30 or less, as measured on Kronecker's scale, which corresponds to an interval of about 27 c.m. between the primary and secondary coils. Such a current was always sufficient to evoke a well-marked response, and did not cause any trouble by spreading to parts other than the root to which the electrodes were actually applied. In all the experiments the first point investigated was the position assumed by the head on excitation of the motor portions of the individual cervical and thoracic roots. The investigation was further conducted so that a given root was excited in some instances and all the muscles which responded were noted, while in other instances a given muscle was kept under observation, while each nerve root of the series investigated was in turn excited, and a note made of those, stimulation of which evoked contraction in the muscle.

Movements of the Head resulting on Excitation of the Anterior Spinal Nerve Roots.

Despite the fact that the precise nerve roots in which a given muscle was represented varied somewhat in different animals, the position assumed by the head on excitation of the different roots remained wonderfully constant, exceedingly slight shades of difference being occasionally detectable in some animals as compared with others, but the predominant movement was always the same for any given root in all the animals investigated. The cervical roots further showed a wonderfully constant paired arrangement as to function, as far as the head movements were concerned, the first two being responsible for a very similar movement, whilst the third

and fourth were responsible for a movement which differed from that observed in the first two roots, but agreed closely with each other ; the fifth and sixth caused little movement of the head on excitation, while the seventh and eighth caused a more decided movement, the character of which was very similar in the two roots.

First Cervical Root.—The movement characteristic of an excitation of this root is a well-marked lateral inclination of the head on to the shoulder of the side excited, so that the side of the head and face are approximated to the shoulder. This is a comparatively pure lateral flexion movement, in which there is next to no backward movement, or rotation of the head about the vertical axis of the body, the chin, however, pointing to the opposite side from that stimulated. (See fig. 1.)[1]

Second Cervical Root.—Excitation of this root produces a movement of the head closely resembling that which resulted on excitation of the first root, *i.e.*, a lateral inclination of the head to the side stimulated, by which means the side of the head and face are approximated to the shoulder. As a rule, there is added to this a slight backward movement of the head to the side stimulated, which is accompanied by the slightest possible rotation of the head on its vertical axis, and the chin inclines to the opposite side to that of the root excited. (See fig. 1.)

Third Cervical Root.—Whereas the lateral movement of the head, by which the side of the head and face are approximated, is the predominant movement represented in the first and second roots, the third root is responsible for a movement in which, though the lateral movement is still evident, the movement which predominates is distinctly one in which the head is drawn backwards, and, the occiput being drawn to the side excited, the chin is directed upwards and to the opposite side. (See fig. 2.)

Fourth Cervical Root.—All that has been said with regard to the movements represented in the third root is applicable to the root now under consideration, the only difference

[1] The figures are from instantaneous photographs taken for me by Dr. Worrall at the moment of excitation of the second and fourth roots respectively.

between the two roots being that in the fourth there is still less of the lateral inclination of the head so characteristic of the first and second roots, while the backward movement of the head, in which the occiput is drawn to the side stimulated, is still more pronounced than in the third root. (See fig. 2.)

Fifth Cervical Root.—In striking contrast to the marked movements of the head noted in connection with excitation of the upper four cervical nerve roots is the exceedingly feeble movement of the head which results on excitation of this root. The little movement that results consists in a slight drawing of the occiput backwards, and to the side stimulated. The degree of this movement varies more than that of the movements represented in the other roots, sometimes being very indistinct and at other times leaving no question in the mind of the observer as to its existence.

Sixth Cervical Root.—The movement represented in this root corresponds, in all respects, to that represented in the fifth, and calls for no further comment than that already offered in connection with the fifth root.

Seventh Cervical Root.—While much less than the movement produced on excitation of the first four cervical roots, the movement which results on excitation of this root is decidedly more pronounced than that observed in connection with the fifth and sixth roots. It consists in a drawing of the occiput slightly backwards to the side stimulated, and is obviously a secondary, or indirect, movement caused by the pull exerted on those muscles attached to the head and scapula, by the vigorous contraction of the latissimus dorsi, which pulls the scapula downwards.

Eighth Cervical Root.—Very similar in all its details is the movement of the head produced by stimulation of the motor portion of this root. As in the case of the seventh, movement of the head appeared to be entirely one indirectly induced by the vigorous drawing down of the scapula by the latissimus dorsi.

First Thoracic Root.—The movement of the head obtained on excitation of this root depends largely on whether the latissimus dorsi is represented in it or not; when it is so

Fig. 1.

Fig. 2.

Fig. 1.—This figure is from an instantaneous photograph taken for me by Dr. E. S. Worrall at the moment of excitation of the second cervical motor nerve root on the left side, and shows the lateral inclination of the head to the side stimulated, so that the left side of the head and face and left shoulder are approximated; the chin is at the same time directed to the right, without being elevated from the thorax to any degree.

Fig. 2.—In this case the instantaneous photograph was taken by Dr. Worrall at the moment of excitation of the fourth cervical motor nerve root on the left side, and shows lateral inclination of the head by which the side of the head and face are approximated on the side stimulated, while the head is drawn back to the left, so that the chin is raised well off the thorax and directed to the right.

the representation is only slight, so that the resulting movement of the head, while resembling that produced by excitation of the seventh and eighth cervical roots, is very much smaller in amount. In those cases in which the muscle has no representation in the first thoracic root, the resulting movement of the head on excitation of this root resembles that about to be described as characteristic of the second and certain other thoracic roots rather than that produced by excitation of the seventh or eighth cervical roots.

Second Thoracic Root.—The movement characteristic of excitation of this root is a lateral curving of the spine by the action of the erector spinæ, and, as a result of this, the head is slightly tilted laterally towards the opposite side. This is clearly a movement of the head brought about indirectly, and in no way due to any direct action of muscles attached to any part of it. The same movement is met with on excitation of the third and other thoracic nerve roots, even as low in the series as the seventh and eighth sometimes. The movement is always only slight, and, as a rule, it becomes exceedingly insignificant by the time these lower roots are reached.

Muscles represented in each Anterior Spinal Nerve Root.

First Cervical Root.—Rectus posticus major and minor, obliquus superior and inferior, trapezius, sterno-cleido-mastoid, sterno-hyoid, sterno-thyroid, omo-hyoid, sometimes complexus and trachelo-mastoid.

Second Cervical Root.—Complexus, splenius, trachelo-mastoid, cervicalis ascendens, trapezius, rectus capitis anticus major, longus colli (upper part), sterno-cleido-mastoid, sterno-hyoid, sterno-thyroid, omo-hyoid.

Third Cervical Root.—Complexus, splenius, trachelo-mastoid, cervicalis ascendens, trapezius, rectus capitis anticus major, longus colli (upper part), levator claviculæ, sometimes sterno-cleido-mastoid and levator anguli scapulæ.

Fourth Cervical Root.—Splenius, cervicalis ascendens, longus colli (middle part), levator claviculæ, levator anguli scapulæ, sometimes complexus, splenius, and tracheleo-mastoid.

Fifth Cervical Root.—Erector spinæ, longus colli (middle part), levator anguli scapulæ, rhomboidei.

Sixth Cervical Root.—Erector spinæ, longus colli (lower part), latissimus dorsi, sometimes levator anguli scapulæ.

Seventh Cervical Root.—Erector spinæ, longus colli (lower part), latissimus dorsi, scaleni.

Eighth Cervical Root.—Erector spinæ, latissimus dorsi, scalenus anticus, medius and posticus.

First Thoracic Root.—Erector spinæ, scalenus anticus, medius and posticus, sometimes latissimus dorsi.

Second Thoracic Root.—Erector spinæ, scalenus anticus, medius and posticus.

Third Thoracic Root.—Erector spinæ, scalenus medius, sometimes scalenus anticus and posticus.

Fourth Thoracic Root.—Erector spinæ, sometimes scalenus medius.

The Nerve Root Supply of the Neck and Back Muscles.

Rectus posticus major, rectus posticus minor, superior oblique, inferior oblique.—These four muscles almost invariably receive their innervation from the first cervical root alone; but in some instances one or other of them receives an additional nerve supply from the second cervical root.

Complexus.—This muscle derives its most constant nerve supply from the second and third cervical roots, and sometimes from the fourth root in addition to these, while in other animals the first cervical is the additional root to supply this muscle.

Splenius.—Like the complexus, the splenius is innervated most constantly from the second and third cervical nerve roots; but it also receives fibres from the fourth cervical nerve root in some cases.

Trachelo-mastoid.—The nerve roots which most commonly supply this muscle are the second and third cervical, but an additional supply is occasionally derived from the first or the fourth cervical roots, and sometimes the supply is from the third and fourth roots alone.

Cervicalis ascendens.—This muscle is supplied by the

second, third and fourth cervical nerve roots, while lower portions of the erector spinæ, including the transversalis cervicis, are innervated from roots aboral to those supplying this muscle and the trachelo-mastoid.

Trapezius.—Few of the muscles investigated had as constant a root supply as the trapezius, which almost invariably responded on excitation of the first, second and third cervical nerve roots.

Rectus capitis anticus major.—This was the only muscle of the group examined, as it was so difficult to obtain a satisfactory view of the rectus minor and oblique. It responded when the second and third cervical roots were excited.

Longus colli.—The upper portion of the longus colli derives its supply from the second and third cervical nerve roots; the middle portion from the fourth and fifth; and the lower portion from the sixth and seventh cervical roots.

Sterno-mastoid.—The most constant innervation of the sterno-mastoid is from the first and second cervical roots, but it is sometimes also supplied by the third cervical.

Sterno-hyoid, Sterno-thyroid, Omo-hyoid.—These three muscles derive their nerve supply from the first and second cervical roots.

Levator claviculæ.—The nerve supply of this muscle is derived from a slightly higher root level than is that of the levator scapulæ, the third and fourth cervical nerve roots being those excitation of which results in contraction of the levator claviculæ.

Levator anguli scapulæ.—The fourth and fifth cervical nerve roots most commonly send fibres to this muscle; but sometimes the third cervical root does so, and in rarer instances the sixth cervical root is responsible for the part supply of this muscle.

Rhomboidei.—The nerve roots excitation of which resulted in contraction of these muscles were, as a rule, the fifth and sixth cervical.

Latissimus dorsi.—This muscle receives its nerve supply from the sixth, seventh and eighth cervical nerve roots; but in some cases the first thoracic root replaces the sixth cervi-

cal, so that the two roots which most constantly innervate this muscle are the seventh and eighth cervical.

Scalenus anticus.—Sometimes excitation of the sixth cervical nerve root resulted in contraction of this muscle; it always responded when the seventh and eighth cervical and first and second thoracic roots were stimulated, and sometimes also from the third thoracic root.

Scalenus medius.—This muscle has the most extensive supply of the group, receiving its innervation from the sixth cervical sometimes, the seventh and eighth cervical and the first, second and third thoracic always, and sometimes from the fourth thoracic.

Scalenus posticus.—The scalenus posticus has the same root representation as the scalenus anticus, receiving its nerve supply from the sixth cervical and third thoracic sometimes, and always from the seventh and eighth cervical and first and second thoracic roots.

The Genio-hyoid Muscle.

As it is stated in Quain's "Anatomy"[1] that the genio-hyoid derives its nerve supply from a branch given off by the hypo-glossal, but consisting of fibres derived from the upper cervical nerves, this muscle was carefully observed in three experiments, while each of the upper cervical nerve-roots was in turn excited; but not the slightest evidence of response could be obtained from the muscle.

The Infra-hyoid Muscles.

My results are in complete accord with those obtained by Beevor and Horsley[2] with regard to the nerve root supply of these muscles; and I am further in a position to corroborate the statement made by them that the sterno-hyoid and sterno-thyroid muscles are chiefly represented in the first cervical root, and the omo-hyoid chiefly in the second cervical root; also that the anterior belly of the omo-hyoid is represented chiefly in the first, while the posterior belly of the muscle is represented chiefly in the second cervical root.

[1] Quain, 10th Ed., vol. ii., part ii., p. 302. [2] *Loc. cit.*

The Diaphragm.

During the course of some of the experiments the nerve-root supply of the diaphragm was investigated by the electrical method. The abdominal cavity was opened under such circumstances, and the muscle observed from below, the liver, stomach and other abdominal viscera being drawn gently down so as to allow of a clear view of this wide expanse of muscle. The nerve-roots, excitation of which resulted in contraction of the diaphragm were the fourth, fifth and sixth cervical; the portion of the muscle nearest the middle line on the side of the root stimulated responded from the fourth root chiefly, while the outermost portion of the circumference of the muscle on the same side contracted chiefly when the sixth root was excited; the intermediate portion of the muscle responded chiefly when the fifth cervical root was stimulated. As far as could be observed the portions of the muscle which responded were limited to the side on which the nerve roots were excited; there was no evidence of bilateral action on unilateral stimulation.

Table of Comparison of my Results with the Statements in the last edition of Quain's "Anatomy."

		Quain.	Author's Results.
Root.		*Muscles.*	*Muscles.*
I. C.	Post.	Inferior oblique Rectus posticus major Rectus posticus minor Superior oblique Complexus	Inferior oblique Rectus posticus major Rectus posticus minor Superior oblique Trapezius
	Ant.	Anterior recti Genio-hyoid Infra-hyoid muscles	——— Sterno-mastoid Infra-hyoid muscles
II. C.	Post.	Complexus Superior oblique (sometimes) Inferior oblique Splenius Trachelo-mastoid	Complexus Trapezius Cervicalis ascendens Splenius Trachelo-mastoid
	Ant.	Rectus anticus major Longus colli Sterno-mastoid Genio-hyoid Infra-hyoid muscles	Rectus anticus major Longus colli Sterno-mastoid ——— Infra-hyoid muscles

		QUAIN.	AUTHOR'S RESULTS.
Root.		*Muscles.*	*Muscles.*
III. C.	Post.	Complexus	Complexus
		Transverso-spinales	(Not observed)
		Splenius	Splenius
		Erector spinæ	Trachelo-mastoid
		———	Cervicalis ascendens
		———	Trapezius
	Ant.	Rectus anticus major	Rectus anticus major
		Longus colli	Longus colli
		Infra-hyoid muscles	———
		Scalenus medius	———
		Levator scapulæ	———
		Sterno-mastoid (sometimes)	———
		Trapezius	———
			Levator claviculæ
IV. C.	Post.	Complexus	———
		Transverso-spinales	(Not observed)
		Splenius	Splenius
		Erector spinæ	Trachelo-mastoid (sometimes)
			Cervicalis ascendens
	Ant.	Rectus anticus major	———
		Longus Colli	Longus colli
		Scalenus medius	———
		Scalenus anticus (sometimes)	———
		Levator scapulæ	Levator scapulæ
		Trapezius	———
		———	Levator claviculæ
V. C.	Post.	Transverso-spinales	(Not observed)
		Erector spinæ	Erector spinæ
	Ant.	Longus colli	Longus colli
		Scaleni	———
		Levator scapulæ	Levator scapulæ
		Rhomboidei	Rhomboidei
VI. C.	Post.	Transverso-spinales	(Not observed)
		Erector spinæ	Erector spinæ
	Ant.	Longus colli	Longus colli
		Scaleni	Scaleni (sometimes)
		———	Rhomboidei
		———	Latissimus dorsi
VII. C.	Post.	Transverso-spinales	(Not observed)
		Erector spinæ	Erector spinæ
	Ant.	Longus colli	Longus colli
		Scalenus medius	Scaleni
		Latissimus dorsi	Latissimus dorsi
VIII. C.	Post.	Transverso-spinales	(Not observed)
		Erector spinæ	Erector spinæ
	Ant.	Longus colli	———
		Latissimus dorsi	Latissimus dorsi
		———	Scaleni

		Quain.		Author's Results.
	Root.		*Muscles.*	*Muscles.*
I. T.	Post.	Transverso-spinales		(Not observed)
		Erector spinæ		Erector spinæ
	Ant.	———		Scaleni
II. T.	Post.	Transverso-spinales		(Not observed)
		Erector spinæ		Erector spinæ
	Ant.	———		Scaleni
III. T.	Post.	Transverso-spinales		(Not observed)
		Erector spinæ		Erector spinæ
	Ant.	———		Scalenus anticus (sometimes)
		———		,, posticus ,,
		———		,, medius ,,
IV. T.	Post.	Transverso-spinales		(Not observed)
		Erector spinæ		Erector spinæ
	Ant.	———		Scalenus medius (sometimes)

Muscles.	Quain. *Spinal Nerve Supply.*	Author's Results. *Spinal Nerve Supply.*
Posterior Division.		
1. Rectus posticus major	1st C	1st C, and sometimes one or other in 2nd C
2. Rectus posticus minor	1st C	
3. Superior oblique	1st, and sometimes 2nd C	
4. Inferior oblique	1st and 2nd C	
5. Complexus	1st, 2nd, 3rd, and 4th C	2nd and 3rd, and sometimes 1st or 4th C
6. Splenius	2nd, 3rd, and 4th C	2nd, 3rd, and 4th C
7. Trachelo-mastoid	2nd C	2nd and 3rd, and sometimes 1st or 4th C
8. Cervicalis Ascendens	(Not separately mentioned)	2nd, 3rd, and 4th C
9. Trapezius	3rd and 4th C	1st, 2nd, and 3rd C
Anterior Division.		
1. Rectus lateralis	1st C	(Not observed)
2. Rectus anticus minor	1st C	(Not observed)
3. Rectus anticus major	1st, 2nd, 3rd, and 4th C	2nd and 3rd C
4. Genio-hyoid	1st and 2nd C	No spinal supply
5. Sterno-mastoid	2nd and 3rd C	1st and 2nd, and sometimes 3rd C
6. Infra-hyoid muscles	1st, 2nd, and 3rd C	1st and 2nd C
7. Longus colli	2nd, 3rd, 4th, 5th, 6th, 7th, and 8th C	2nd, 3rd, 4th, 5th, 6th, and 7th C
8. Levator scapulæ	3rd, 4th, and 5th C	4th and 5th, and sometimes 3rd or 6th C
9. Rhomboidei	5th C	5th and 6th C

Muscles. Posterior Division.	Quain. *Spinal Nerve Supply.*	Author's Results. *Spinal Nerve Supply.*
10. Latissimus dorsi	7th and 8th C	6th, 7th, and 8th C, sometimes 1st T instead of 6th C
11. Scalenus anticus	5th and 6th C, and sometimes 4th C.	6th, 7th, and 8th C, and 1st and 2nd T, and sometimes 3rd T
12. Scalenus medius	3rd, 4th, 5th, 6th, and 7th C	6th, 7th, and 8th C, and 1st, 2nd, 3rd, and sometimes 4th T
13. Scalenus posticus	5th and 6th C	6th, 7th, and 8th C, and 1st and 2nd T, and sometimes 3rd T

Comparison of the Tabular Statements.

On comparing these tabular statements of my results with similar statements taken from the last edition of Quain's "Anatomy" we find the following discrepancies, which I propose to point out root by root. No mention is made of the trapezius being supplied by the first cervical root, a supply which I constantly observed; while the complexus is said by Quain to be represented in this root, but I only found it exceptionally represented. Owing to the difficulty of obtaining a satisfactory view of the muscle, I did not investigate the rectus anticus minor, so that it may be represented in this root as stated by Quain; but with regard to the rectus capitis anticus major, I could observe no response in the muscle when the first cervical root was excited. The genio-hyoid is said by Quain to be supplied from this root; but in the three experiments in which I paid special attention to the innervation of this muscle, I could obtain no response on excitation of the first cervical root.

As in the first root, so in the second, Quain makes no mention of the trapezius being represented, while I found it constantly so. Then again, no mention is made of the cervicalis ascendens, which I find supplied by this root. The inferior oblique is included by Quain with those muscles constantly supplied by this root, while in my experience such a supply is exceptional. With regard to the genio-hyoid, what was said when speaking of the first cervical root

applies here also; no response was obtained in the muscle on excitation of this root.

Like the first and second roots, the third, I find, innervates the trapezius, but no mention of such an innervation of this muscle is made by Quain. No special mention is made of the trachelo-mastoid and cervicalis ascendens, both of which I find represented in this root; but it is stated broadly in Quain that the "erector spinæ" is innervated from this root. The infra-hyoid muscles are given by Quain as represented in this root; but, like Beevor and Horsley, I only find these muscles represented in the first and second cervical roots.

The supply of the scalenus medius and levator scapulæ, and the occasional supply of the sterno-mastoid from this root, as stated in Quain, I have been unable to corroborate. It is interesting to note in this connection that, while I failed to find the levator scapulæ represented in this root, I nevertheless found the levator claviculæ (a muscle well developed in the monkey) so represented.

In the fourth root the complexus is included, in Quain's description, among those muscles constantly represented, but I find such a representation exceptional, and in no case have I found the complexus represented in the first and fourth cervical roots together, as stated in Quain. I do not find the rectus capitis anticus major represented in this root, while Quain does. The same statement applies to the trapezius, and scalenus anticus and medius. Then, again, I find the trachelo-mastoid sometimes supplied from this root, while no mention of such a supply is made in Quain.

With regard to the fifth cervical root, our only point of difference is as regards the supply of the scaleni. That they are innervated from this root, as stated by Quain, I have not been able to find any evidence.

In the sixth root I only find the scaleni sometimes represented, and not constantly, as stated by Quain. On the other hand, I find the latissimus dorsi frequently represented in this root, while no mention of its being so represented is made by Quain.

The seventh cervical root I find supplying all the scaleni;

but Quain only mentions the scalenus medius as supplied from this root. I find the scaleni supplied by the eighth cervical root, but no such supply is mentioned in Quain, whereas the longus colli is said to be innervated by this root, contrary to any experimental evidence I have been able to obtain on this point.

In that I further find the scaleni supplied by the upper three thoracic nerves, and the medius also from the fourth sometimes, while Quain gives the seventh cervical as the lowest root in which any of these muscles are represented; the discrepancy is very great.

General Remarks.

The impression with which I am left after comparing the results of this investigation with those I obtained formerly in connection with investigations into the functions of the spinal nerve roots responsible for the nerve supply of the muscles of the limbs, is that there is considerably more individual variation with regard to the nerve root supply of the neck muscles than of those of the limbs.

As in the case of the limb muscles, so in those of the neck, it was abundantly evident that different segments of any given muscle responded when one or other of the nerve roots supplying the muscle was excited electrically. In some muscles this differentiation of parts was such that, instead of the whole longitudinal extent of the muscle contracting on stimulation of a given root, where the muscle derived its nerve supply from three roots the upper portion would alone respond on excitation of the highest root of the series from which it derives its supply, while the middle portion would alone do so when the intermediate root of the series was stimulated, and the lower portion when the lowest of the three roots was excited. In other muscles, instead of its being a segment above or below another segment of a muscle which responds on excitation of this or that nerve root, the whole length of muscle responds, but only in so far as one part of it is concerned, so that if supplied by two nerve roots, the whole longitudinal extent of one lateral half of the muscle

would respond, while the other lateral half of it would remain quiescent in so far as active contraction of the muscle fibres are concerned, any movement in this part of the muscle being only brought about indirectly by being pulled on by the actively contracting portion which is contiguous to it.

This differentiation of one part of a muscle from another by means of its nerve root supply served as a means of differentiating different parts of a complex muscle such as the erector spinæ in a most interesting manner. Thus excitation of the second, third and fourth cervical nerve roots resulted in contraction of different lateral parts of the cervicalis ascendens muscle, the inner portion of the muscle, *i.e.*, nearest the middle line dorsally, responding chiefly from the second root, the outer portion of the muscle, *i.e.*, that farthest away from the middle line posteriorly, responding chiefly from the fourth root, while the intermediate portion of the muscle responded chiefly from the third root. On excitation of the fifth cervical nerve root the portion of the erector spinæ which now responded was that which is nearest to the middle line dorsally, altogether below the lower limit of the cervicalis ascendens, while excitation of the sixth root resulted in contraction of a portion of the muscle a little farther from the middle line dorsally, and so on.

On comparing the combined movement which results on excitation of a given nerve root with the individual muscles actually represented in the same root, it becomes obvious that the resulting combined movement must depend on the preponderance of certain muscles over others, which, if acting alone, would bring about a very different movement.

The muscles deriving their nerve supply from any given nerve root occupy both the dorsal and ventral aspects of the neck and trunk. Despite the variations met with as regards the precise muscles represented in a given root, or the roots supplying a given muscle, the movement resulting on excitation of each root remains wonderfully constant, such variations as are met with being so slight as not to alter materially the predominating movement characteristic of each root.

In the case of such small short muscles as the posterior recti and obliques, the nerve supply appeared to be derived

from a single nerve root in most cases, but this is quite exceptional, as all the other muscles of the neck and back investigated derived their nerve supply from two, three, or more nerve roots.

Practical Bearing of the Results on the Treatment of Wry-neck.

A knowledge of the precise muscles innervated by any given nerve root cannot fail to be of great advantage in any consideration as to the nerve roots, division of which is most likely to be attended with relief of the spasm in wry-neck. This is, of course, of special value with regard to those muscles sufficiently superficial in position to allow of their being seen or felt to be in a state of spasm. But the knowledge is also of use to us with regard to muscles too deeply seated to be seen or felt with certainty, for a knowledge of the movement brought about by the action of this or that muscle enables us to ascertain fairly satisfactorily which muscles are probably in a state of spasm.

Important as is this knowledge of the precise innervation of the various muscles responsible for the movements of the head on the trunk, no less important is the information which we derive from those experimental results in which the position assumed by the head in relation to the trunk was noted. The results obtained in this connection point clearly to the fact that in the most common forms of torticollis surgical treatment must be directed, chiefly at all events, to one or all of the four upper cervical nerve roots. It is further evident that where the lateral inclination of the head, by which the shoulder and side of the head and face are approximated, exists, the first and second cervical nerve roots are those to which attention should be directed, while in those instances in which the chief movement of the head is one in which the occiput is drawn backwards, so that the face looks more or less upwards, the third and fourth cervical nerve roots should engage our attention.

The results further point to the fifth and sixth cervical nerve roots being the least likely, of the cervical series, to

conduct impulses from the central nervous system, the cutting off of which are likely to be followed by any material relief in any muscular spasm which may be present causing wry-neck.

It is also important, with a view to prognosis after such operations, that even muscles supplied by the seventh and eighth cervical nerve roots may by their spasm bring about movements of the head on the trunk indirectly, in which connection the trivial indirect movement of the head resulting on excitation of the upper thoracic nerve roots may not be altogether without a certain amount of importance.

One class of cases of wry-neck, happily not common, is placed beyond the possibility of surgical relief, as far as division of nerve roots is concerned, by a consideration of the results obtained in this investigation. I allude to those cases in which the spasm is limited to, or chiefly involves, the scaleni muscles, which, as we have seen, are innervated from the sixth or seventh cervical to the third or fourth thoracic nerve roots. Apart from the obstacle to interference with the lower cervical and first thoracic nerve roots, owing to the innervation of the upper limb being derived from these roots, that accelerator and augmentor fibres of the heart are contained in the upper thoracic roots[1] may be a point not to be altogether neglected in any operation directed to these roots.

A not unimportant consideration in prognosis after such operations for wry-neck is the question whether in those cases, for instance, in which a muscle is represented mainly in two nerve roots, but also to a lesser degree in a third, the full force of nervous discharge may reach the muscle by this comparatively insignificant path now that it can no longer reach the muscle by the main paths. That this is not probable is suggested by the fact that any given nerve root only supplies a portion of muscle and not the whole muscle, as I have shown in this and previous papers; and that, in a case such as that supposed above, the portion of muscle would be very small, and, therefore, even if receiving the

[1] cf. Bradford and Dean, *Journal of Physiology*, vol. xvi., 1894, p. 34.

full force of the discharge from the central nervous system, the muscular fibres capable of responding are too few to give anything like an adequate exhibition of the full force of the central disturbance. As further supporting this view, may be instanced the fact that, in investigations in which the spinal nerve roots which contribute to the formation of the limb plexuses were the subjects of experimentation,[1] I showed that division of one or more nerve roots altered the position assumed by the limb during general convulsions subsequently induced, by the intra-venous injection of absinthe, in a manner directly proportional to the amount of this or that muscle excluded from taking part in the convulsions, in consequence of the division of one or more of the nerve roots from which it is innervated.

But as opposed to these considerations must be borne in mind the fact that, in the investigations dealing with the limb plexuses, I further showed that the power of recovery after division of a nerve root was wonderful, and that this occurred when all possibility of reunion of the divided ends of the roots taking place was placed beyond question. The immediate effect of the division showed itself in the altered position of the limb in standing, or of its movements in progression, weakness of one or more movements of the limb being evident; but in a wonderfully short time all traces of these defects passed off, and it became difficult to detect any abnormality in the position of the limb in standing, or of its movements in progression. That the provisions for compensation were great was evident, but it was not so easy to explain how this compensation was brought about. Of the theories that might be advanced in explanation of the fact, two appeared to me to be worthy of consideration. The first of these supposed it possible that certain cells of the cerebral cortex were responsible for impulses which could reach the same muscle by different nerve roots, and that the division of one of these nerve roots by which such impulses pass might produce a reflex inhibitory effect[2] on the cortical cells from which the impulse it conducts are

[1] *Phil. Trans. Roy. Soc.*, vol. clxxxiv., 1893, p. 41; and *Proc. Roy. Soc.*, vol. liv., 1893, p. 243.

[2] cf. Bubnoff and Heidenhain, *Pflüger's Archiv.*

derived, so that the amount of actual loss of motor power in the first instance is more than that resulting from the exclusion of the parts of the muscles innervated by the divided root, and that as the cerebral cells recover from the inhibitory action so power is restored to the parts of the muscles still in connection with the central nervous system. The second hypothesis supposed no such inhibitory effect on the cells of the cerebral cortex; these cells were regarded as being capable of discharging the same amount of energy as before, but the impulses meet with a block, owing to the division of the fibres along which they formerly passed. Gradually, however, the impulses are diverted, it may be through the anterior horn cells of the spinal cord along other channels, and thus, in time, the fibres of the muscle which are supplied by an intact root receive almost the same amount of stimulation as did the whole muscle formerly.

Between these two hypotheses there seemed little to choose, when dealing with the limb muscles, most of which received their innervation from two roots, and the difference of the amount supplied by the two roots was not too great to make either of these theories untenable.

A third possibility suggests itself to me as worthy of passing consideration. Is it possible that the normal stimulus reaching the fibres of the part of a muscle still in connection with the central nervous system is capable of inducing secondarily a contraction of other fibres of the same muscle no longer directly connected with the central nervous apparatus? In this connection it is worth remembering that, even after complete section of all the nerve fibres to a muscle, the muscle fibres do not become fattily disintegrated, but undergo a very slow process of atrophy. It is not unreasonable, therefore, to suppose that when only part of the nerve fibres of the muscle are divided the muscle fibres formerly in direct connection with them may be prevented from undergoing any great amount of this simple atrophy even, being brought into physiological action indirectly through the agency of some influence derived from the muscle fibres which are still in connection with the central nervous system.

NOTES OF TWO CASES OF PERIPHERAL NEURITIS, WITH COMPARATIVE RESULTS OF EXPERIMENTAL NERVE DEGENERATION AND CHANGES IN NERVE CELLS.

BY ROBERT A. FLEMING, M.D., F.R.C.P.E.

In this paper I wish to give an account of several cases of peripheral neuritis, mainly from the pathological point of view, to give a brief *résumé* of the results of my experimental nerve sections in dogs and rabbits, with a note of the changes found in nerves of stumps at varying dates after amputation, the changes in nerve cells of ganglia and cord on the affected side, and thereafter to show the bearing of certain of the results on peripheral neuritis.

So much has been written about peripheral neuritis—so many views formulated only to be afterwards demolished—that a disease of such varied modes of origin and clinical history requires to be attacked from many different standpoints. Perhaps there is no better method of approaching the problem than from the experimental side, and, although rabbits' nerves differ from those in man, the results obtained appear to me to throw an instructive light on the pathological anatomy of peripheral neuritis.

By the term "peripheral neuritis" I understand an affection primarily of the neuron, in whole or in part, and not a disease having its origin in some other part of the spinal cord.

The cases referred to will be published in full at a later date; all I wish to do here is to bring out the prominent

features, clinical and pathological, for the purposes of this paper. I wish to draw special attention to the changes in the vasa nervorum and the minute fibres in the affected nerves and in the nerve cells of the cord in one of the cases. No reference is made to the bibliography of the subjects discussed. I did not have the opportunity of examining Case I. during life.

Case I.[1]—Mrs. H., aged 38, admitted to Ward 25, Royal Infirmary, Edinburgh, on April 2, 1894, complaining of debility, great weakness of the hands, with almost complete loss of power in the feet and legs, with numbness and a sensation of "pins and needles" in the legs and arms. The weakness in the legs had lasted a fortnight; the numbness and loss of power in the lower limbs had been present for four days. She had six healthy children, all living—no miscarriages, and nothing to report in the family or personal history of special interest. Her last labour occurred three months before the commencement of the illness for which she was admitted, but it appears to have been normal in every respect.

The illness began a fortnight beforehand with a shivering attack, and she was confined to bed for one day. She resumed her household duties with difficulty for one week, and then was compelled to take to bed again. She had now severe pain in the back, relieved on sitting up. She tried to get up several times, but was unable to do so.

She had not been well fed, and was given to drink, locking herself into her room for the apparent purpose of indulging in that vice, and she admitted that during New Year week they had consumed nine bottles of whisky without help from visitors. There was no history of diphtheria or sore throat.

On admission—

Sensory Functions.—Pain in small of back, increased by palpation; numbness in hands and feet, and "pins and needles" sensation in hands.

Tactile Sensibility.—Unimpaired localisation and rate of conduction perfect.

Pain Sensation.—Apparently perfect.

Temperature Sense.—Impaired; delayed conduction, but she could distinguish heat from cold.

[1] History of case abstracted from Edinburgh Hospital Reports, vol. iii., p. 417.

Case I.

Cervical Enlargement.

Group.	Right Anterior Cornu.				Left Anterior Cornu.			
	Cell.	Nucleus.	Nucleolus.	Endo-Neucleolus.	Cell.	Nucleus.	Nucleolus.	Endo-Neucleolus.
Ventro-mesial	56μ × 40μ	22μ × 20μ	8μ	2·5μ	84μ × 60μ	26μ × 26μ	8μ	3μ
	44μ × 24μ	14μ × 12μ	8μ	3μ	80μ × 44μ	22μ × 22μ	6μ	3μ
Ventro-lateral	80μ × 34μ	20μ × 16μ	10μ	3μ	76μ × 50μ	26μ × 20μ	8μ	3·5μ
	30μ × 24μ	14μ × 12μ	8μ	2·5μ	68μ × 60μ	24μ × 24μ	10μ	4μ
Dorso-lateral	64μ × 40μ	20μ × 14μ	10μ	4μ	60μ × 50μ	9μ × 9μ	6μ	3μ
	44μ × 30μ	14μ × 12μ	8μ	3μ	50μ × 40μ	16μ × 14μ	8μ	4μ

Note.—The measurements of the cells and nuclei represent their greatest length and breadth, and in the case of the cells are taken from the roots of the processes.

Muscular Sense.—Noted as markedly impaired.

Skin Reflexes.—Plantar on each side abolished; others present.

Tendon Reflexes.—Patellar reflex absent on both sides.

Voluntary Movements.—Distinct pointing of the toes on both sides, due to a condition of talipes equinus since birth. Patient could not draw up her knees, but, if flexed, she could extend them. The inability to use her flexors was most marked on the right side. Grasp was feeble, and the flexors and extensors of the forearm were weak. The muscles of the upper arm were a little stronger.

Muscles of lower extremity did not react to faradism; but reaction was obtained in the upper extremities, though weak, but was specially deficient on the right side.

Vasomotor symptoms were absent, except a slight cyanosis of the feet and distension of the superficial veins.

Patient became extremely weak and irritable; complained of great pain in the back, which precluded a thorough investigation of the case.

Patient became cyanosed on April 5, at 9.15 a.m., from paralysis of the diaphragm and lower intercostal muscles, and artificial respiration, by Silvester's method, was kept up continuously for sixty-eight hours, when she died from exhaustion. Ether and strychnine were administered subcutaneously. She could swallow up to a late stage. Muscular weakness rapidly increased, the right arm being much weaker than the left.

Micturition and defæcation remained unaffected up to a late stage.

Post-mortem examination performed by Dr. Leith yielded nothing worthy of note.

Microscopical Examination.

The pathological examination of the nerves, etc., I made in the Practice of Physic Laboratory, Edinburgh University. The brain, pons, medulla, cord, and the following nerves were examined:—The brachial plexuses; the right ulnar; the sciatic, posterior tibial, and the right anterior crural nerves; the nerve to the left gastrocnemius muscle, with a portion of the muscle attached; the fourth right intercostal nerve; the phrenic nerves, with portions of the diaphragm; the vagi nerves and the sympathetic on the right side, with the superior cervical ganglion. Unfortunately, the external popliteal nerves were not obtained, nor any of the ganglia on the posterior nerve roots.

The changes met with were more remarkable than any I have seen described in the literature of peripheral neuritis, and the case was a unique one, inasmuch as the disease only lasted about twenty days from the commencement of symptoms, and the function of the intercostals was only seriously affected from seventy to eighty hours. On examining the brain I thought it might be worth while to try a modification of Golgi's method, described by Berkeley,[1] for the demonstration of dendrites, in a paper on lesions produced by the action of ethylic alcohol on the cortical nerve cells. Even though my specimens were somewhat over-hardened for this purpose in Müller's fluid, I found, in thus treating a portion of the left cortical motor area for the arm, that several of the ganglion cells showed the same marked bulging on their dendrites which has been described by Berkeley. The terminal dendritic swelling is the most marked, though, on close examination, other small moniliform swellings can be seen affecting several dendrites. None of the swellings appeared quite close to the cell body. These dendritic changes are best marked in the cortex of the motor areas, but were also noted over a wider area, and were seen in the cell groups forming the ganglia of the cranial nerves. The gemmulæ of the dendrites did not come out well, but they appear to be diminished in number, at least on the affected processes.

In the Cord.—The changes here are of the greatest interest. There are a great number of recent and older hæmorrhages best marked in the cervical and dorsal regions, and mostly found in the grey matter. At the level of the cervical enlargement a small encapsulated hæmorrhage is seen in the right antero-lateral ground tract, and is evidently from a centrifugal arteriole. The extravasation is surrounded by a laminated, hyaline wall, and there are a small number of leucocytes in the neighbourhood. In the course of the same arteriole, and nearer the anterior cornu, are several other smaller and apparently more recent hæmorrhages.

In the dorsal region, at the level of the second dorsal segment, one of the main branches of the anterior spinal artery to the right side of the canal is cut transversely and is seen to be blocked with a recent thrombus surrounded by numerous leucocytes. At the level of the ninth dorsal segment a large recent hæmorrhage is seen along the line of a branch of the anterior spinal artery, running to the outer side of the posterior cornu, in one of the septa. Here there is no great emigration of leucocytes.

[1] Brain, Part VI., 1895.

These hæmorrhages are limited to the anterior spinal artery and to the right side of the cord. Beyond the hæmorrhages of recent date, there are no vascular changes such as are seen in the nerves. The tracts of white matter show practically nothing. The nerve cells in the cord, although fixed in Müller's fluid, and, therefore, not so capable of showing nuclear changes, still demonstrate certain interesting facts, because the multipolar cells on the two sides of the cord show distinct differences. On the left side the cells are practically the nerve cells of a normal specimen hardened in Müller's fluid. On the *right* side we find great shrinking of the cells as a whole (see table of measurements). The nuclei are much smaller, and, in place of having a distinctly rounded contour, their outline is jagged and totally irregular. On the *left* side the cells show a clear, distinct nucleolus and endo-nucleolus, and a rich refractile chromatin network (see fig. 1). On the *right* side the whole nucleus stains more deeply and homogeneously, the nucleolus is with difficulty made out, and it is sometimes impossible to distinguish the endo-nucleolus. There are one or two healthy cells in each cell group of the *right* anterior horn ; and there are rather larger numbers of degenerated cells in the groups of the *left* anterior horn ; still the degenerated cells on the one side and the apparently normal cells on the other are quite characteristic. Anilin blue black reveals further that the affected cells possess fewer processes.

The ventro-mesial group of nerve cells is believed by Kaiser and others to be the centre for the nerves of the dorsal muscles, and the ventro-lateral group for the lateral and ventral muscles of the trunk and the muscles of respiration. In the cervical region the spinal accessory nerve is supposed to arise from the lateral group, the phrenic from the mesial. Now in this cord the ventro-lateral cell group is very markedly affected, and the inference is obvious. In this case, the patient had paralysis of the diaphragm on the right side more markedly than on the left, and the right intercostal muscles appeared also more markedly affected than the left. The greater number of degenerated cells at this level on the left side are in the ventro-lateral group, although there are a number of apparently healthy multipolar cells in addition.

As I chanced to have another cord with hæmorrhages at more than one level, and especially in the region of the cervical enlargement, I took several sections and made measurements of the cells and nuclei, selecting the largest cells from the different groups, and I noted that the mere effect of hæmorrhages being present in proximity to the cell groups made no difference in the

respective size of cells and nuclei, although the hæmorrhages in this case were very extensive.

The nerves were examined with particular care, and not merely were a few sections made from each, but many series of sections were examined, and portions of the same nerve were investigated at different levels.

In not a few of these nerves, the same description will suffice. In the *right sciatic, right anterior crural, left sciatic*, etc.

The Weigert-Pal specimens show many changes with only about half the fibres stained, and many that stain do so in a manner suggestive of an early stage of degeneration. Whereas this method stains most deeply the peripheral portion of the medullary sheath of a normal fibre, leaving a clear and faintly-stained central part, sometimes with a darker ring closely surrounding the axis-cylinder itself, a fibre in an *early* stage of degeneration stains often uniformly thoughout and uniformly deeply. In fact, with a carefully-stained section, if properly decolourised, a uniformly black fibre, with sometimes a clear central spot representing the axis-cylinder, would point to the degeneration of the myelin alone, or what Gombault describes as the condition constituting periaxial neuritis, whereas disappearance of the clear spot in the centre implies that the continuity of the axis-cylinder is broken as well as the myelin.

The most degenerated fibres are in groups arranged throughout the funiculi. The fibres staining a uniform colour are close to these groups, or are intimately mixed up with the fibres composing them. The healthy fibres are also generally arranged in groups together. Specimens stained with hæmatoxylin and eosin show a considerable number of comparatively healthy fibres in most of the funiculi, and the grouping of the degenerated fibres is very marked. Careful examination of the various funiculi shows that there are very few fine fibres, and that those which are degenerated appear to take their place. These fine fibres occur in groups, and I shall have to refer to them again at length.

In connection with the degenerated groups and the funiculi generally, there are a number of nucleated cells. Some of these nuclei are very large in size.

(*a*) Many belong to the spindle-shaped cells, whose protoplasm is very small in amount, staining brightly with eosin and showing marked signs of mitosis ; these are newly-formed, connective-tissue cells.

(*b*) Many are leucocytes, and these are chiefly in the neighbourhood of vessels, although they have spread amongst the

surrounding nerve fibres, being specially numerous near the little degenerated groups mentioned already. (See fig. 2.)

(*c*) A smaller proportion are enlarged and proliferated segmental nuclei, but, unless in fibres which have broken down, these can be seen inside the sheath of Schwann, and projecting into the lumen of the nerve fibre.

The axis-cylinders do not stain so sharply, and may be seen, even in transverse section, to be finely granular. There are few signs of proliferation of the segmental nuclei, though they are generally much distended, bulging into the lumen of the fibre. The larger septa of the endoneurium are not thickened; the finer septa, especially those separating individual fibres or smaller groups, show greatly enlarged and evidently proliferated connective tissue nuclei, but only in the neighbourhood of degenerated fibres, and these are also very marked in the proximity of vessels, and near the inner surface of the perineurium. The perineurium is very slightly thickened, but only in parts. The thickening is limited to the inner layers, and where vessels of small size are met with in the sheath, the newly-developed connective tissue nuclei are very numerous.

The epineurium shows little that is abnormal. The vessels are specially interesting because they illustrate the changes described by Minskowski in 1888, and later by a number of writers. Almost all the smaller arterioles and capillaries in the endoneurium or perineurium of those funiculi showing many degenerated fibres demonstrate the changes about to be described, though comparatively few in the epineurium do so. These changes are—distension and proliferation of the nuclei belonging to the endothelial cells of the intima, and to a less extent a similar nuclear increase in the media and adventitia. Comparatively few capillaries are so nearly blocked by the huge endothelial nuclei as in the more peripheral nerves. A considerable number of these nuclei show karyokinetic figures. The walls of the smaller arteries and arterioles are distinctly thickened. Almost every vessel within the perineurium seems to be surrounded by a greater or smaller number of leucocytes, and a huge diapedesis is shown in fig. 2, taken from one funiculus of the nerve. There is evidence of a considerable amount of exudation, which is greatest between the perineurium and the nerve fibres, and also in the neighbourhood of the arterioles and capillaries, well seen in fig 3.

In the more peripheral nerves the arterioles and capillaries have suffered even more markedly, whether in endo-, peri-, or

epineurium; they all show marked proliferation of nuclei, especially those in the intima, being greatly enlarged and showing very distinct mitotic figures. Many capillaries are so occluded that a red blood corpuscle could scarcely, if at all, pass through their lumen. The venules also show proliferation of endothelial nuclei. Corpora amylacea are present in many of the nerves more markedly degenerated.

The nerve to the left gastrocnemius muscle shows beautifully the effects of a recent large exudation, best marked in the neighbourhood of the vessels. It is present not merely between the nerve fibres and the perineurium, but it is well seen also along the septa. The specimen has been extremely well fixed, and the paraffin sections give what I believe to be a truthful picture. The effects of the exudation are very apparent; those fibres which have been pressed upon show marked degenerative changes, namely, granular myelin and irregularly distended and granular axis-cylinders. The fibres of the endoneurial septa and the inner lamellæ of the perineurium are separated by the effusion, and present an open network-like appearance. The exudation itself forms fibrinous threads or strands, here and there replaced by newly-formed connective tissue cells, and a considerable number of leucocytes are in its vicinity. The nerve fibres at some distance from the exudation are mostly normal, but still some show granular changes in the myelin and distended irregular axis-cylinders, staining feebly with eosin or benzo-purpurin. On the other hand, certain larger fibres appear to resist pressure more than others, but the comparative rarity of these, where the exudation is great in amount, is very striking. The fine fibres have evidently suffered most severely, and the connective tissue nuclei, increased in size and number all over the funiculi, are specially prominent near groups of what had once been these fibres. The segmental nuclei are enlarged, but there is little evidence as yet of proliferation.

The vessels of the endo- and perineurium show marked nuclear increase in the intima. The media of the arterioles in the same localities is thickened.

All the funiculi have not suffered alike, but all show some exudation, appearing first around the vessels in the neighbourhood of the perineurium.

Some of the nerves—as, for instance, the right ulnar, and especially the intercostals—show much more advanced changes in the myelin and axis-cylinders; and in these nerves, corpora amylacea are extremely numerous.

In several of the nerves of this case I endeavoured to study:—

(1) Whether the leucocyte emigration was confined to parts of the nerve or was generally met with throughout its length?

(2) Whether the diapedesis of red blood corpuscles was local?

(3) Whether the degenerated fibres (which could be fairly well followed in groups by noting their relationship to the blood-vessels) were healthy at any part of their course; or, if not, whether the degeneration varied at different levels as stated by many writers?

(4) Whether the nuclear changes in the blood vessels varied at different levels, and if they had any relationship to the phenomena noted under the other three headings?

The *phrenic nerves* were selected because they were small, and there was some chance of recognising the approximate position of fibres, or even identical fibres, in each section. My results are, in brief:—

(1) The leucocyte emigration varied greatly in different portions of the nerve, and where one vessel showed marked evidence of emigration most of those in the funiculus did the same. The leucocyte emigration tended to increase in the phrenics from above downwards—being most marked close to the diaphragm.

(2) The diapedesis of red blood corpuscles was equally local, and the emigration of leucocytes and diapedesis of red blood corpuscles in the nerves generally occurred together; but diapedesis of red blood corpuscles was less frequent.

(3) The degenerated fibres in the phrenics showed the most interesting changes. The fine fibres very gradually appeared to decrease in number from above downwards to the diaphragm, probably leaving the nerve to supply minute vessels *en route*. The larger fibres seemed to suffer more from the effects of pressure at a higher level than from a true parenchymatous degeneration. They were more dropsical-looking, *i.e.*, more distended, and of less regularly round and oval outline below as compared to the most

proximal sections. The medullary sheaths were granular in appearance, but the axis-cylinders were generally perfectly recognisable. One or two groups of markedly degenerated fibres in the right phrenic I followed carefully. These at one level were compressed together, by a large exudation and many leucocytes, so as to form a mass of myelin with unrecognisable sheaths of Schwann, with segmental and connective tissue nuclei, a few leucocytes, and even one or two axis-cylinders apparently unaffected. At a lower level these fibres had not recovered from their compression, and, although they gradually separated to some extent, they remained distended, and were not, in this nerve at least, absolutely normal-looking below. Apparently, with the possible exception of a few fibres, the changes in this nerve were not due entirely to a parenchymatous neuritis, but were largely the result of pressure, causing a secondary descending degeneration.

(4) The nuclear changes in the vessels vary very considerably at different levels, being much better marked where the emigration of leucocytes and red blood corpuscles were most in evidence, and less marked between.

Case II.—Mrs. M., aged 38, was admitted to Ward 25, Royal Infirmary, Edinburgh, on November 4, 1895, suffering from paralysis of the arms and legs, difficulty in swallowing, and loss of the power of speech.

She had a healthy family history, has had ten children, eldest aged 19; youngest, 3 years. Three are dead—one of bronchitis, and two of chest disease; the others are strong and healthy.

Patient has had no other illness, but had suffered from an occasional sore throat, yielding generally to local applications. Her mother suffered in the same way, and, in both cases, any slight exposure to cold was sufficient to bring on an attack. Mrs. M. had been very alcoholic, but to what extent could not be ascertained; though it is admitted that, during the greater part of her married life, she had been imbibing larger and larger quantities of spirits, beer, &c. She was rarely actually drunk, but often slightly stupefied.

There was no diphtheria in the district at the time of patient's illness, nor had the patient anything suggestive of the disease. She simply complained of one of her usual sore throats, which she

attributed to exposure to cold, and which on this occasion rapidly yielded to treatment. The patient never had any serious illness since childhood. There was no history of influenza as an antecedent to her condition, nor had she previously been attacked by it.

Three months ago the patient complained, for the first time, of numbness of the legs and swelling of the feet. These gradually increased, and patient was known to have resorted to an extra allowance of alcohol in the hope of curing the unpleasant symptoms. Patient took to bed on October 21, 1895, having only previously been laid up when the numbness and swelling incommoded her, as it did to an increasing extent; and she found a day's rest appeared to give some relief. She now had shooting pains and twitchings in her left arm, and, to a less extent, in her legs. On October 29 she found great difficulty in swallowing, and the power of speech was gradually lost. The patient, in a few days, became semi-comatose.

Notes on Admission.—She is a stout, flabby woman, with a somewhat bloated expression of countenance. There is nothing specially worthy of note in any system, excepting the nervous. The heart and lungs show nothing of special interest; the temperature, which was normal when the patient was admitted, rose to 102° Fahrenheit, where it remained for two days before death, and the pulse became at the same time correspondingly rapid.

Sensory Functions.—As the patient is almost unconscious, can be only with difficulty roused to the slightest extent, and is totally unable to speak, her subjective symptoms are unobtainable. Pain is evinced on pressure over the nerve trunks, and over certain muscles. The pupils are equal, and react sluggishly to light. The fundus of both eyes is normal.

Motor Functions.—Swallowing being almost impossible, the patient is fed by the rectum. Breathing is slow, somewhat stertorous, and from time to time simulates the Cheyne-Stokes type. The bowels and bladder are evacuated involuntarily. Skin reflexes and tendon reflexes of the legs and arms are abolished.

Electrical Reactions.—In examining the electrical reactions, I had the assistance of Dr. Garbut. On the first occasion the patient's skin was extremely hyperæsthetic, and it was very difficult to get accurate results, but our later efforts were more successful. One of the outstanding features was that a very powerful faradic current had to be used, far in excess of what a normal individual could endure. We tried at first a current which was too weak, although producing considerable pain when applied to our own persons, and with this we only got, almost universally,

a negative result. We next tried as powerful a current as we could obtain from a large induction coil; and when the skin was sufficiently anæsthetic to permit of observation, we noted that with nearly every muscle which had given us negative results before we now got a contraction, though, in some cases, an extremely feeble one; and this contraction was sometimes coincident with the qualitative change in the reaction of degeneration. Where the qualitative change, constituting one of the phenomena of the reaction of degeneration, was observed, the contraction was invariably noticed to be very sluggish, and obtainable with a smaller number of cells than usual.

The muscles of the extremities were examined in detail. It seems hardly necessary here to give the tables I prepared. Suffice it to say that the peronei and the tibialis anticus muscles of the left leg especially and some of the extensors and flexors of the fingers and the small thumb muscles of both arms gave the reaction of degeneration.

Vaso-Motor and Nutritive Functions.—There is slight œdema of both ankles. There is no evidence of any bedsore. None of the muscular groups are very markedly wasted—certainly the tibialis anticus and the calf muscles show considerable atrophy. The right leg is slightly smaller in circumference than the left. Both buttocks are flattened. The muscles of both fore-arms are flabby. Sweating is profuse, and has the peculiarly sour smell so often noted in cases of multiple neuritis.

Cerebral and Mental Functions.—Patient lies semi-comatose, as already described, and cannot be roused so as to comprehend questions asked of her. She evinces feelings of pain by a slight cry and a feeble, lethargic attempt to remove the limb from the source of the irritation.

Locomotory System.—There is nothing worthy of note as regards the joints. There are occasional slight jerking movements, especially of the arms.

The patient continued much in the same condition at first, gradually losing ground, then more rapidly after her temperature rose to 102° Fahrenheit. On the night of the 7th inst. her breathing became more difficult, the pulse more rapid and weaker, and she died on November 8th.

It is to be regretted that a more complete history of the patient's condition before admission to the hospital was not obtainable in this case. *Post-mortem examination* by Dr. Leith showed nothing worthy of special note here.

The following organs, &c., were examined microscopically.

The brain and cord, the eleventh and twelfth dorsal posterior nerve roots with ganglia, the right and left sympathetics, the optic nerves; the brachial plexus, the ulnar, median, musculo-spiral, musculo-cutaneous, sciatic, external popliteal, internal popliteal, posterior tibial, and the anterior tibial nerves of both sides were examined along with the extensor communis digitorum, extensor hallucis, and the tibialis anticus muscles; also the phrenic nerves, with diaphragm; the seventh intercostal nerves and muscles; the semi-lunar ganglia; pieces of the ventricular wall of the heart, the liver, &c. The specimens were all preserved in Müller's fluid, and unfortunately the ganglia were put into the same solution.

The motor areas of the brain were examined by the same method as used in Case I., but without any conclusive result.

The cord gave considerable trouble by not hardening properly. The multipolar cells in the anterior cornua show, however, the same changes as those described in Case I. There are no patches of degeneration in the white substance, and there are no hæmorrhages. The medium-sized vessels, especially between the cord and the membranes, show great thickening of the middle coat—the thickening having a hyaline appearance. Anterior and posterior roots are alike normal. The nerves showed the same arrangement of degenerated fibres in groups. The description of the right sciatic will suffice for the larger nerves.

Right Sciatic Nerve.—Many nerve fibres are normal, many show granulation or more advanced segmentation of myelin, and these fibres are mostly arranged in groups. They are associated with the presence of a greater number of segmental and connective tissue nuclei, and also leucocytes. There are some fibres which show loss of axis-cylinders; and, generally, these are in or near the groups referred to. Very few fine fibres are seen to be normal; most appear to be included in these groups. There is homogeneous pink-stained (eosin) fibrinous exudation between many of the fibres. This is most marked in connection with these groups of degenerated fibres, and they bear a distinct relationship to the position of the smaller arterioles and capillaries, from which the exudation may have originated. There are, however, far fewer leucocytes in connection with the exudation than might be expected, and these are mostly seen nearer the degenerated groups and the vessels. The segmental nuclei, especially near the degenerated groups, are greatly distended, and show evidence of proliferation, some of them showing well-marked mitotic figures.

The connective tissue nuclei have also increased in size and number, but only near the bundles of degenerated fibres. The smaller arterioles and capillaries show very marked enlargement and proliferation of the nuclei of the endothelial cells of the intima; most of the smaller vessels appear almost entirely blocked by these nuclei projecting into their lumen, and the nuclei may be seen in all stages of karyokinesis. These vascular changes, mainly involving the intima, and, to a much less extent, the media and adventitia, are best marked in the endoneurium, and are noticeable also in the perineurium and epineurium, but to a less extent.

The larger arteries in the endoneurium show a marked hyaline change in the media, a structureless-looking coat, often with few nuclei and staining feebly with eosin, and thus strongly contrasted with the fibrous tissue of the intima and adventitia. Arteries so affected measure in total diameter 80μ or thereby: the larger vessels do not show this change, nor do those which are much smaller in calibre than the measurement given. This hyaline appearance of the media is almost entirely confined to the arteries in the endoneurium. The perineurium is somewhat thickened in part, but almost entirely by the increased number of young connective tissue cells in the inner lamellæ of the sheath. Speaking generally, whereas all the funiculi show the vascular changes as regards nuclei, some do so much more markedly than others, and in those in which they are best developed the degenerative changes in the nerve fibres are much more in evidence.

Right Anterior Tibial Nerve.—This nerve is very markedly degenerated. The normal fibres are in little groups, but by far the greater proportion show segmentation of myelin, sheaths distended with droplets of myelin, and axis-cylinders very few in number. Where the axis-cylinders are seen, they extend generally for a short distance alone, and these small portions show typical vacuolation and granulation.

Fig. 4 represents the very advanced segmentation of the myelin in the fine fibres, as compared with the ordinary-sized fibres, and in peripheral neuritis, so far as my experience goes, these fine fibres nearly always suffer at an early date, and very severely.

The nuclei are increased in number, especially those of the connective tissue: there are a few leucocytes near the vessels, and the segmental nuclei are greatly enlarged, and, in not a few instances, proliferated.

The vascular changes are most typically seen in the endoneurium, also in the epineurium. Fig. 3 shows in transverse section the appearances just described, especially the vascular changes and the exudation.

The Right Ulnar Nerve is the last nerve of this case I shall describe here. It is much more markedly affected than the brachial plexus. Two-thirds of the fibres in all the funiculi show degenerative changes by Marchi's method. This nerve presents very different appearances at different levels. The axis-cylinders are much less interfered with than the medullary sheaths, although the latter are considerably thinner than normal. The degenerated fibres in a section which, at first sight, might be mistaken for a healthy nerve, are best marked round the periphery of each funiculus near the septa, and especially near the vessels. Those fibres show distension of sheath and loss of axis-cylinders, and are probably nerve fibres destroyed by pressure higher up, and in which the axis-cylinders may be actually broken across. Those fibres are not more numerous than one-tenth of the total number in the section at the level referred to. Many of the fibres possessing axis-cylinders have an irregular outline and seem somewhat separated the one from the other.

A longitudinal section shows all the appearances at different levels, the localised bulging of the sheath of Schwann by segmented myelin, the effusion between the fibres, and yet many of these very fibres appear above and below with fairly normal axis-cylinders. These changes may depend on vascular conditions, as an effusion is distinctly present along the line of certain of the vessels, and where these vessels run across the funiculus the exudation appears to spread widely. In the gap between the vessels and the neighbouring nerve fibres there are the fibrinous-looking remains of an exudation staining very feebly with eosin, and the marked changes in the medullary sheaths of the contiguous nerve fibres (segmentation, bulgings of wall, etc.), all bear witness to the detrimental effects of the pressure, while the connective tissue and segmental nuclei are enlarged and proliferated, but not very extensively. There are a few leucocytes.

The vessels in the endoneurium show marked nuclear changes, especially those vessels probably causing the exudation—but they are not so evident in the epineurium.

As a good instance of the change in the muscles of the affected limb, I describe the right tibialis anticus muscle.

Right Tibialis Anticus Muscle.—The transverse striation is better marked than could be expected, but many fibres show it

somewhat feebly. There are many nuclei, but these are mostly in the walls of the lymphatics and the capillaries. The sarcolemma nuclei are slightly increased in number, especially those belonging to the fibres which have lost, to some extent at least, their transverse striation. These nuclei may be seen in all the different stages of proliferatien. The capillaries between the fibres are strikingly empty of blood, and many of them are blocked with enormously swollen endothelial nuclei.

These cases are most instructive, because while differing in some respects they yet agree in others.

Firstly.—We have in both cases the same effusion. This effusion is most characteristic; it is found specially around the arterioles and capillaries in the endoneurial septa, often between the nerve fibres and the perineurium, and separating the inner lamellæ of the perineurium. It is not, in these cases at least, a typical inflammatory exudation. In Case I., where in many nerves it is less in amount, it is accompanied by a far greater number of leucocytes than in Case II., where the exudation is much more excessive. This exudation, where referred to at all in most text books on neuritis, is manifestly regarded as an evidence of inflammation, and is not accorded a prominent position. In both cases I have endeavoured to demonstrate that this exudation is greater in one part of the nerve than in another, and that it acts injuriously by compressing the nerve fibres in its neighbourhood, thus causing degenerative changes in them, not merely at the level where it occurs, but also to a greater or less extent peripherally. The exudation tends in time to organise, as many of my preparations show. (See fig. 3.)

Were the exudation to occur in a healthy nerve, it would cause pain, and possibly interfere with the conduction of impulses, sensory or motor, or both; but it could not occasion degenerative changes similar to those seen in my two cases, unless from some cause the nerve fibres were interrupted, or the effusion, becoming organised, had contracted, so as to exert hurtful pressure. But in these cases the stage of organisation of exudation is in its infancy, and the effusion

could, by spreading upwards and downwards, against no very excessive resistance, be prevented from doing any great amount of damage—were it not that the fibres from their condition are specially prone to injury.

Secondly.—In Cases I. and II. the vascular changes were the same; the same increase in size and number of the nuclei of intima, media, and adventitia, limited to the smaller vessels, and first found in endoneurial vessels, and, as the periphery is reached, extending to the vessels of the perineurium and epineurium.

There is no point more difficult to determine with certainty than the existence or non-existence of proliferation of nuclei in vessels. Enlargement can be decided by measurement, but not proliferation. Many of my specimens show, however, mitotic figures in these nuclei, and this seems to me to be sufficient evidence.

I noted in both cases that the effusion, whether with or without many leucocytes, was always accompanied by the vascular change, and that these vascular changes became better marked as the nerves were traced peripherally. Then the larger arteries in the endo- and sometimes the perineurium showed thickening of the middle coat, and the thickening was homogeneous in appearance, and with few nuclei in its structure.

Thirdly.—The condition of the nerve fibres themselves. Taking almost any of the affected nerves, a transverse section shows that effusion and degenerated fibres are in conjunction, whereas the healthier fibres are those least affected by an effusion. Many of the nerve fibres situated near an exudation are greatly distended. Fibres in the presence of an exudation often swell up much as normal nerve fibres do when placed in water; probably it means endosmosis.

But sometimes groups of degenerated fibres are seen without any effusion. This indicates that the fibres are probably pressed upon by an effusion at a higher level. I do not mean to assert that all peripheral nerve fibres degenerate as the result of pressure alone, but this was the principal cause, in the two cases I have recorded.

To put this point in a different way, I contend that although in alcoholic neuritis the nerve cell suffers chiefly, and with it the process—*i.e.*, the nerve fibre—yet the cell intoxication is not the chief cause of the advanced changes met with more in one fibre in a nerve than in another, but that a local agent—the effusion depending on vascular conditions—greatly expedites the degenerative changes in the previously weakened nerve fibres.

A true parenchymatous degeneration may occur in toxic peripheral neuritis, but there is, in addition, an interstitial effusion which aids in the process, and the greater the effusion the less is the chance of subsequent recovery.

I have noted in several of the nerves the fact that the very fine fibres, which are, I believe, mostly vaso-motor in function, are nearly always markedly degenerated.

Comparing fig. 4, already referred to, we see how much further advanced are the changes in the fine fibres than in those of average size. I believe that it is to the early changes met with in these fibres that the vascular conditions are partly due, but as after nerve section effusion is not the rule, but the exception, and the vascular changes are much less pronounced than in peripheral neuritis, I do not believe that the degeneration of the fine fibres is the cause of the effusion, but only aids in the nuclear changes in the arterioles, capillaries, &c. The changes in the axis-cylinders are well shown in both cases, granulation, swelling, etc.

Fourthly.—I found in Case I. the diminution and alteration in shape of the nuclei in the multipolar cells of the most affected side of the grey matter of the cord, and in some cells on the other. Had all the cells shown the same characteristics I might be compelled to admit that the condition was artificial, but the contrast between the two sides, as shown in my photograph, is too marked to allow of such a conclusion. The fact that the cortical cells in Case I. agree with Berkeley's observation on rabbits is of interest, suggesting that these cells are also affected.

In Case II. the electrical reactions compare in a remarkable way with the pathological descriptions, and it is specially interesting to note that an extraordinarily powerful

Faradic current had to be used before contraction of the muscles under examination were obtained.

In conclusion, in Case I. there is, with less exudation, less change in myelin and axis cylinder, and less marked change in vessels, the same amount of paralysis as in Case II., which shows more exudation, more segmentation of myelin and destruction of axis-cylinders and greater vascular changes; therefore in Case I. we have probably a much more advanced change in the central cells than in Case II., and the inference is that, but for the effusion, Case II. might possibly have survived much longer.

In connection with these two cases just contrasted together, I should like to note the similarity of changes in the right posterior tibial nerve from a case of diabetic neuritis, a male, aged 36, who died from a pulmonary complication, and not of diabetic coma. I am indebted for the specimen to Dr. Alexander Bruce, of Edinburgh.

Case of Diabetic Neuritis—Right Posterior Tibial Nerve.

Many fibres appear normal. These are mostly seen in groups, and the groups form a more or less irregular pattern in the funiculi. Between these groups of fibres those that are degenerated appear also grouped together. The myelin is extremely granular and the neurolemma tends in certain fibres to give way. The axis-cylinders of affected fibres are distended, sometimes showing regular bead-like swellings, granular, often vacuolated, and stain feebly. Among these groups of degenerated fibres many nucleated connective tissue cells appear. Some of these show greatly distended nuclei with a very small amount of protoplasm, the protoplasm staining deeply with eosin. They show evident traces of proliferation, and it is possible that by their presence they may exert a prejudicial influence over the nerve fibres in their neighbourhood. Segmental nuclei are not markedly proliferated, although they are distinctly enlarged in size. The exudation described in Cases I. and II. appears here

also, being specially well marked between the perineurium and the nerve fibres and along the lines of the septa.

The vessels show the most typical enlargement and pro liferation of the nuclei of the endotheliel cells of the intima; to a less extent the nuclei of the media and adventitia are similarly affected. The smaller vessels suffer most, although every vessel in the endoneurium appears affected, the arteries much more markedly than the veins. The vessels of the perineurium are involved, but to a less extent. In some of the larger arteries a local proliferation of the endothelium seems to have occurred, the process thus formed suggesting an appearance not unlike little endothelial buds. This appearance suggests the possibility of these little processes becoming detached and forming emboli in the minute capillaries. There are certainly little cells apparently free in the blood stream which are somewhat suggestive of free endothelial cells.

The appearances noted in the muscles of Case II., especially the left tibialis anticus, strongly suggest that these little emboli do occur.

Ascending Degeneration in Nerves.

A summary of the results of my experimental work on dogs and rabbits, and the observations made on the ulnar nerve of a stump ten years after the amputation of the arm, were given at the British Medical Association meeting in Carlisle last summer, and are to be found in the October number of the journal, and they appear in a more extended form in the January number of the *Edinburgh Medical Journal.* I shall, therefore, only give my conclusions in the briefest possible way.

In the ulnar nerve of the patient referred to, the fine medullated fibres have suffered most; they are markedly compressed by connective tissue. These fine fibres, which may be easily seen occurring in little groups, are most affected close to the terminal neuroma. The arterioles and

capillaries in the nerve (endo- peri- and epineurium) show the nuclear changes described in peripheral neuritis, but the changes become less obvious as the nerve is traced centrally. My inferences are that the fine fibres whose function is lost are replaced by connective tissue; that those fibres which have so lost their function become proportionately less numerous as the nerve is traced centrally; and that the nuclear changes in the vessels depend on the minute fibres whose function is not abrogated being compressed by the connective tissue replacing minute fibres whose function is abrogated. In other words, I believe that fine fibres are chiefly vaso-motor, and mainly suffer in ascending degeneration. The connective tissue increase around the larger fibres is extremely slight in proportion, even around fibres which must have lost their function. I should note that corresponding vascular changes occur in the muscles, &c.

In a large series of experiments on dogs and rabbits, mainly the latter, I find similar results.

Figs. 5 and 6 show longitudinal sections of the two sciatics from a rabbit. Fig. 5 is the normal nerve, and the fine fibres can be seen about the centre of the funiculus, forming a distinct strand. Fig. 6 is from the central end of the left sciatic twenty-three days after a double ligature was applied to the nerve. The well-marked connective tissue septa mark the site of fine medullated fibres, all of which suffered severely from the pressure, and there is little, if any, connective tissue increase elsewhere.

This change begins about the fourth day, and is observable in a week's time after operating; it occurs whether sections or ligatures be made, and is found in dogs and rabbits alike. The arterioles and capillaries show corresponding changes to those found in the human ulnar nerve after amputation. In short, the changes found in the central ends of rabbits' sciatics after section or ligature appear closely to correspond to those in the nerve of the stump in man. I have had the opportunity of corroborating these observations on the nerves of human stumps at different dates, but have still an insufficient number of cases at my disposal.

Descending Degeneration in Mixed Nerves, after Section or Ligature.

It is my intention only to refer to those changes which have any bearing on peripheral neuritis.

(1) Many nerve fibres in the peripheral end of a divided nerve in dogs and rabbits are distended. This distension, best marked within a few days after the operation, may be due to aggregation of myelin droplets; but where there has been an exudation from neighbouring vessels the fibres may swell up, probably from inbibition. This appearance is of very common occurrence in peripheral neuritis, although accidental in experimental sections.

(2) I do not propose to discuss at any great length the changes observed in the axis-cylinder. I have noted a granular, vacuolated, or distended appearance very commonly in the peripheral end of a divided nerve up to such a time as axis-cylinders are easily observed.

The life of an axis-cylinder in a severed nerve is of great interest. Observers state that they ought to be become unrecognisable in three to four weeks, and disappear altogether (*i.e.*, are unstainable) in a period not exceeding six weeks. In the rabbit, the peripheral end of the axis-cylinder remains for a longer time nearly normal in appearance after ligature than after section, just as segmentation of myelin and proliferation of segmental nuclei are slower in taking place; but, whatever happens, an axis-cylinder appears to derive trophic influence of some nature from the segmental cells.

An axis-cylinder may be seen swollen and distended in a fibre with apparently normal myelin, although the distension might be artificial. An axis-cylinder may be healthy, or apparently so, where myelin is granular or broken up (to a very limited extent), but an axis-cylinder is rarely, if ever, found to be normal in function or appearance where segmental nuclei are proliferated. It is difficult to bring forward any photographic proof of this, but a careful study of many sections of many specimens gives strong corroborative evi-

dence in favour of the truth of this theory, however unlikely it may seem from an embryological standpoint.

(3) The change in the vessels of a nerve undergoing Wallerian degeneration is described as consisting in enlargement and proliferation of endothelial nuclei of arterioles and capillaries, and slighter nuclear increase in media and adventitia. Venules are less affected. I would only here point out that these changes are not nearly so well marked in most of my experimental sections and ligatures as in peripheral neuritis, and in many cases not marked at all.

(4) The question so much discussed as to where the degeneration process begins in the peripheral end of a divided or ligatured nerve is a very difficult one to answer. I made a large series of experiments on rabbits with double sections and double ligatures, so as to observe whether the middle or peripheral portion of the nerve degenerated first. The operations were done without displacing the nerve and as long a middle portion was left as possible between the two sections or ligatures.

Notwithstanding that the middle portion is at a disadvantage, because the blood supply is presumably more interfered with, the balance of my evidence is in favour of the peripheral part commencing to degenerate first.

In one rabbit, for example, in the middle part the segmental nuclei have not proliferated to nearly the same extent as in the peripheral. In another rabbit, 23 days after double ligature, the segmental nuclei have evidently only just ceased proliferating, whereas the process had long ceased in the peripheral. After four days ligature or section the degenerative process appeared to commence all along the severed nerve, but to be most advanced in the peripheral part, with the exception of certain fibres in that part which long retained a fairly normal appearance, though they also eventually suffered.

I cannot, however, leave this subject without referring again to fig. 4, which is taken from the right anterior tibial nerve of Case II. It shows what is likewise well seen in the peripheral ends of the divided or ligatured nerves—namely, that the fine fibres suffer most severely. In the photograph

they may be seen markedly segmented. Their medullary sheaths show far more advanced degenerative changes than the ordinarily-sized fibres. In only one of the whole series of experiments on rabbits was the evidence not corroborative.

I propose next to contrast peripheral neuritis with descending Wallerian degeneration.

If a nerve fibre be severed completely from its centre, it undergoes Wallerian degeneration, but in peripheral neuritis the fibre below the level of the degeneration is apparently normal. There may be several such degenerated patches in the same fibre, with intervening and almost healthy-looking portions, although it is not likely that the peripheral end organs, whether sensory or motor, could be entirely, if at all, responsible for these healthy intervening portions.

The obvious conclusions are, that the fibres which show these alternating changes are still nourished by the cells from which they arise, and that the local changes are due to a locally injurious, or toxic, agent acting only in a limited area.

It has been stated that these degenerated tracts are at points where the nerves are exposed to external injury or pressure, for example, the ulnar nerve at the elbow, but this is far from being a complete explanation of the difficulty.

These degenerated patches have an infinitely wider range than merely points of possible external pressure; they are more marked peripherally than centrally, but, what is of much greater importance, these changes bear a distinct relationship to the condition of the vessels.

Wherever an exudation is well marked the vessels show most distinctly enlargement and proliferation of nuclei, and the hyaline-looking thickening of the middle coat of the endoneurial arteries. Why may not these conditions be due to the toxic action of a poison on the vaso-motor cells in the medulla? It cannot be solely due to this, or else all the vaso-motor nerves would be simultaneously interfered with, and it is undoubtedly true that in Cases I. and II. the vascular changes varied greatly in the different nerves, as the pathological specimens demonstrate. But the toxic agent may act locally as well as centrally. An injury to a nerve, as, for example, a squeeze or any local irritant, may damage

the fine fibres, which are probably vaso-constrictor, much more markedly than the larger fibres; and in any of the cases of peripheral neuritis which I have had the opportunity of examining, these fine fibres, arranged in groups, seem to be the point where the earliest and most marked proliferation of connective tissue nuclei is observable.

Each individual fibre obtains its nutritive supply from lymph or blood. The toxine in the blood will act with great virulence upon these fibres brought into immediate contact with it. The nerve fibre may be deriving nutritive material, more or less throughout its whole length, whether by means of segmental cells or not we cannot say; still there seem to be certain points along its course where it comes more directly under the influence of the blood current. The fact of the existence of the nuclear changes may be due to the lymph surrounding the fibre being to a certain extent toxic, and specially at the points where a further nutritive supply is derived from the capillaries, and where the greater toxic effect may be produced. This may cause an effusion at the level of distribution of the fibres to the nerve vessels. When a bundle of fibres shows degenerative changes at one level it shows similar changes, not merely repeatedly as the periphery is approached, but with shorter and shorter intervals of more normal nerve fibre between.

Changes in Nerve Cells. Ganglia on posterior nerve roots and multipolar cells in anterior cornua. (Rabbits and Dogs.)

After a very considerable amount of experiment, I found that a solution of corrosive sublimate (saturated in a 0·75 per cent. solution of common salt) with equal parts of water, heated to the temperature of the tissues, was the most satisfactory fixing agent for the nerve cells of rabbits' ganglia and cord, and I succeeded in getting fairly satisfactory nuclear networks (as compared at least with my earlier specimens) and almost no evidence of cell shrinkage. I had control nerve cells to compare with those which were abnormal. The stains used were toluidin blue and eosin, a method for which I was originally indebted to Dr. Gustav Mann.

Quoting from my paper, read before the British Medical Association at Carlisle, the changes I obtained were—

(1) The cells of the ganglia on the posterior nerve roots undergo definite changes as the result of nerve section or ligature, and do so at a much earlier period than the multipolar cells in the cord—beginning probably as early as the fourth day, and certainly by the seventh day.

(2) That one of the very first changes observed in the cells of ganglia and anterior cornu is a diminution in the size of the nucleus in proportion to the size of the cell, and that sometimes, but not in all cases, nucleoli also become smaller, and very frequently the nuclei take up an eccentric position, sometimes even bulging the cell wall.

(3) That in both sets of cells Nissl's granules, otherwise known as the chromatic granules, are either smaller in size, fewer in number, and scattered through the cell body tending to be most numerous round the nucleus, or else they are grouped together in large masses round the nucleus, leaving the periphery of the cell quite clear.

(4) That pericellular lymph spaces may become enlarged, especially around the ganglia cells, and where the enlargement is very marked the cells become proportionately smaller in size, although an actual atrophy may also occur. In several of my specimens I found large vacuoles—not the vacuoles described by many writers as occurring in the cells of the cord and cerebral cortex, which are probably to some extent artificial—but big vacuoles, more resembling hugely distended pericellular lymph spaces. They differ, however, inasmuch as they are surrounded by the remains of cell protoplasm containing chromatic granules.

(5) That in the multipolar cells not merely are there these changes in position and size of nuclei, and arrangements and number of chromatic granules, but there is, as a later phenomenon, marked disintegration of cell protoplasm, well seen in some of my specimens. This disintegration has been described by Marinesco as occurring in certain cord lesions in man. It consists of patches, which, with toluidin blue and eosin, are whitish in colour and surrounded by masses of chromatic granules.

This stage of disintegration follows only at a very late stage in the ganglion cells of the posterior nerve roots. It should, however, be stated that a varying number of normal cells occur in abnormal ganglia, and that a very few abnormal cells occur in normal ganglia. The changes in the cells most commonly observed in normal ganglia are an aggregation of chromatic granules round the nucleus, and more rarely in eccentric position of nuclei. By normal ganglia I mean ganglia of presumably healthy rabbits never submitted to any experiment at all, as well as ganglia unaffected by the experiments performed. The proportion of such abnormal cells in a normal ganglia rarely exceeds 2 per cent. of the whole. Vacuoles may also appear, but much more rarely, and their presence is quite exceptional.

Figs. 7 and 8 show the changes described in the ganglia on the posterior nerve roots—7 is the normal, 8 the affected side twenty-one weeks after double ligature of the sciatic. The ganglia are a pair belonging to the sciatics, and the operation in this case was successful in every way as regards healing by first intention and the subsequent health of the animal.

Fig. 9 shows the multipolar cells from the anterior cornua of a rabbit six weeks after a double ligature had been applied to one sciatic. The cells are taken from the same level of the cord in the "sciatic" region. The affected cells are to the left side of the photograph.

Tables of measurements showing the difference in size of neuclei and cells in the affected and normal sides will, I anticipate, be published in the March number of the *Edinburgh Medical Journal*. I may state, however, that with very few exceptions, in my forty to fifty experiments on rabbits, the changes recorded appeared in the affected side, and that the exceptional cases referred to were some two or three in number, in all of which the wound had become septic.

It seems quite comprehensible that the cells of the ganglia in the posterior nerve roots should suffer first, because nerve impulses pass normally up to them from the site of the lesion; whereas the cells of the multi-

polar cells in the anterior cornua normally send impulses downwards from them, although there is no question that impulses can pass in both directions along a motor nerve fibre.

Are the connective tissue changes along the lines of the fine medullated fibres in the central end of a divided nerve and the changes in the nerve cells of ganglia and cord due to irritation? This is a very difficult question to answer. I do not believe irritation from the site of the lesion is alone the cause, because very different results were obtained on examining the nerve cells in septic cases; and further, the rabbits never showed any signs of pain, eating a hearty meal whenever they recovered from the anæsthetic, and feeding regularly and well till the day of their death. I am about to try to eliminate still more the possible irritation factor in producing "ascending degeneration" in a new series of experiments, as irritation may not always be evidenced by pain.

The suggestions which may be culled from comparing the cases of peripheral neuritis and the nerve degeneration, and the changes in nerve cells as the result of experimental section or ligature, are:—

The fine medullated fibres, which appear to play such an important *rôle* in "ascending degeneration," degenerate very early in peripheral neuritis, and are associated with marked vascular changes.

The comparative absence of the vascular changes in Wallerian degeneration is evident from an examination of my series of specimens.

The limited nature of the vascular changes in the central end of the divided sciatic in dogs and rabbits, and in the nerves of stumps in man, suggests not merely the relationship of fine medullated fibres to the vessels, but is also a strong contrast to the great vascular changes found so frequently, at least, in peripheral neuritis.

Peripheral neuritis is different from either ascending or descending degeneration, and is manifestly caused by a toxine acting on cells as well as nerve fibres, and possibly vessels.

The fact that in Case I., and also Case II., the multipolar cells showed the same changes that appeared after experimental section (so far as the methods of fixing would permit of comparison), is most suggestive, and I would draw especial attention to the diminished size of the nuclei in the affected cells, a point I have not seen noted by other observers.

The changes which I found in the cells of the ganglia on the posterior nerve roots after a nerve section or ligature appeared at such a very early period, much earlier in fact than in the multipolar cells, that they suggest the theory that the division of an axis-cylinder process acts more rapidly in a prejudicial manner on that cell to which the process normally conducts nerve impulses.

This would naturally suggest that if the multipolar cells and ganglion cells were affected by a toxic agent, the processes normally conducting *from* the cell would suffer more rapidly than the processes normally conducting *to* the cell.

My results show that, up to a certain date after section or ligature, the ganglia will suffer most organic change, but that after three weeks the multipolar cells rapidly undergo alteration, and that in six to seven weeks, according to Marinesco, Golgi, and others, disintegration of protoplasm occurs. My results, however, only show distinct disintegration of protoplasm of the cells of the posterior nerve root ganglia after fifteen to twenty weeks, although it is well marked in multipolar cells in three to seven weeks. May not this explain why, in peripheral neuritis, long after paralysis is almost complete, sensation to pain may persist, to some extent at least?

In peripheral neuritis, the condition of these ganglia cells have not met with much attention, "vacuolation" and "hyaline swelling," &c., being referred to in only a few cases; but I trust to be able in a few months to have some work completed upon the condition of these cells, in one case, at least, of this disease.

In conclusion, I may state that I have been engaged upon the histology of these fine fibres, and their distribution, etc., at different stages of life, in human nerves. The results are

as yet incomplete, but this much may be asserted: that the connective tissue supporting the fine fibres becomes gradually increased as age advances, and that diseases causing arterial degeneration appear to expedite the process.

The methods used in the microscopical work were numerous; nerves were, however, always fixed in Müller's fluid, and portions of cord, etc., in corrosive sublimate. Hæmatoxylin and benzo-purpurin were mostly employed for staining nerves, in addition to Weigert-Pal's method, and toluidin blue and eosin for nerve cells. I cannot close this paper without expressing my thanks to Sir Thomas Grainger Stewart and Dr. George A. Gibson for permission to give details of the two cases of peripheral neuritis which were under their care, and by whose courtesy I obtained the specimens described in the earlier part of this paper.

DESCRIPTION OF FIGURES.

FIG. 1.—Multipolar cells from the anterior cornua of the cervical enlargement of the spinal cord of Case I.

a. = Cells from left anterior cornu.
b. = Cells from right anterior cornu.
nc. = Multipolar cell.
nu. = Nucleus.
nl. = Nucleolus.
enl. = Endonucleolus.

FIG. 2.—Right sciatic nerve, Case I., showing recent leucocyte exudation around arteriole.

arl. = Arteriole.
l. = Leucocytes.
ex. = Exudation.
n. = Normal nerve fibre.
nx. = Degenerated nerve fibre.

FIG. 3.—Left sciatic nerve, Case II., showing exudation, with commencing organisation, and the change in coat of arteriole.

arl. = Arteriole.
ex. = Exudation.
fn. = Newly-formed connective tissue fibres.
en. = Endothelial nuclei.
me. = Media.
ad. = Adventitia.
adn. = Nuclei of adventitia.

a. *b.*

FIG. 1.

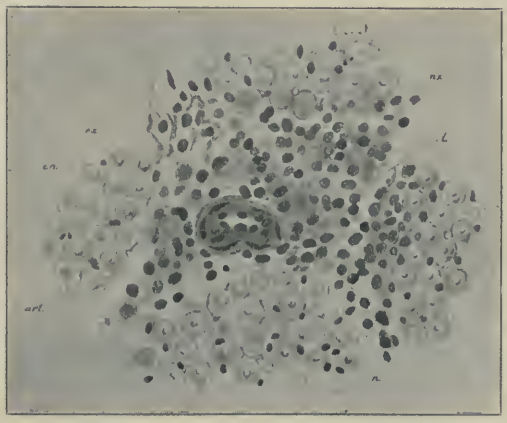


FIG. 2.

FIG. 3.

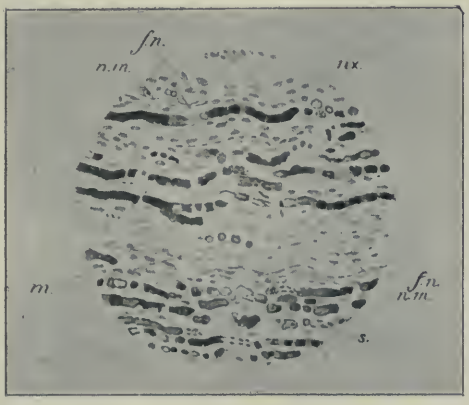


FIG. 4.

FIG. 5.

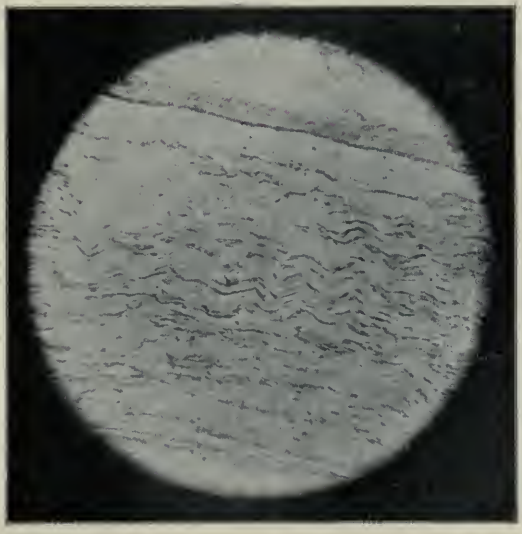


FIG. 6.

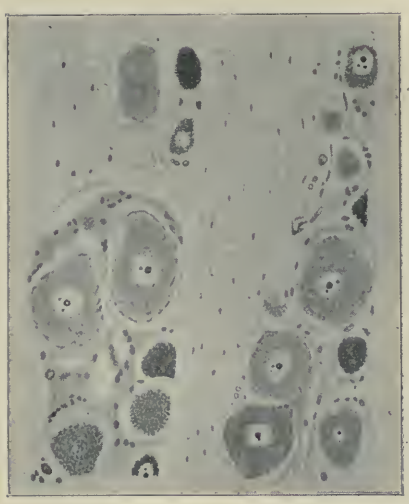


FIG. 7.

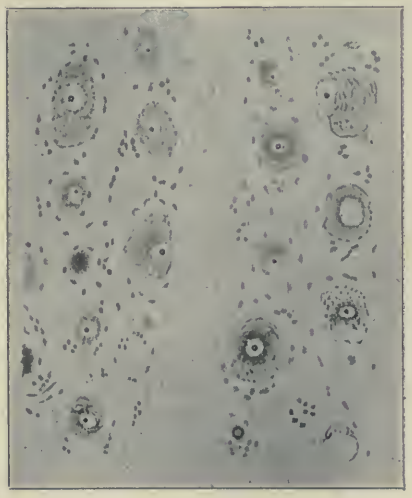
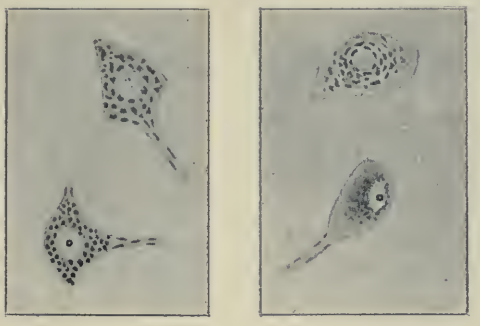


FIG. 8.



a. *b.*

FIG. 9.

FIG. 4.—Right anterior tibial nerve, Case II , showing greater segmentation of fine than of average-sized fibres.

nm. = Degenerated fine fibres.
fn. = Proliferated connective tissue in relation to these fibres.
s. = Enlarged segmental nucleus.
nx. = Degenerated nerve fibres—average size.
m. = Myelin droplets.

FIG. 5.—Right sciatic nerve of rabbit—unaffected side—showing fine medullated fibres in groups or strands.

FIG. 6.—Left sciatic nerve of rabbit—central end, twenty-three days after application of a double ligature—showing thickened connective tissue septa replacing fine medullated fibres.

FIG. 7.—Ganglion on right posterior nerve root (sciatic region) of rabbit—unaffected side—stained with toluidin-blue and eosin.

FIG. 8.—Ganglion on left posterior nerve root (sciatic region) of rabbit—twenty-one weeks after double ligature—stained with toluidin-blue and eosin.

FIG. 9.—Multipolar cells from anterior cornua (sciatic region) of rabbit's cord.

a. = Normal side.
b. = Cells from affected side, six weeks after a double ligature had been applied to sciatic nerve.

TWO CASES OF PORENCEPHALY.

BY J. WIGLESWORTH, M.D.LOND., M.R.C.P.

Case 1.—Joseph B., aged 24, single, was first admitted into Rainhill Asylum in March, 1890. He was the youngest of five children, all of whom were living and healthy. His parents were living, his father being 60 years of age and his mother 64, their respective ages at marriage having been 25 and 29; there was no consanguinity. His father was intemperate, and was said to be of an excitable temperament, but, with this exception, there was nothing of note in the family history, and, in particular, there was no evidence of epilepsy or other form of nervous trouble. The patient himself had smallpox when 3 months old, and had been delicate since; he did not walk until he was 4 years of age; went to school at 6, and remained until he was 14. He was said to have been intelligent and bright up to the age of 13 years, but he did not advance after that; he was tried at office work at 14, but failed in memory, and had to give it up. At 15, epileptic fits supervened, and he gradually became more dull; he assisted, however, in his father's shop up to the age of 23; the fits, however, became worse, and during the year previous to admission he became maniacal and unmanageable after them, and had at length to be placed under restraint. There was at no time any history of injury.

The patient, on admission, was a young man of fair physical development, 5 ft. 6 in. in height, and weighed 10 st. 2 lbs. Regular features; brown hair; grey irides; pupils, 4 mm., equal, regular, reacted normally to stimuli. Deep reflexes exaggerated. No definite paralysis, but he had a little weakness of the right hand, and he used to wear a leather band round his wrist to support it. Thoracic and abdominal viscera normal.

He had a marked stammer in his speech. No sensory defect observed. He had an intelligent expression. He was rational and coherent in conversation, and able to give a very fair account of himself, and quite realised his position; he was, however, evidently rather simple, and there was general slight obtuseness of his mental faculties, with some impairment of memory. He was very fanciful and hypochondriacal, constantly complaining about trivial matters and imaginary ailments, after the manner of epileptics generally; and at times he was excitable, and disposed to be impulsive and aggressive. He had a large number of fits, especially at night. His mental condition might, indeed, be described as one of moderate imbecility. He improved somewhat under treatment, and in December, 1890, his father was allowed to take him out. He again, however, proved unmanageable at home, and was brought back to the asylum ten months later, viz., in September, 1891. His condition then had not altered materially, but he was somewhat more dull. His fits continued numerous, and these frequently assumed a hysterical character, with apparently purposive movements. During the year 1892 he had 453 fits; during 1893 he had 226; and 152 during 1894; of the last-named number, 47 occurred during an attack of status epilepticus in the month of May. In 1895 the fits again increased to 239 in number. In February, 1896, he was again seized with an attack of status epilepticus, which proved fatal in five days, after a series of 54 fits of a very severe character.

Autopsy (*Fifteen Hours after Death*).

An examination of the head only was permitted.

Cranium.—Skull symmetrical externally, somewhat increased in thickness and density, the left side being on an average 3 mm. thicker than the right; sinuses and venous system generally gorged with blood; a general absence of fluid in the subdural space. Pia-arachnoid somewhat increased in thickness, the vessels of the pia being intensely congested. Thinly scattered over the surface of the convolutions in both hemispheres were some small circular, slightly-elevated nodules, about 1 mm. to 2 mm. across, which, for the most part, came away with the pia when this was stripped, and consisted chiefly of fibrous tissue. The whole brain was much congested, the grey matter of the cortex and basal ganglia being everywhere dark, and the consistence was firm.

Weights.

Brain (immediately after removal)	=	1,372 grammes
Right hemisphere (unstripped)	=	662 ,,
,, ,, (stripped) ...	=	640 ,,
Left ,, (unstripped)	=	539 ,,
,, ,, (stripped) ...	=	515 ,,
Cerebellum	=	148 ,,
,, Right half	=	69 ,,
,, Left half	=	79 ,,
Pons and medulla	=	32 ,,

Right Hemisphere.—Convolutions large and well formed, and these and the main sulci follow a typical course. No focal lesion. In fact, with the exception of the general congestion, due to the mode of death, this hemisphere was perfectly normal.

Left Hemisphere.—This presented, at first sight, nothing particularly abnormal (except the general increase of vascularity above mentioned), but when the membranes were stripped it was found that these covered, and for the most part concealed, a considerable cavity which, roughly speaking, occupied the upper part of the ascending limb of the fissure of Sylvius, and which had been formed by the removal of considerable portions of the convolutions bounding this area. Strictly speaking, indeed, it was scarcely correct to talk of cavity formation, as before the removal of the membranes the surrounding parts were so nearly in apposition that the aperture which had existed was almost obliterated, so that there was no cystic appearance, and it was not until the lobes were separated from each other that the considerable loss of substance which had occurred was clearly apparent. The missing portions are as follows:—At the upper part, the lower fourth of the ascending frontal and ascending parietal convolutions, together with the anterior half of the supra-marginal gyrus have completely disappeared, whilst inferiorly the temporo-sphenoidal lobe is divided by a deep fissure situated at about the junction of the middle with the posterior third, which cuts through all the three external convolutions of this lobe to a depth of about 1½ cm.; the fissure is 2 cm. broad at its upper end where it traverses the first temporo-sphenoidal gyrus, and tapers off gradually to a breadth of 1 cm. at the third temporo-sphenoidal gyrus; the adjacent long convolutions of the island of Reil, which run into the temporo-spenoidal lobe have also disappeared. The lower ends of

the two central gyri have a slightly eroded appearance, but the ends of all the other defective convolutions where they abut on the cavity are neatly rounded off, and there is no induration round the margins of the cavity. There is no communication with the ventricle. Round the hollow thus formed the different sulci and gyri (especially those at the posterior part of the hemisphere) are grouped in a distinctly radiate manner, so as greatly to obscure the normal arrangement, and make recognition of the different parts somewhat difficult. By tracing up, however, the callosomarginal sulcus on the inner aspect of the hemisphere (where the convolutions follow a normal course) it is possible to locate accurately the fissure of Rolando, and it is thus found that this fissure is not only situated further back than normal, but that it runs in an almost vertical direction from above downwards, its lower end terminating in the cavity above described; it is also shorter than normal, owing to the loss of substance of the two limiting convolutions, ascending frontal and ascending parietal, which in addition to the loss of their lower fourths, are reduced in size throughout their remaining lengths. Behind the fissure of Rolando run a series of deep fissures from before backwards, spreading out, roughly speaking, like the branches of a fan from the cavity above-mentioned, and the convolutions bounding these fissures have, of course, a similar arrangement. Immediately behind the fissure of Rolando, and almost parallel with it, is a fissure which appears to represent the anterior half of the intraparietal sulcus, or post-central sulcus, running in a nearly straight direction from above downwards; and behind this, the ascending limb of the fissure of Sylvius runs in a sloping direction, almost up to the margin of the hemisphere; behind and below this again comes a deep fissure, obviously the posterior part of the first temporo-sphenoidal or parallel sulcus, which cuts right through the parietal lobe, and almost joins the parieto-occipital fissure. Towards the lower and outer margin of the temporo-sphenoidal lobe, the third temporo-sphenoidal sulcus runs back as a deep and well-marked sulcus from the fissure cavity in this lobe, almost to the tip of the occiput; whilst between this sulcus and the first temporo-sphenoidal one, above-mentioned, two more deep sulci run backwards from the fissure-cavity to the occiput in an approximately parallel direction, one of which is, however, interrupted by a bridging convolution; these two latter sulci appear to represent a re-duplicated second temporo-sphenoidal sulcus. All these temporo-sphenoidal sulci run directly into the fissure-cavity which cuts across the temporo-sphenoidal lobe and appear to terminate

in it, the first one alone having an apparent continuation on the opposite (anterior) side of the lobe; on the anterior side of this lobe, however, two other short but deep sulci curve upwards and backwards over its lower margin as if attempting to meet those on the opposite side of the fissure-cavity.

The first temporo-sphenoidal gyrus is much reduced in size, but the other convolutions of this lobe appear to be of average depth and size, except of course where their structure is destroyed by the fissure-cavity which cuts across them.

Turning now to the frontal lobe the first thing that strikes one is its apparent increase in size owing to the throwing backward of the fissure of Rolando, and the convolutions and sulci present considerable abnormality, they having a definitely too vertical direction in the posterior part of this lobe, whilst in the anterior part there is a tendency for them to run in a curved direction from before, backward and downward instead of following the usual horizontal direction; so that in this lobe also there is a tendency for the convolutions to assume a radial direction though in a much less marked manner than in the posterior part of the brain. Immediately in front of the fissure of Rolando and parallel with it, is a deep and well-marked sulcus, which runs in a vertical direction from the margin of the hemisphere into the Sylvian fissure, being interrupted only in one spot by a short, partially sunken, bridging convolution, which probably represents the præ-central sulcus. In front of this again is another deep fissure running in a vertical direction, but with a little inclination from before backward, which, however, stops short before it reaches the upper margin of the hemisphere, whilst at its lower extremity it is separated from the Sylvian fissure by a well-marked fold of convolution. Immediately in front of this again is a deep vertical fissure running upward for about an inch from the Sylvian fissure, which appears to represent the sulcus diagonalis. The ascending ramus of the Sylvian fissure is about normal in length, but the anterior horizontal ramus of this fissure is both deeper and longer than usual. The other sulci of this lobe have a somewhat irregular course, but the tendency is for them to run from before backwards and downwards, in a curved direction as before remarked. The convolutions of this lobe are large and well-formed, and have of course a direction corresponding to that of the sulci. On the orbital surface a deep and well-marked fissure runs from before backward, throughout the whole length of this lobe parallel with the sulcus olfactorius, and separated from the tri-radiate sulcus, which is formed much in the usual fashion, by a well-formed con-

volution, running parallel with it. The convolutions and sulci on the inner aspect of this hemisphere have a quite typical arrangement.

The *Basal ganglia, Pons, and Medulla,* and *Cerebellum* shared in the general congestion, but appeared quite normal, except that, as indicated in the weights given above, the right half of the cerebellum was somewhat smaller than the left.

Microscopical Examination by Dr. Campbell.

Left Hemisphere (fresh cortex, stained with aniline). Sections taken from the para-central lobule showed a somewhat dense epicerebral fibrillation; in the first layer a few large succulent spider cells are visible, but the nerve-cells throughout the section show practically no morbid change. Vessels normal.

Crus, Pons and *Medulla* (method of Pal and ammonia-carmine). Sections made from the level of the third nucleus down to the olives, showed no sign of secondary degeneration or disease, except that it was thought that the superior cerebellar peduncular fibres were a trifle larger on the left side than the right.

Remarks.

Although it may be impossible to determine the exact date of the lesion in this case, it may, I think, with some confidence be assigned to the period of intra-uterine life, and probably to about the sixth or seventh month of development. Kundrat drew special attention to a radiate arrangement of the convolutions round the margins of the cavity, which he found in some of his cases, and considered that such an arrangement pointed to the congenital origin of the cases in which it was found, and on this point I am quite in agreement with him; the profound changes which have occurred in the grouping of the sulci and gyri in the case just described, point clearly to some morbid agency at work at a comparatively early period in the development of the brain. As to the nature of this agency, however, there seems room for more difference of opinion, and, indeed, writers on the subject have expressed very diverse views with reference to it. Into a general discussion of the question space will not

permit me here to enter, but I would point out that it is incorrect to use the term "porencephaly" as connoting a separate pathological entity, since different cases undoubtedly have their origin in different pathological processes. In the case before us the chief part of the destroyed area, that, namely, comprised by the lower ends of the two central gyri, anterior part of supra-marginal gyrus, posterior convolutions of insula, and portion of posterior part of first temporo-sphenoidal gyrus, is just that region which is very liable to be destroyed by softening in adult life and in old age, as a result of obstruction of a branch of the middle cerebral artery by thrombosis or embolism; and that the greater part of the lesion in this case, as above indicated, has had such an origin is hence strongly suggested. But it seems impossible to account for the deep fissure which cuts across the temporo-sphenoidal lobe—that is to say for that portion of the fissure which lies below the first temporo-sphenoidal gyrus—on any theory of vascular obstruction, or localised encephalitis; and it seems probable that we have here to deal with a true developmental defect. Such an idea is favoured by the great depth of the fissure in proportion to its width, by the neatly rounded ends of the convolutions abutting on it showing no trace of previous erosion, and by the manner in which it runs without break into a deep normal fissure, the third temporo-sphenoidal.

That the main portion of the lesion which has produced the porencephalic cavity in this case has been instrumental in causing the profound changes in the grouping of the sulci and gyri around it hardly admits of doubt; and that such a disturbing influence on development operating on the temporo-sphenoidal lobe may in like manner have caused the fissure cavity which cuts across it, seems a reasonable supposition. On this view, then, the main portion of the porencephalic cavity (as above defined) would have had its origin in a definite pathological process; whilst a minor portion would be due to an abnormality of development resulting therefrom. Such a view would at any rate account for the appearances presented, without inventing imaginary arterial distributions to fit in with them.

Case 2.—John Y., aged 21 years, had been an inmate of Rainhill Asylum for five and a-half years. He was sent from the workhouse, and no friends ever came to see him, so that, unfortunately, it was impossible to obtain any history of the case. He was about 5 ft. 2 in. in height, and 98 lbs. in weight; poorly developed. His features were very coarse and heavy, and he had no growth of hair on his lips or chin. Ears large and coarse, directed forwards. He had a left-sided partial spastic hemiplegia; the left arm was retained in the position of flexion, pressed against the side, and showed but little voluntary movement, whilst the left leg was the subject of a marked talipes equino-varus; the patient could, however, walk fairly well without assistance; the left arm and leg were also much shortened, and exhibited both in the muscular and osseous systems a remarkable diminution in size in all measurements as compared with the opposite limbs. The knee-jerk was exaggerated on the left side. His viscera appeared sound. Mentally he was an idiot of a most degraded type; he had a most fatuous expression; he had no power of speech, and showed hardly the least sign of intelligence, scarcely seeming to understand the most simple things; he masturbated freely, and was wet and dirty in his habits; he had to be washed and dressed, and required attending to almost like an infant; he was, however, able to feed himself. He was subject to frequent severe epileptic fits, having on an average about 180 annually. He eventually died of phthisis in July, 1896.

Autopsy (*thirteen hours after death*).

Thorax.—Both lungs full of tubercle and contained cavities at apices.

Heart.—Normal.

Abdomen.—Abdominal viscera normal, with the exception that the ascending colon contained a few tubercular ulcers.

Skull.

Cranium.—Antero-posterior diameter, 6½ inches; temporal diameter, 4½ inches; considerable asymmetry of the skull cap, the bones of the vault on the right side being distinctly smaller than those on the left; the frontal, temporal, and occipital fossæ on the right side are also clearly smaller than those on the left; the crista galli is pushed towards the right side; the left cerebellar fossa is smaller than the right. The lines of the middle meningeal

arteries are clear on both sides, but the ramifications are more distinct and deeper on the left. Sinuses contain black and soft white clot. Dura mater markedly relaxed on right side, not decidedly thickened anywhere; no unusual adhesion to bone, but a few more adhesions to arachnoid than usual on both sides. About $1\frac{1}{2}$ oz. of clear fluid in subdural space, but no blood or membrane. The arachnoid does not dip down between the hemispheres in the frontal regions, and the two inner surfaces of the frontal segments are loosely adherent to one another. Arachnoid and pia covering the whole of the right hemisphere, well vascularised, markedly thickened, opaque, and œdematous, and not adherent to the subjacent cortex of those convolutions which remain intact (to be subsequently described), but strips quite easily. On the right side a small anterior cerebral artery can be made out, but one can only find traces of a middle and posterior cerebral; the right internal carotid is markedly smaller than left. On the left side the pia-arachnoid is practically normal.

Left Hemisphere.—The ascending parietal convolution at its lower end, for a distance of about 2 cm., is markedly shrunken so as to form a mere ridge of nervous tissue, the atrophied part, however, does not extend quite to the lower end of the gyrus, but is separated from the Sylvian fissure by a normal fold of convolution. There is, however, no other lesion, and the rest of the hemisphere may be said to be perfectly normal. The convolutions are large and well formed, and follow for the most part a typical course, the only exception being that there is an unusually deep external parieto-occipital fissure, which extends for about 3 cm. on to the external aspect of the hemisphere, and joins the posterior part of the intra-parietal sulcus.

The Right Hemisphere resembled at first sight a large cyst, and was indeed converted for the most part into a bag of fluid. Broadly speaking, the cerebral substance has almost entirely disappeared over the posterior two-thirds of the brain, the only portions of the brain remaining partially intact, being a part of the frontal lobe and the extreme tip of the occipital. The best preserved portions of the frontal lobe are the convolutions of the mesial aspect and of the orbital surface; on the mesial aspect indeed, the first frontal gyrus (marginal gyrus) and the gyrus fornicatus are well formed for about the anterior two-thirds of their length, although reduced to about one half the normal size, the latter gyrus, however, extending a little further back than the former. The posterior terminations of these two gyri are not rounded off, but are somewhat sharply and irregularly cut. The

continuation of the first frontal gyrus on the orbital surface—the gyrus rectus—is also well formed, as are also the orbital continuations of the second and third frontal gyri; in fact, the convolutions of the triangular orbital plate may be said to be intact, although like the gyri on the mesial aspect, reduced in size, but the outermost portion of this plate is turned upwards by the distortion of the brain, so as to appear on the exterior instead of the under surface of the brain. On the external aspect the ascending frontal gyrus has nearly disappeared, though remains of it can be made out; of the frontal gyri in front of this the first or superior is fairly well formed, though a portion of the anterior end has disappeared; the second frontal gyrus is fairly, though imperfectly, developed in its posterior half, whilst the anterior half has quite disappeared, the convolution again re-appearing on the orbital plate; the third frontal gyrus has nearly disappeared, the anterior two-thirds having gone altogether, and the posterior third being only represented by a few vermicular remains of gyri—the portion of the gyrus which bends round to assist in forming the orbital plate is, however, fairly intact. The convolutions of the island of Reil have completely disappeared, their former position being merely indicated by a few faintly elevated lines. At the posterior end of the hemisphere the convolutions of the extreme tip of the occipital lobe are fairly intact, although reduced in size. The whole of the brain, however, between this part and the frontal region, comprising the whole of the parietal lobe, the whole of the temporo-sphenoidal lobe, and the greater portion of the occipital lobe, may be said to have almost entirely disappeared, there being left throughout the whole of this large region merely an exceedingly thin layer of nervous tissue adherent to thickened membranes upon which outlines of former convolutions can here and there be faintly mapped out. In places, indeed, the nervous substance has gone altogether, nothing being left but thickened pia-arachnoid and ventricular ependyma, and where a layer of nervous tissue exists between these two membranes it is for the most part no thicker than writing paper, but as the frontal lobe is approached the remains of nervous tissue become more pronounced, and the region comprised by the two central gyri is less completely destroyed than the other parts of the area indicated. There is, however, no actual communication between the lateral ventricle and the surface of the hemisphere, and the cystic appearance presented when the brain was first removed was caused by the great distension of the lateral ventricle, and in particular of its posterior and descending

cornua, the outer wall of the cyst being formed by thickened pia-arachnoid and ventricular ependyma containing in places remains of nervous tissue as described above. The ventricle contained about $3\frac{1}{2}$ ozs. of clear fluid.

The *Corpus Callosum* is fairly well developed in its anterior half, although reduced to about one half the normal size, but it rapidly tapers off as the anterior region is receded from, and is represented in its posterior half merely by a very thin film, about the thickness of tissue paper. The *corpus striatum* and *optic thalamus* are reduced to about one half the usual size, but are otherwise normal in appearance and consistence. (Owing to the disappearance of the temporo-sphenoidal lobe, these bodies form a prominence in the anterior part of this region when the brain is viewed from the external aspect.) The *corpora quadrigemina* are symmetrical, but appear smaller than normal. The third ventricle, velum interpositum, infundibulum, pituitary gland, and pineal gland can be seen as usual.

Cerebellum.—The left half is appreciably smaller than the right, the diminution being general, and not confined to any particular region. The *crus* on the right side is very small as compared with the left. The *fornix* is very small. The *pons* and *medulla* are asymmetrical, showing a diminution in size on the right side as compared with the left. *Right olfactory nerve* and *bulb* appear normal. *Left optic nerve* remarkably reduced in size; right less so.

Weights.

Encephalon (with contained fluid)	...	820 grammes
,, (drained of fluid)	...	700 ,,
Right hemisphere	...	102 ,,
Left ,, (unstripped) ...	...	455 ,,
,, ,, (stripped) ...	...	433 ,,
Cerebellum...	...	118 ,,
Pons and medulla...	...	25 ,,

Microscopic Examination of Crus Cerebri, Pons Varolii, Medulla Oblongata, and Spinal Cord, &c., by Dr. A. W. Campbell.

Serial transverse sections of the crus, pons, and medulla were made, and every fifth section mounted and examined. From the spinal cord, sections from every alternate segment down to the conus terminalis were taken. The stains employed were—(1) the

method of Pal, for the demonstration of medullated nerve fibres, and (2) ammonia carmine, for the display of cellular and connective tissue elements.

Crus Cerebri (Plate III., fig. 1).—In all the sections of the crus marked asymmetry is evident, the right half being obviously smaller than the left; this asymmetry is due to non-development of a number of structures pertaining to the right side. The right pedal system is exceedingly small, possessing not more than one-third of the bulk of the left, and it is interesting to note that while its inner third is fairly rich in nerve fibres (probably the frontal pontine tract), its outer two-thirds is almost entirely deprived of them. The right brachium conjunctivum, or superior cerebellar peduncle, is likewise reduced in size; above the decussation, where the two tracts lie close together on either side of the middle line (forming the so-called "white nuclei"), the right is seen to be barely half the size of the left, and the decussation, which takes place lower down, is of course very unequal. The right lemniscus is much paler and more slender than the left; also the bundle from the lemniscus to the pes, which is normal on the left side, can scarcely be defined on the right. The posterior longitudinal bundles appear normal on both sides. The fibres streaming over the upper border of the red nuclei towards Meynert's decussation (Meynert's "*fontainartige Fasern*") are distinctly scanty on the right side, and Forel's decussation is ill-defined. The nuclei and roots of the third nerve are approximately equal on either side. The anterior corpora quadrigemina seem to be normal, but the right posterior corpus quadrigeminum is obviously smaller than the left; its myelinic network is not so complex, its nerve cells are less numerous, and its brachium, compared with that of the left, possesses hardly any healthy fibres. The right red nucleus is distinctly diminished in size; it shows an excess of connective tissue, and is poor in nerve fibres—in its lower two-thirds especially. The right substantia nigra Soemeringii is reduced in extent, but the myclinic plexus in this structure on the two sides is alike.

Pons Varolii (Plate III., fig. 2). The asymmetrical appearance of the sections is altered in character as one proceeds down the pons. Owing to the decussation of the fibres of the superior cerebellar peduncles, the right peduncle becomes the larger, while the left diminishes in size correspondingly; hence the right upper half of the section appears greater than the left, while in the lower half of the section the disparity remains the same as it was above. With regard to the course of the right pyramidal tract in the pons, only a few small bundles of medullated nerve fibres can be seen,

and these gradually decrease in calibre, and eventually disappear at about the level of the sixth pair of cranial nerve nuclei. Below this the position of the right pyramidal field is just indicated by some contracted fields containing fibrous tissue. The pyramidal tract on the left side is normal. With reference to the superior cerebellar peduncles, that on the right side progressively gains in size over the left as the decussation proceeds, and finally when the crossing is completed it is to be noted that the left is little more than half the size of the right. The disparity is best seen where the two structures, having assumed their position on either side of the fourth ventricle prior to disappearing in the cerebellum, can be viewed in complete transverse section. The right lemniscus inspected in its varying positions along the pons continues markedly smaller than the left. The diminution in size seems to be more pronounced in the mesial than the lateral portion. The bundles of fibres composing the middle cerebellar peduncles seen in transverse sections, sweeping across the ventral segments of the pons are of somewhat smaller calibre on the left side than on the right. Also at the lower end of the pons the bundles of fibres proceeding to form the corpora restiformia, or inferior cerebellar peduncles, appear reduced in calibre on the left side. (This reduction in size of the left middle and inferior cerebellar peduncles is obviously the outcome of the crossed cerebellar atrophy consequent on the cerebral defect.) The posterior longitudinal bundles and the small bundles of fibres cut transversely in the upper part of the tegmentum appear of equal size, but the delicate horizontally directed bundles of the tegmentum are more obvious on the left side than on the right, though the right tegmentum forms a more extensive field than the left. The nuclei and roots of all cranial nerves in the pons may be considered healthy.

Medulla Oblongata (Plate III., fig. 3.).—There is not a single medullated nerve fibre in the right pyramid, its position is simply indicated by a small crescentic area which stains deeply with ammonia carmine and remains unstained by the method of Pal. The left pyramid is of normal bulk, but it is rounded instead of being flattened on its inner surface, evidently owing to withdrawal of the support normally given by the opposite pyramid. The nucleus of the right anterior cornu seen above the pyramidal decussation is distinctly small in size and poor in nerve cells, but below the decussation it rapidly gains a normal aspect, while the left anterior cornu, the nucleus of which was normal above, now diminishes in size. The reduction in volume of the right lemniscus is well seen

between the two great olives, and at the lemniscar decussation (the raphe), and below it, the fibræ arcuatæ internæ of the right side form much larger bundles and are much more prominent than the same fibres on the opposite side, also the nuclei of the funiculus gracilis and funiculus cuneatus, as well as the substantia gelatinosa Rolandi, to which the arculate fibres run are distinctly larger and contain more nerve cells on the right side. The hilum of the right great olive is wider than that of the left, and the right olive generally is expanded, but this is probably the result of the absence of the right pyramid, and of no special degenerative significance, because there are no changes in its nerve cells, or myelinic plexus of any import, and the accessory olives, with the exception of slight alterations in position, are equal on either side.

Spinal Cord (Plate IV., fig. 2).—Along the whole length of the spinal cord down even to the lower sacral segments there is most pronounced asymmetry, the left half being greatly smaller than the right. The inequality affects grey and white matter alike, and is appreciated best when the lateral columns are compared. The area of the left lateral pyramidal tract is marked by the presence of a small sclerosed field containing few healthy nerve fibres, and this sclerosis can be followed down as far as the eleventh dorsal level. In the position of the right direct pyramidal tract there is no sclerosis, but the anterior column on that side is evidently smaller than the left, and the disparity is maintained as low down as the eighth dorsal segment. The left anterior cornu is smaller, and contains less nerve cells than the right, all the way down the cord; the lateral projection at the cervical and lumbar enlargements has particularly suffered from wasting. The left posterior cornu is short and stout compared with the opposite one, but seems to contain as many nerve cells.

Peripheral Nerves.—The musculo-spiral and sciatic nerves of either side were examined; those of the left side presented a general reduction in calibre of the fasciculi, a thickening of the perineurium, a marked falling out of nerve fibres, and an undue prevalence of endoneural connective tissue and nuclear elements, while those of the right side were approximately healthy.

Microscopic Examination of Cerebral Cortex.—Portions of the cerebral cortex of the right hemisphere, taken (1) from the first frontal convolution, where it had maintained its shape, and (2) from the exceedingly wasted temporal segment, were closely examined for nerve cells and nerve fibres. In sections of the latter no sign of a nerve cell or a nerve fibre can be made out; all that is to be seen is a thick and cellular pia-arachnoid membrane,

enclosing blood vessels with thickened walls, and beneath this a cellular layer, which seems to be the thickened ventricular ependyma. In the sections of the first frontal convolution are numerous nerve cells, and these can be differentiated into layers; the first layer is thin and pale, and appears to contain no nerve cells; the second layer shows only a few small pyramidal cells, with a large nucleus; the layer of large pyramidal cells is soon reached, and continues for some depth; the cells of this layer are of smaller size than normal, and their processes are stunted; they possess a large nucleus, a definite nucleolus, and the investing protoplasm, though thin, shows a definite arrangement of chromophilic particles. Before the white matter is reached there is a recognisable layer of oval or fusiform cells. All the cortical blood vessels are thickened. In the white matter are numerous nerve fibres, but the reticulum formed by them is far less dense than that seen in the normal brain.

Remarks.

It is perhaps a question whether this case should in strictness be included under the term "porencephaly," but similar cases have been described by Kundrat and others under this head, and it is convenient provisionally, at any rate, so to classify it.

In discussing the pathology of the case, it is clear, from the nature and distribution of the cerebral loss, that we have not here to deal with a case of developmental defect, but that the lesion has been caused by a definite destructive process of some sort. And the first thing that strikes one is that the lesion corresponds in a general way with arterial distribution, the portions of the hemisphere which have suffered most being those supplied by the posterior and middle cerebral arteries, whilst the region supplied by the anterior cerebral is comparatively intact; and the blocking of the two former vessels by thrombosis or embolism occurring during fœtal life or about the period of birth or early infancy, might be considered a sufficient explanation of the appearances presented. It may be remarked here that the small size of the opto-striate bodies, seems clearly to be due to want of development, their general integrity being incompatible with

the idea that their arterial supply has been cut off. If, therefore, the above-named vessels—the posterior and middle cerebral—have been occluded, they must each have been blocked independently just after the vessels which supply the basal ganglia, have been given off. But the distribution of the lesion is not so exactly confined to arterial territories as we should expect to find it, had the lesion the origin above suggested. For, in the first place, though the territory supplied by the anterior cerebral artery is that which is most intact, this region is still by no means free from invasion. For on the exterior aspect of the frontal lobe, it is the *anterior* portions of the first and second frontal gyri, which are most destroyed—the parts of these gyri, that is, which get their blood from the anterior cerebral; whilst on the mesial aspect the intact convolutions do not extend further back than about the middle line of the brain, whilst the portions between this and the parieto-occipital fissure—paracentral and quadrate lobules, &c.—have completely gone. But it is well known that the anterior cerebral artery supplies the whole of the upper edge and mesial aspect of the hemisphere, as far back as the parieto-occipital fissure, so that portions of the area supplied by the anterior cerebral have disappeared as completely as the region supplied by the other vessels. Again, at the other extremity of the hemisphere, it is difficult to account for the comparative retention of the tip of the occipital lobe upon the theory of vascular obstruction. That area, though small, is quite distinct, and must, therefore, have retained at any rate a portion of its blood supply from the posterior cerebral; but the remainder of the area supplied by this artery has so completely disappeared, that if the wasting were due to arterial obstruction, no partial occlusion of the artery would suffice to explain it, but the blocking must have been complete, and hence it is very difficult to see how any area, however small, can have survived.

A destructive encephalitis occurring during fœtal life or early infancy, would, no doubt, explain the appearances presented, although it might be difficult to understand how such a process could be started. I am rather, however,

inclined to the idea that the lesion is the result of injury inflicted during the process of birth, not improbably by means of the forceps. A severe crushing of the hemisphere thus produced, with effusion of blood, might not improbably have caused such complete disorganization of the area involved, that recovery was impossible, and hence a slow process of absorption of the injured parts might have set in, which eventually produced the appearances found. The very slight lesion of the opposite hemisphere is quite compatible with this view.

This view, at any rate, harmonises with the facts that the lesion must have dated either from about birth or very early life, as evidenced by the asymmetry of the skull, and the arrest of development of the paralysed parts, and the non-development of the pyramidal tract: and it also fits in with the view that the patient was, so to speak, an accidental idiot—one in whom the idiocy was due, not to a congenital mal-development, but to some supervening accident (using this word in its widest sense); for the left hemisphere was, as I have elsewhere observed, well developed, and there is no reason to suppose, from an examination of this, that the patient would not have possessed the average intelligence of his class, if his other hemisphere had been in a similar condition. A history would, no doubt, have thrown some light on the case, but, as before remarked, no account was obtainable of the patient's antecedents.

NOTE ON LITERATURE.

Among the more important memoirs dealing with the subject of Porencephaly, the following may be mentioned:—

KUNDRAT. "Die Porencephalie, eine anatomische Studie." Graz, 1882.

AUDRY. "Les Porencephalies," *Revue de Médécine.* June and July, 1888.

SCHATTENBERG. "Ueber einen umfangreichen porencephalischen Defect des Gehirns bei einem Erwachsenen." *Beiträge zur path. Anat. und zur allg. Path.*, 5. Band, 1. Heft. Jena, 1889.

NORMAN AND FRASER. "A case of Porencephaly." *Journal of Mental Science.* Oct., 1894.

Anyone interested in the subject will find in the above communications abundant references to other published papers and cases, which limits of space will not permit of being reproduced here.

PLATE I.

104'

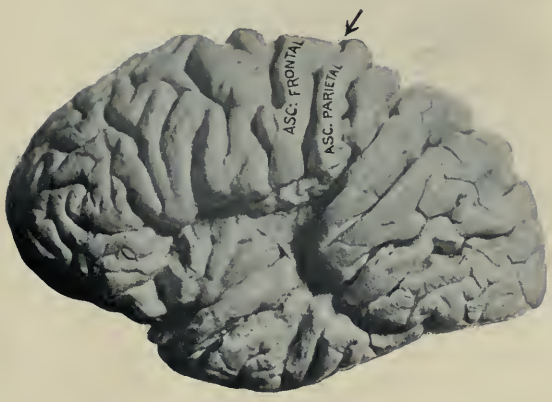


FIG. 1.

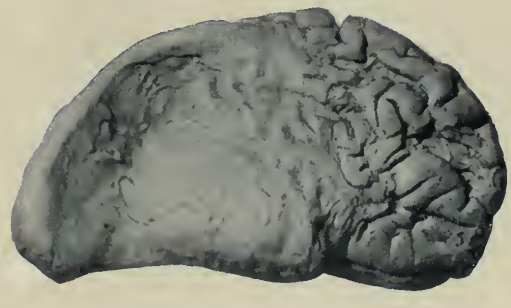


FIG. 2.

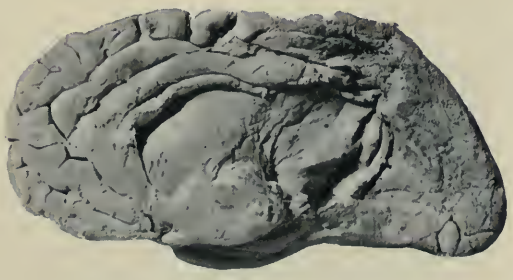


FIG. 3.

PLATE II.

FIG. 1.

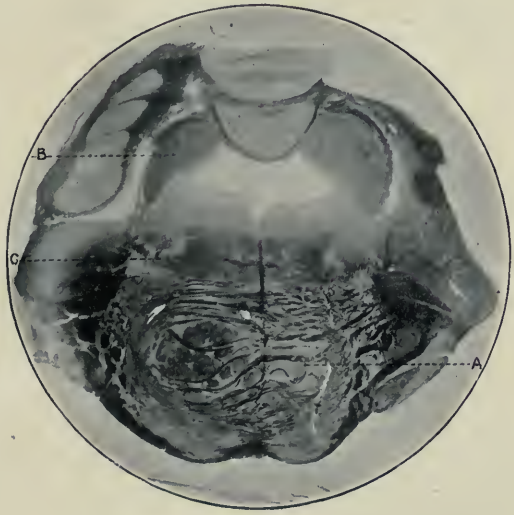


FIG. 2.

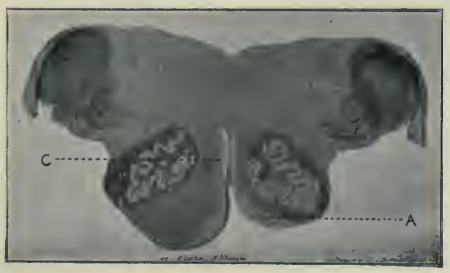


FIG. 3.

PLATE III.

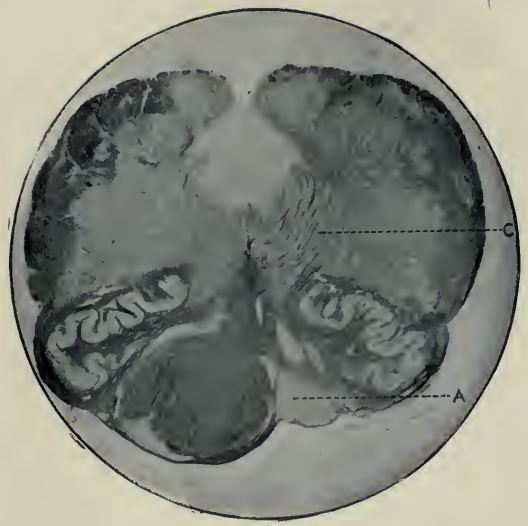


FIG. 1.

FIG. 2.

EXPLANATION OF PLATES.

Plate I.

Case 1.

Fig. 1.—Left hemisphere of J. B., outer surface. Absence of lower ends of two central gyri and anterior half of supra-marginal gyrus, &c., together with fissure cutting across temporo-sphenoidal lobe. Radiate arrangement of convolutions round the porencephalic defect. The arrow indicates the fissure of Rolando.

Case 2.

Fig. 2.—Right hemisphere of J. Y., outer surface. The prominence in the temporo-sphenoidal area consists of the opto-striate bodies, covered by the membranous remains of the temporo-sphenoidal lobe. Immediately in front of this projection is seen the orbital plate of the frontal lobe, drawn upwards and outwards, so as to appear on the outer surface of the hemisphere.

Fig. 3.—Right hemisphere of J. Y., inner aspect.

Case 2.—*J. Y.*

(Explanation of Plates II. and III.)

The figures are all reproductions of photo-micrographs of sections, stained by the method of Pal—medullated nerve fibres appearing black, nuclear matter and sclerosed tissues white.

A = Pedal system, or fibres of motor tract.
B = Brachium conjunctivum, or superior cerebellar peduncle.
C = Lemniscus, or fillet.
D = Substantia nigra Soemmeringii.

Plate II.

Fig. 1.—Section through the crus, above the decussation of the brachia conjunctiva. The posterior corpora quadrigemina appear at the top of the figure, the pedal system at the base, and the brachia conjunctiva on either side of the middle line. There is pronounced asymmetry, owing to reduction in size of the right pedal system, substantia nigra, brachium conjunctivum, lemniscus, and posterior corpus quadrigeminum.

Fig. 2.—Section through upper part of pons, below the decussation of the brachia conjunctiva, showing the left brachium (B) to be much the smaller. Right motor tract (A) absent.

Fig. 3.—Section through upper end of medulla oblongata. Right pyramid (A) absent, and interolivary layer (lemniscus) markedly narrowed on right side. General asymmetry obvious.

Plate III.

Fig. 1.—Section through lower end of medulla oblongata. Right pyramid (A) absent. Fibræ arcuatæ internæ (C) normal on right side, wasted on left. Nuclei of funiculus gracilis and funiculus cuneatus and the substantia gelatinosa Rolandi, of small size, on left side.

Fig. 2.—Section through spinal cord in upper cervical region. Left half greatly smaller than right. Sclerosis in position of left lateral pyramidal tract (A). Right anterior column reduced in size. Left anterior cornu smaller than right. Left posterior cornu short and stunted.

ON A CASE OF PSYCHRO-ÆSTHESIA.

BY LEONARD G. GUTHRIE, M.A., M.D., OXON.; M.R.C.P., LONDON.

Physician to Regent's Park Hospital for Epilepsy and Paralysis, Physician to Out-patients, Paddington Green Children's Hospital, and North-West London Hospital.

I. F., bricklayer, aged 52, has been under my care at Regent's Park Hospital for Epilepsy and Paralysis since March, 1894. He complained then, and has constantly complained since, of a sensation of painful coldness chiefly affecting the right lower extremity at first, but more recently involving to a less extent the right arm and left leg.

He gives the following history:—

His general health has always been good, but he has always suffered from cold hands and feet.

In July, 1892, whilst kneeling at work, his right knee became locked, and always did so subsequently whenever he knelt down.

In August, 1893, he fell whilst walking from a similar cause. Effusion took place into the joint, which incapacitated him for work for several weeks. He attended St. Bartholomew's Hospital for this and for coldness in the foot and leg, and was found to have loose cartilage in the right knee. The loose cartilage under treatment soon ceased to give trouble, but the sensation of coldness in the leg has increased until it amounts to positive pain, which he thus describes:

"The coldness in my right leg has gradually got worse, with peculiar pains such as aching, smarting, and numbness, all at once and mixed together. If I put my foot to the ground the cold pain is there with aching and numbness, and it gets worse the longer it is on the ground. If I sit with it on the floor it is the same, and like a cold wind blowing on my thigh. After dressing I have to sit with my foot up in front of the fire, and then the cold wind seems on my thigh the same. I cannot walk for more than a few minutes without wanting to sit down and raise

my leg. When the coldness first came on it seemed as though my foot was placed on ice, in the hollow of the foot, but now it is all over the foot, top and bottom alike, and the coldest pain comes from the roots of my toes. The foot and leg always seems as though there was a lot of insects trying to pass each other and cannot, and so cause the pain such as I have described. I am easiest when I can put my foot and leg in a hot bath, or when lying in bed, but I cannot bear anything to touch me on the leg besides the bedclothes, as it seems to stop all circulation and cause numbness and aching."

Since he has been under my care, this account of his symptoms has not varied, except that he has recently complained of sensations of coldness in the right arm, particularly the shoulder, and in the left leg. The right leg is, however, much the more severely affected. He has been unable to work for two years, and has increasing difficulty in getting about.

Present Condition.—He is a strong-looking but loosely-built man. He complains of no symptoms but those mentioned. His thoracic and abdominal organs are sound. His digestion and appetite are good There is nothing abnormal to be seen about the upper extremities. The left lower extremity has some varicose veins. The right lower extremity is flabby. The thigh measures an inch less, and the calf three quarters of an inch less, in circumference than the corresponding portions of the opposite limb. There are no varicose veins and no œdema here. There is now no evidence of loose cartilage in the knee joint. The limb is weak in proportion to the wasting. He uses two sticks in walking for fear of falling, but is able to walk unassisted.

Reflexes.—The knee jerks are present and equal on both sides. He can stand steadily with eyes shut and heels together.

Sensation.—Tactile sensation is perfect, also appreciation of heat and cold, but the application of cold to the right leg is distressing, whilst that of heat is agreeable to him. There is no difference in the temperature of the two limbs. Both are warm to the touch, and he admits that this is so, although the sensation of coldness in the right leg obliges him to wear two or three thick stockings.

Pain.—He recognizes a pin's prick readily and winces. He states that he is unable to feel faradism applied to the right leg by means of a wire brush so readily as he used to do when under this treatment two years ago. He certainly tolerates, without showing signs of pain, a much stronger faradic current than I could endure myself. The electrical reactions are quite normal.

His muscular sense is unaffected. He imitates correctly any attitude in which one leg may be placed, with the other, and describes their positions correctly, his eyes being closed. There are no tender points nor patches of anæsthesia in the course of the nerves of the limbs. His sensations of cold are not confined to the area of distribution of any particular nerves.

His family history is unimportant as regards nervous disorders.

Remarks.

Psychro-æsthesia (derived from the Greek Ψυχρός "cold") is the term used by Silvio (*La Riforma Medica*, February 17th and 18th, 1896) to describe symptoms somewhat resembling those of my patient, but differing from his in the limited area of their extent.

Silvio's case was that of a healthy man, aged 62, who for twenty years had suffered at intervals from a sensation of cold affecting the outer surface of the right thigh. The sensation was induced by standing, and relieved by lying down. He never had pain or anomalous heat sensations in the affected part. Ordinary sensibility seemed normal in the right thigh and elsewhere. Silvio contrasts this condition with that of subjective sensations of heat, which he terms "kauma-æsthesia."

As an instance of the latter, he relates the case of a man, aged 44, who for three years had complained of a burning sensation in the right thigh, at first confined to a limited area, but later involving almost the whole of the thigh, and accompanied by a feeling of deadness in the skin. It occurred intermittently, chiefly in the winter, was relieved by friction or by resting the body on the other leg, or by sitting down. Sensibility to pain and heat was normal, tactile sensibility was slightly weakened. Twenty years previously he had suffered for about a year from a sensation as of drops of water trickling down the inner surface of the right thigh.

Under the title "Meralgia Paræsthetica," cases similar to this last one have been described by W. K. Roth, of Moscow (Berlin, 1895). The painful sensations affected

chiefly the outer side of the thigh, with subjective or objective anæsthesia. The pain was mostly burning in character, and was rendered worse by standing or walking. The paræsthesia and anæsthesia were generally limited to the distribution of the external cutaneous nerve of the thigh, but the sensation of burning sometimes extended beyond this area. Roth regarded the symptoms as due to an affection of the above-named nerve, as their duration (in one case fourteen years) seemed to preclude the idea that they were dependent on an early lesion of the spinal cord.

Cases of paræsthetic meralgia have also been collected by Bernard, of Berlin (*Révue Neurologique de Paris*, Nos. 20 and 23, 1895), and by Escat.

More recently the subject has been discussed in the *New York Journal of Mental and Nervous Disease* (March and April, 1896) by Osler, Hirsch, and Weir Mitchell. Weir Mitchell mentioned patients in whom paræsthesia and pain in the thigh have turned out to be due to unsuspected periosteal disease.

Hirsch had met with a sailor, aged 50, who, after being shipwrecked and suffering from long exposure, as well as having to bear considerable weight on one leg for over an hour, was attacked by a peculiar sensation which he described as "burning cold," over a region the size of the palm of the hand, four inches above the knee, on the outer side of the thigh. He had suffered from syphilis eighteen years previously, and had been addicted to alcohol. Hirsch regards these as the predisposing, and the exposure as the exciting cause of the complaint.

The general opinion on these cases, to which my own bears a certain resemblance, appears to be that they are indications of local mischief. As such, they seem to be akin to those originally described by Weir Mitchell under the name of causalgia ("Injuries of Nerves," p. 159). But in Weir Mitchell's causalgia there is a history of direct injury, usually by gunshot, to the nerves themselves. The peculiar pain was associated with redness or glossiness of the skin, and with thickening of the nerves' trunks above the site of pain.

Similar trophic changes are found in the nerves when causalgia is dependent on central lesions of the brain or cord. In my own case, however, and also apparently in recorded cases of meralgia, there is no evidence of such trophic degeneration of nerves. Causalgia again expresses the type of suffering in Morton's disease (metatarsalgia), and in the similar affection which I have described as "painful toe" (*Lancet*, March 19th, 1892). But in both these complaints a definite local cause exists in the shape of tight boots, which produce displacement of some of the metatarsal or phalangeal bones, which, accordingly, press painfully on the subjacent nerves. In my own case of psychro-æsthesia, and apparently in Silvio's, no such local cause can be discovered. The sensations in my case are not relieved by removal of the boot. Moreover, such an explanation would not account for the extension of the symptoms to the right arm and left leg.

There is no evidence of peripheral neuritis. As to other local causes, I at first thought that the vasor-motor apparatus might be at fault, as in the minor forms of Raynaud's disease; but in Raynaud's disease the numbness, tingling, and coldness complained of, are associated with actual loss of temperature and pallor of the parts affected; whereas my patient has no loss of temperature, local or general, no blanching, and no œdema of the limb. The only physical signs present are slight wasting and slight weakening of the right lower limb. It might be thought that these conditions are dependent upon the loose cartilage from which he has suffered. Yet neither this nor any other local cause can account for the involvement of the right arm and left leg.

It is, therefore, probable that the wasting and weakness, and, perhaps, the loose cartilage also, are indications of general dystrophy resulting from some central nervous conditions, which also affects the right arm and left leg similiarly, but to a less extent than the right lower limb. If central, the lesion may be either cerebral or spinal. If cerebral, one must first ask whether the symptoms are due to a mere psychosis. It must be admitted that his symptoms are mainly subjective. The only objective evi-

dence of hindered sensation is his remarkable tolerance of strong faradic currents, the significance of which is obscure. The pathology of subjective sensations must always be elusive. One is apt to assume that it is non-existent in the absence of physical signs, and to attribute complaints, for which there is little to show, to malingering, hypochondriasis, or hysteria. The patient has nothing to gain by malingering, as he has enough money to live upon, at all events, for the present. His symptoms are not such as would be hit upon by a malingerer. He seems as much troubled by his presumably enforced idleness as by the sensations of which he complains. Hypochondriacs often discover strange symptoms, but they soon appear to tire of them, and invent new sets as the novelty of the old wears off. But this patient's account of himself has never varied during the two years I have known him. He is not, moreover, gloomy or depressed, but takes a semi-humorous view of his condition; being cheerfully apologetic when remedies fail, and also appearing grateful for any attempts to relieve him. This is unlike the ordinary hypochondriac.

The duration and unchanging nature of the symptoms, and the absence of special signs of hysteria, and of the hysterical temperament generally, are against the diagnosis of this affection. It is difficult, therefore, to dismiss the symptoms as imaginary, functional, or feigned.

As regards gross cerebral lesions; only multiple and bilateral affections could involve the sensory tracts of both legs and one arm; and it is extremely improbable that the face, the special sense tract, and also the motor tract, would escape were this the case. There is, moreover, no evidence, as far as I am aware, that the peculiar sensations, of which this patient complains, are ever associated with cerebral disease, either situated in the cortex, or elsewhere. On the other hand, there is abundant evidence that parallel, if not absolutely identical, sensations may be associated with disease of the spinal cord or of its nerve roots. This was well brought out in the discussion on "Dissociated Sensations as a Means of Diagnosis," reported in

the *Journal of Nervous and Mental Disease*, New York, for April, 1896.

Perversion of sensations of heat, cold, pain, and of tactile sensibility, as well as of the muscular sense, may occur in spinal meningitis, whether due to curvature, fracture, or syphilis, and depends on involvement of the spinal nerve roots. Similar perversions of sensations may be due to disease of the spinal cord itself.

Dana, in the above-named discussion, summarised the effects of lesions in various parts in producing differentation of cutaneous sensations, as follows :—These effects were least, he said, in peripheral nerve lesions, increased in spinal root lesions, and again in central spinal lesions, most striking and complete in ponto-bulbar lesions. They became very slight again in cortical lesions.

In the present case, we may exclude lesions of the spinal roots, because they could not produce the wide-spread sensation of cold alone without giving rise to other palpable defects. We have excluded peripheral nerve lesions, and also cerebral affections. There only remains to be considered a lesion of the spinal cord.

His symptoms are not distinctive of disease of the lateral or posterior columns, but it may be that the anterior horns are implicated because of the slight wasting and weakness of the right lower extremity which prevail. The course of thermal sensations in the cord, and also the course of sensations of cold, are unknown. The frequency, however, with which such sensations are altered or annulled, together with those of pain, suggests that the paths of heat, cold and pain may be contiguous.

There is evidence that painful sensations pass through the posterior commissure on their way to the antero-lateral tract on the opposite side.

The disease in which all these sensations are frequently perverted or absent is syringomyelia; and it is possible that involvement of the posterior commissure in this disease may account for the symptoms.

According to Gowers, subjective sensations of heat and cold not unfrequently precede the characteristic symptoms

of syringomyelia. The duration of the symptoms here does not exclude the possibility that they are caused by this disease; for symptoms may be absent altogether, although the conditions of syringomyelia must have existed for many years.

It is usual in cases of syringomyelia for the upper extremities to be affected earlier and more seriously than the lower, doubtless because the site of the disease is usually in the upper part of the cord. But cases have been reported in which the converse order of symptoms prevailed. This may be a case in point; and, accordingly, we may have to deal with syringomyelia, in which the function of the posterior commissure is perverted though not destroyed. If so, the lesion must have commenced low down in the cord and spread upwards; whilst in order to account for the weakness and wasting of the right leg it must be assumed that the anterior horn is involved.

Treatment. — Silvio found that massage and thermo-mineral baths appeared to give relief in his case, but did not cure. In my own case, rest and the actual application of heat give temporary relief. Stimulating embrocations, ointments containing capsicum, and flying blisters applied to the thigh, have been beneficial for a time; electricity, in the form of galvanism and faradism, has been useless. All kinds of nerve sedatives and tonics have been tried in vain.

If the pathology of the case is, as has been suggested, any form of treatment can only be palliative.

NOTE.—The patient, who provides the subject of this paper, was shown at a meeting of the Harveian Society of London, on May 21, 1896. Of the speakers who discussed the case, Dr. T. Savill was inclined to agree that the symptoms were due to syringomyelia. Dr. Cagney suggested that some obscure lesion of the cerebral cortex might be the cause.

STUDIES ON THE NEUROGLIA.[1]

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No so-called interstitial tissue has given rise to so much discussion as the neuroglia. The most far-reaching advances in our knowledge of it are, undoubtedly, the discovery of its fibro-cellular structure, and the recognition of its epiblastic origin. Upon this latter point, as far as it concerns pathology, we would here—for want of a better place—say a few words; and not for reason of any novelty, but because it does not as yet appear to enjoy general recognition. We refer to the classification of the gliomata, and to some incongruity of nomenclature still in vogue. If neuroglia is an epiblastic structure, then it follows that tumours composed of it are epiblastic also, and should not be classed under the mesoblastic growths, as some recent text-books[2] still do. Further, it follows that the term "glio-sarcoma," if it has any legitimate meaning at all, is a misnomer. This term is, at present, applied to tumours composed of cells which are supposed to possess characters common to glioma and sarcoma. Such a use of the word is, of course, radically wrong; and should only be employed to denote a simultaneous growth of epiblastic and mesoblastic elements side by side in a neoplasm—an occurrence, the possibility of which cannot be denied, however improbable.

[1] Being, in part, the substance of a paper read at the Annual Meeting of the British Medical Association at Carlisle, July, 1896.

[2] *E.g.*, Hamilton's "Text-book of Pathology," part i., p. 443.

The rapid advance of our knowledge of the subject during the last decade we owe to Golgi's method. Applied to the neuroglia, the silver impregnation method has exhibited a wealth of fibres and cells where nerve-elements were once supposed mainly to exist; and has proved the neuroglial nature of structures, such as the septa of the spinal cord, which were once believed to be true connective tissue. But it is not our present object to give in detail the results obtained by this stain; they can now be found in every recent text-book of anatomy. It will suffice if we briefly enumerate some of the more important points as they concern us here. According to Golgi, Cajal, Kölliker, and the host of other observers, the neuroglia is said to consist of cells possessing processes varying in number, length, and thickness; and while the processes of some cells are smooth and undivided, others are rough, bushy, and branched. Transitional forms have also been noted. It would appear that the ependyma-cells lining the central canal are the parent stock from which the glia cells are primarily descended. In the amphioxus these central ependyma-cells compose the sole supporting structure; in the ascending scale of the vertebrata, cells can be seen which betray, to a greater or less extent, the original idea embodied in the ependyma-cell—that is to say, the pyriform body and the strong main process passing towards the periphery, actual or virtual. By a virtual periphery we mean, for example, the blood vessels, which, entering the nervous organ, invaginate the periphery, and with it the neuroglia. Additional prolongations, however, put in their appearance; and, in the most advanced form, the original main fibre has dwindled and decayed, allowing the secondary branches to constitute the principal feature of the adult neuroglia cell. This process of evolution, as exhibited in the various orders and genera of vertebrated animals, is passed through with leaps and bounds, and, probably, with short cuts, in the development of the human nervous system. Golgi's method, with its various modifications, has been the sole means by which these conditions have been studied in the lower animals, and,

till recently, in the human being also. But before the close of last year another method saw publication. We refer to Weigert's neuroglia stain, a detailed account of which appeared in November last.[1] It is an intricate, and still somewhat uncertain, method. Our failures with it have been many—partly for the reason given; partly because suitable material is scanty, as a longer period than twelve hours after death lessens considerably the chance of obtaining even a fairly satisfactory result, if it does not ruin it altogether. Some parts of the nervous system, too, seem to resist the stain more than others, *e.g.*, the cerebral cortex; while some pathological conditions—above all, œdema—would appear to render satisfactory results particularly difficult to obtain. Our researches with this method are, therefore, still somewhat fragmentary; and it is our intention to give in this paper not any new results of normal anatomy, but to consider various points that have suggested themselves to us in the study of the pathological brain and spinal cord.

The effect of the method in the case of man—for to the adult human brain alone is it applicable—is briefly this: it dyes neuroglia fibres a fine blue; similarly all nuclei; it does not, however, stain fibrous tissue;[2] while the various nervous structures either remain unstained, or receive a delicate yellow tint. The advantages to the pathologist are that it is less capricious than Golgi's method, and that it permits the study of the distribution of the fully-formed neuroglia. Sections treated by this method present appearances which tend greatly to modify existing views. Hitherto we have believed, with Golgi, that in the human adult the neuroglia is purely a cellular structure; that its multitudinous fibres are not fibres in the strictest sense of the term, but cell processes. Weigert, however, on the strength of his method, comes to the conclusion that things are not so simple. He finds that the blue fibres shown in the preparations are no longer cell processes;

[1] C. Weigert, "Beiträge zur Kenntniss der normalen menschlichen Neuroglia," November, 1895, Frankfort-on-Main: M. Diesterweg.

[2] If one stage be omitted.

nowhere can fibres be seen joining the cell protoplasm; everywhere they pass over, under, and around the nucleus. He, therefore, concludes that here, as in the true connective tissue, the cell processes have become differentiated from the cell—have become separated from it. These, and only these fibres, are stained by the new method in the healthy brain or cord. But it would be an error to suppose that the human neuroglia can exist in no other form. For we reason that, as in some lower vertebrate, glia cells persist in several shapes characteristic of various stages of their evolution, so may neuroglia elements in man stop short of this terminal condition, in which fibre and cell cease to be a corporate whole. This intersection of fibres round the nucleus has still a superficial resemblance to a spider-cell; and if we adopt the term "astrocyte," as a synonym for "spider-cell," we may call these imitation spider-cells "pseudo-astrocytes."

This view of Weigert's has met with some opposition on technical grounds, which need not, however, concern us here; ample support can, on the other hand, be obtained from certain appearances to be noted under some pathological conditions, as, for instance, in the cerebral cortex of a general paralytic. What the most careful examination of healthy brain tissue fails to find—viz., a fibre stained blue like the rest, but still an integral part of the cell body—can frequently be seen in these morbid conditions. We see a number of fibres passing towards the body of one of these spider-cells; but as they approach it their tint gradually fades, merging gently into that of the cell protoplasm. All stages of this process can be observed—from that in which the fibre has taken up the faintest possible blue colouration to that in which it is difficult to tell whether the fibre is still part of the cell or not. It seems to have escaped Weigert's opponents that while the new statement of things runs counter to the simplicity of Golgi's theory, on the one hand, it tends to clear a difficulty on the other. We refer to the question of the multiplication of neuroglia cells. It had for some time been suspected that sclerosis—for instance, in the tracts of the cord—was

due to a proliferation of the supporting neuroglia; Weigert himself had demonstrated, by means of a method of his own, the presence of karyokinetic figures in such cases; and the difficulty naturally encountered was this: what becomes of the long and numerous branches of these cells in the process of division? No satisfactory explanation has ever, we believe, been offered on this point; refuge has usually been taken in assuming the existence of so-called abortive neuroglia cells—masses of protoplasm that have lain dormant from an early period of intra-uterine life. The new interpretation simplifies matters somewhat; for there can now be no difficulty in picturing the proliferation of cells that have previously parted with their processes. Weigert's stain, moreover, sets any doubts that may still have existed as to the glial nature of all sclerotic and cicatricial formations within the central nervous system at rest. Disseminated sclerosis, all so-called system diseases, and all reparative processes consist essentially of newly-formed neuroglia. If such a lesion be examined—and, for the sake of simplicity, let us take a sclerosis of the lateral or posterior columns of the cord—two changes in particular obtrude themselves. We find, on transverse section, that the neuroglia fibres are also cut transversely, appearing as fine blue dots between the nerve tubules; in other words, neuroglia fibre and nerve fibre run practically parallel to each other, the former having developed in the direction of least resistance. This is one feature; the other is, that the septa and trabeculæ are thickened, and that the perivascular neuroglia is denser and more plentiful than normal. In advanced chronic cases, not only do the septa become more bulky, but the subpial layer of neuroglia also, from which they take their origin, increases in thickness, even if a healthy tract intervenes. The crossed pyramidal tract, for example, may degenerate and sclerose, and the corresponding stretch of subpial neuroglia thicken; while the direct cerebellar tract between them remains apparently healthy. The most plausible explanation of these facts that we could think of is the following, based upon the principle that the proliferated cells, upon which a reparative process depends, tend to conform in shape to early types.

The type, in the present instance, is that of the modified ependyma cell, the chief characteristic of which is a main process, thick and strong, stretching to the actual or virtual periphery, and attaching itself there. A similar tendency is manifested by these newly-formed glia cells. Among their fibres one, or perhaps two, can be seen, coarser and thicker than the rest, passing to the periphery, which is usually represented by a blood vessel. Some of these branches are undoubtedly short, but their true length cannot in all instances be determined, for their course is tortuous, rarely continuing for any distance in the plane of section. It is these cells, with their strong "vascular processes," that have been dubbed "scavenger cells" by Bevan Lewis. In accordance with this explanation of ours is the interesting fact that the nuclei (or cells) within the subpial layer of neuroglia are altogether disproportionately few in number compared with the wealth of fibre; the natural inference being that the parent cells of most of these fibres lie elsewhere.

But the possibilities regarding the mechanism of a sclerosis are not yet exhausted. Weigert's new method does not, as we have already said, pretend to stain every fibre; there are regions in which we should, *a priori*, expect to find neuroglia, and in which a true sclerosis is known not unfrequently to occur. Such an area is the deepest layer of the cerebral cortex; and while scarcely a single blue fibre can be found in it in health, yet, under certain morbid conditions, a felt-work of neuroglia becomes visible. Fresh sections, stained with aniline blue-black, reveal, during the earlier stages of disease in this layer, a comparative richness in "scavenger cells," when contrasted with the poverty of the more superficial strata. A process of immigration from the upper to the deeper layers is, therefore, improbable; the scavenger cells, and the fibres derived from them, must have developed *in loco*, and from cells which had not attained to the final stage of fibrillation. Thus the old question again arises—if richly-branched astrocytes, or spider-cells, divide and multiply, what becomes of their processes? Investigations on this point

are still a desideratum; we can only offer an opinion—and we do so with some diffidence—to the effect that, prior to their division, the astrocytes may possibly become differentiated into free fibres and free cell body. It is from comparative pathology, we think, that light may be expected. The normal anatomy of the purely nervous structures is already much indebted to this branch of science; but it is a remarkable fact that we know nothing, or next to nothing, of the effects produced upon the neuroglia by interference with the nervous centres of the lower vertebrates. What goes on, for instance, during reparative processes following an ordinary lesion; or how does the glia behave when the development of some particular system—say the central optic ganglia and tract—is experimentally arrested? We are in possession of numerous sections from the brain of a cat, which had been subjected to enucleation of one eye soon after birth; in none of them can any excessive development of true fibrous tissue be discovered. But in dogs, rabbits, and the like, the conditions are too complicated; we must descend still lower, and make animals in which the glia elements still bear a close resemblance to their ancestral cells—such as the batrachia, fishes, and, perhaps, birds—the subjects of our investigation. A trustworthy method, however, is still wanted for such a study, as Weigert's stain is as yet unavailable for comparative pathology. So far, then, our knowledge of the morbid anatomy of the neuroglia is confined to this fully developed sclerotic tissue.

We recognise that the process of fibrillation is not a confused, hap-hazard one, but follows certain laws; the felt-work formed is not so intricate, but that one or two fundamental principles cannot be traced. One is that the fibres develop in the direction of least resistance; and another that, to a certain extent, the newly-formed neuroglia follows in the path of the old. Examples of this are seen in the thickening of the subpial zone, and in perivascular sclerosis; but the pictures obtained in cases of superficial cerebellar sclerosis are even more instructive. In the outermost, or molecular, layer of the cerebellar

cortex, the neuroglia is normally represented by somewhat sparsely scattered fibres, passing radially towards the pia, while their cells of origin lie at the level of Purkinje's cells. With a slight and superficial lesion, this radial distribution becomes exaggerated, the fibres are crowded closer together, and appear decidedly thicker; but the one direction —outwards towards the pia mater, is strictly maintained. But given a severer case, then a further bundle of fibres makes its appearance at right angles to the last, running parallel to the surface of the lamina. Where these originate we are not prepared to say; but their direction is evidently that of certain axis-cylinder processes, derived from the small nerve cells of the molecular layer.

We would here like to call attention to an important point that has already been hinted at in some of the foregoing remarks; it is this: that an increase of neuroglia over a certain area is not necessarily tantamount to a primary nerve lesion of equal extent. As an example of this we would again instance the thickening of the trabeculæ passing through the direct cerebellar tract in some cases of lateral sclerosis; or, better still, to the cerebellar cortex, in which the radial fibres of the molecular layer undergo an increase throughout the whole thickness of that layer, even if the lesion is superficial. This is based on the principle that sclerosis following a given primary nerve lesion is due more to the activity of those neuroglia cells from which the pre-existing glia fibres of the affected area had originated, than to an influx of wandering cells—if, indeed, such a thing exists. The more complex the ramifications of axis-cylinders and protoplasmic processes in a given region, the more intricate and tangled is the neuroglial felt-work; it is from the simple anatomical conditions only that we can hope to unravel the principles of its development.

Defective as is our knowledge of the normal and pathological anatomy, that of the functions of the neuroglia is even more so. It is usually described as a supporting structure; and there is nothing to oppose this view. In one particular instance, indeed, as Weigert and Andriezen have pointed out, this function appears almost obvious—

namely, in the case of the blood vessels. These are notoriously poor in adventitia, and the glia fibres passing obliquely along, and also across them, are well calculated to prevent "undue expansion," as Andriezen puts it. But it would seem to us that they may exceed this function under some conditions of perivascular sclerosis, and make the vessels too rigid, thus impeding the influx of blood to a greater or lesser area. The effects of such defective nutrition would be greatest where the arteries are terminal ones, as in the cerebral cortex; and this may perhaps be *one* reason for the merciless, ever-spreading course of some chronic diseases.

That such a wonderfully complex system should subserve no other function but that of a supporting structure, gives rise to a feeling akin to disappointment. The numerous theories on the subject are evidence of this; but, it must be confessed, we have got little further than theory.

In its first beginnings the glia possibly directs the outward passage, and, to some extent, the grouping of the neuroblasts. But from that period till near the end of intra-uterine life, its function, if other than supporting, is a mystery; perhaps it lies latent, preparing for its subsequent duties. After birth it would appear to assume, to some extent, the *rôle* of an insulator. This is the opinion put forward by Sala y Pons and others, who based it on the apparently richer development of neuroglia in the white matter than in the grey. Weigert has demonstrated that Golgi's stain, with which these men worked, is anything but a sure guide in questions of quantitative distribution; and that, as a matter of fact, the grey matter is the richer of the two. Yet Sala's theory can stand. What necessity is there for such an insulator in the white substance where the medullary sheaths already serve that purpose? But in the grey matter things are different; here the axis-cylinders, and their collateral branches, break up into a multitude of naked terminal ramifications, interlacing among each other, and around the nerve cells. Here an insulator is obviously needed; and the neuroglia may well serve this end. Hence, Sala y Pons is probably right in his conclusion, though his premises are faulty.

As a matter of curiosity, we may mention a theory, propounded by Rohde, for the invertebrates: that neuroglia fibres enter the nerve cells and supply them with nutriment. Such a condition has never, as far as we know, been observed in the vertebrata; and much might be said against it.

Diametrically opposed to it, however, is a theory, which appears to enjoy some popularity, and to which some reference is due: that is the "scavenger cell" theory of Bevan Lewis. According to this view, the glia cells possess not only the function of removing detritus, but also the power of attacking diseased nerve fibres and cells. In spite of the somewhat tempting nature of this theory, we cannot but think that shape and name of the cells in question have had their share in suggesting it. It is a theory as easily advanced as it is difficult to prove or disprove. No one, we think, has ever seen a spider cell in such relation to a nerve cell as to prove conclusively that it was attacking the latter. But to argue the function of a cell from its shape savours of the antique, and cannot be taken seriously. The doubts and uncertainties that still surround the lymphatics of the nervous system; the constant proliferation of neuroglia cells in all destructive lesions of that system; and the possession by these cells of a thick process attaching itself to a vessel wall, are the only grounds on which the theory can hope to stand. After all, it is simply a question of interpretation, and of the greater or lesser probability of one view over another. Twenty years ago Weigert developed the principle that diminished mutual resistance between tissues is followed by a process of proliferation. And the neuroglia is no exception; loss of power of resistance on the part of the nervous structures is followed by a corresponding increase of the neuroglia. Obliteration of the central canal furnishes an instructive example; the cubical cells becoming loosened, and packing the lumen of the canal, are followed by an increase and invasion of neuroglia—fibres from the latter passing between them in all directions. Yet these cubical cells are not attacked by spider cells, though such "scavengers" must

precede the stage of fibrillation. It may further be shown that a dead nerve cell is not necessarily removed, in spite of the presence of large numbers of spider cells; it may calcify, as is so often seen in sclerosis of the hippocampal gyrus in epilepsy. The distribution of the neuroglia is also a point of some moment; for there exists no parallelism between the amount of nerve tissue and neuroglia: witness the wealth of the latter in the anterior horns of the spinal cord, and its comparative scarcity in the motor cortex. In some of the lower animals, such as the calf, scavenger cells, distinguishable in no way from those seen in man under pathological conditions, are found in some abundance in the cerebral cortex. The deduction has been drawn that domestication has a deleterious influence on the nervous system, causing degeneration of its elements, though other evidence of such degeneration is wanting—a good example of what a too dogged adherence to a theory, in the face of facts, can lead to. Our own belief is that the so-called "scavenger cell" is but a form characterising the neuroglia cell in one period of its life-history; and that, in any proliferative process, this earlier stage must, in the natural course of things, be returned to, before fibrillation, as the final result, can be attained. It is true we have no new theory to offer; but if we cannot advance anything ourselves, we may, at least, sweep the path a little for what is yet to come.

NOTES ON GRANULES.

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In a paper on the "Chrome-Silver Method," published in Brain, part lxxiii., p. 1, Spring Number, 1896, I described some small cells, which I found in the cerebellum, under the name of "granules with centripetal axis-cylinder processes." At the time I had found them only in two-day and four-day-old rats, but I have since found them in older rats, and am satisfied that they are not Golgi-cells in an embryonic condition, but structures which retain the same form throughout life. I find these cells near the summits of the folia and only in the deeper stratum of the granule layer. Their transverse diameter ranges from 10 μ to 15 μ (in the rat); their diameter is therefore about the same as that of an ordinary cerebellar granule.

Their axis-cylinders run parallel with the fibres of the arbor vitæ; often they traverse the granule layer for a very considerable distance before joining the fibres. In one case the axis-cylinder, after running towards the base of the folium for a considerable distance, loops upon itself, and returns towards the apex; owing to the irregularity of their course, it is often difficult to determine the destination of the axis-cylinders. Usually destitute of collaterals or side branches, they occasionally give off side branches which turn towards the molecular layer. Most of the cells are carrot-shaped, the horn of the carrot being often forked. Some of the cells are fusiform, a long process tapering from either end; the axis-cylinder then comes off from one of these two processes.

The centripetal course of the axis-cylinder distinguishes these cells from the type of cell hitherto described as "granules" of the cerebellum in so marked a way that it may be desirable to describe them under a different name. Since Golgi was the first to describe the large nerve cells of the granule layer, it seems appropriate to name these cells "small Golgi-cells," although they are quite unlike any cells described by the Italian anatomist.

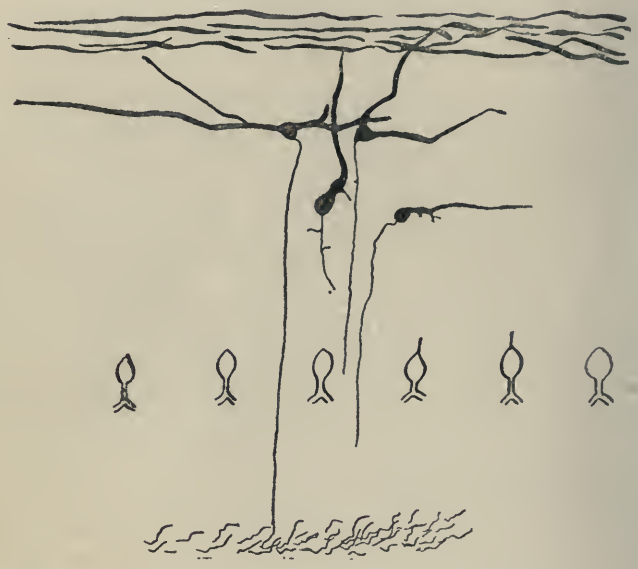

Fig. 1.

A group of carrot-shaped granules of the cerebellum with centrifugal axis-cylinder processes. The fibres of the arbor vitæ are seen at the top of the sketch. The cells of Purkinje are marked in outline. The axis-cylinders of the granules pass between the cells of Purkinje to bifurcate in the molecular layer. Section slightly oblique to the long axis of the folium. Kitten three weeks old.

Since finding the cells just described as small Golgi-cells, I have discovered, both in the cat and in the rat, cells almost identical with them in form (fig. 1), but provided with an axis-cylinder process, which bifurcates in the molecular layer. In the particular specimens examined, these

cells are slightly smaller than those (found in other subjects) with centripetal axis-cylinders, but otherwise they resemble them very closely. Their transverse diameter is 8 or 9 μ in a three-week-old kitten. They are carrot-shaped, the horn often forked, or double, or spindle-shaped, with the ends of the spindle of very considerable length. The dendrites (if we may apply this term to the tapering processes of these granules) usually incline towards the fibres of the arbor vitæ. The axis-cylinder is exceedingly delicate, comes off from the end or side of the cell, and makes its way between the cells of Purkinje to the molecular layer. Its course is almost straight, though undulating. Within the molecular layer the axis-cylinder process bifurcates after the manner described for the axis-cylinder processes of ordinary cerebellar granules by Ramón y Cajal and Pedro Ramón.

Dogiel[1] has recently described the cells of the granular layer as he finds them in the dove, with the aid of the methylene-blue method. He divides them into two classes —the "small cells of Golgi" and the "large cells of Golgi." The small cells he figures with more slender processes than the chrome-silver method shows them to possess. The cells near the cells of Purkinje are not unlike the granules as they ordinarily appear. The more deeply-placed cells are larger. As Dogiel gives no measurements and makes no statement on the subject, it is difficult to tell whether he regards these small cells of Golgi as identical with the "granules" which Golgi describes and figures—the well-known round granule with three or four arms, ending in bent claws or irregular patches—or as cells of a kind not hitherto described. In either case it appears to me to be a mistake to call a small cell with a centrifugal axis-cylinder a "Golgi-cell." These small cells are the "granules" of the cerebellum, of which the nervous nature was proved by Ramón y Cajal, when he discovered their axis-cylinders. The term "granule" is an extremely convenient term for cells of this type. It is a pity to confuse them with the huge cells with centripetal axis-cylinders, which were first described by Golgi, and are

[1] Dogiel, "Die Nervenelemente im Kleinhirne der Vögel und Saügethiere," *Archiv f. Mikr. Anat.*, xxxvii., 4, July, 1896.

now very properly known by his name. Ramón y Cajal makes a similar protest (*Rev. Trim. Micr.*, I., p. 176).

Granules of the Olfactory Bulb.—It was the object of my investigation into the chrome-silver method to examine

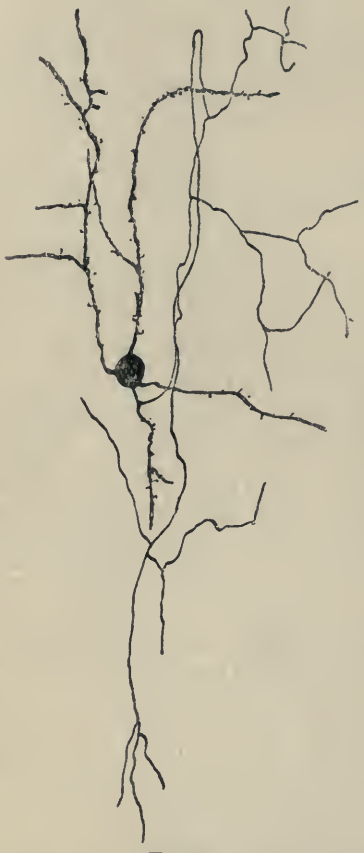

Fig. 2.

Larger granule of the olfactory bulb. The axis-cylinder comes off from a central dendrite, courses peripherally almost to the mitral cells, and then folds back towards the bulbus ventriculi. It gives off six collaterals. Guinea pig, six weeks to two months old (half grown).

its credentials with a view to judging how far we can trust it as a witness on whose authority we are justified in accepting the conclusions, as to the plan of structure of the nervous system, of those who have chiefly worked with this method—

conclusions which are completely subversive of all that had been believed hitherto. Among these innovations was the introduction of the idea of the existence of "amacrine cells," *i.e.*, cells destitute of axis-cylinders. They are described particularly in the retina and olfactory bulb. The bulb seemed the easier situation in which to test their existence. The chrome-silver method ought not to be appealed to to prove a negative, but when it gives a positive result the picture is, fortunately, so clear as to admit of no dispute. After a long search I found the axis-cylinders of the granules, which I described and illustrated by a photograph in the paper referred to. These were granules of the form which I am about to describe as type 1, for I now find the granules of the olfactory bulb to be of two types :—

(1) The larger granules, of which fig. 2 is an illustration. The cell figured has a diameter of 14 μ. Its axis-cylinder turns at first towards the glomeruli, and then, looping upon itself, runs for a great distance in a central direction, giving off side branches. The total length of the traject displayed in this section is ·75 mm. The cell lies in the midst of the granule layer, and is best—as it seems to me—termed a "granule," for it contrasts in a marked way with the large nerve cells of the bulb, especially with the very big cells which I have termed "bracket cells." It is equally distinct from the cells of Golgi's type II. as figured by Golgi, Ramón y Cajal and Van Gehuchten. Although its dendrites are rather larger and more numerous than those of the granules of the second class, they resemble them in disposition, passing outwards as far as the mitral cells, and also inwards towards the ventriculus bulbi. Many other cells of the same kind are to be seen in the preparation; their axis-cylinders being invisible, no histologist would hesitate to term them "granules."

(2) The bulb also contains granules, which give off centripetal axis-cylinder processes of excessive tenuity (fig. 3). These axis-cylinders are very rarely coloured by the chrome-silver method, and I have not yet obtained a specimen with the axis-cylinder coloured and the dendrites well displayed at the same time. At present, therefore, I cannot say how

many of the granules usually seen belong to the first class and how many to the second. The granules of the bulb present every gradation in size, from the minute granules (8 μ), in which I have seen the straight centripetal axis-cylinders, to the larger granules (14 μ), with tortuous axis-cylinders.

Fig. 3.

Three of the smaller granules of the olfactory bulb with centripetal axis-cylinder processes, and two mitral cells. Hedgehog seven days old.

The form of the granules of the bulb, the disposition of their dendrites, their thorns and their reaction to chrome-silver and other stains would have appeared to me to mark them as indubitably nervous, even if I had not discovered their axis-cylinders.

NOTE ON "THORNS," AND A THEORY OF THE CONSTITUTION OF GREY MATTER.

BY ALEX HILL, M.A., M.D.

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THE minute lateral branches born by the dendrites of all classes of nerve cells in the central nervous system were termed "thorns" by Ramón y Cajal. In their typical form they appear, in preparations made with the chrome-silver method, as minute stalks, each bearing at its end a black bead. Hence they have been called "gemmules," and the bead at the end (the "contact granule") has been supposed to be a dot of naked protoplasm, by means of which the dendrite establishes contact with nerve fibres which run parallel to itself.

Semi Meyer[1] has questioned the existence of thorns as real structures on the ground that he could not find them in preparations made by subcutaneous injection of methylene-blue. Kölliker[2] regards them as embryonic structures not to be found in the adult unless they appear as artifacts. Ramon y Cajal[3] has, however, shown that they can be displayed by methylene-blue staining both in the young animal and in the adult. He concludes that, since they are displayed by three such diverse methods as those of Golgi, of Cox, and of Ehrlich, there can be no doubt as to their existence.

[1] S. Meyer. Die subcutane Methylenblauinjection, ein Mittel zur Darstellung der Elemente des Centralnervensystems. *Archiv f. mikr. Anat.*, xlvi., 1895; und über eine Verbindungsweise der Neuronen *ibid*, xlvii., 1896.

[2] Kölliker. Handbuch der Gewebelehre des Menschen. Sixth edit., vol. ii., pp. 647, 755, &c., 1896.

[3] Ramón y Cajal. Las Espinas Colaterales de las Células del Cerebro teñidas por el Azul de Metileno. Revista Trimestral Micrográphica, vol. i., p. 123.

I also find them in their typical form in nine preparations out of every ten, but I have nevertheless come to the conclusion that they are structures which are only partially revealed by either the chrome-silver or the methylene-blue, and that the typical form of a rod with a dot at the end is due to *post-mortem* change in the tissue.

I have examined the thorns in the brains of a large number of animals, young and old, all healthy, and all killed with chloroform, but hardened in various ways; and I find

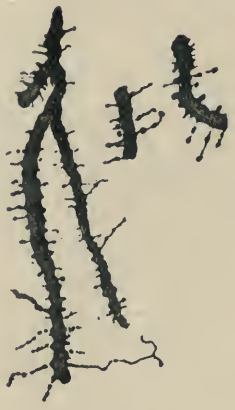

FIG. 4.

Protoplasmic processes of pyramids of cortex cerebri showing thorns prolonged into filaments. Adult hedgehog. Blood vessels washed out with salt solution, dilated with 1 per. cent. lactic acid, and then injected with 4 per cent. formaline. Hardened in bichromate of potassium and formaline.

so great a variation in their appearance that I have come to the conclusion that they are organic structures which are not shown in their entirety by the chrome-silver method.

(1) They may be totally absent. I first noticed this in a brain injected through the aorta with warm bichromate of potassium and osmic acid solution, after washing out the blood-vessels with salt solution, and their dilation with 1 per cent. of lactic acid. They are present in certain other preparations injected in this way. I cannot, therefore, attribute their absence to the method. It is, however, very difficult to make sure that the irritating osmic mixture has penetrated all parts of the brain.

(2) When present they vary much in length, from less than 1 μ to 8 or 10 μ. The longest are found on the granules of the olfactory bulb.

(3) They present a considerable variety in form, the typical stalk and dot giving place to a filament with two or three dots on its course, or the filaments being invisible and

FIG. 5.

Cell of nucleus lenticularis. Dendrites bearing filaments in place of thorns. Adult hedgehog. Blood vessels washed out with normal salt-solution, dilated with 1 per cent. lactic acid, and then injected with 4 per cent. formaline. Hardened in bichromate of potassium and osmic acid.

the dots appearing alone, in a parallel row on each side of the dendrite.

(4) In certain specimens, as shown in figs. 4, 5 and 6, the thorns are replaced by filaments of varying length, which appear to be in direct continuity with the cell or its dendrites.

It is the main trunk and the large branches of the dendrites which usually carry lateral filaments in place of

thorns. The filaments are best seen (*a*) on the apical dendrites of large cortical pyramids, near to the cell, or (*b*) on the centripetal (or apical) dendrites of the double pyramids (giant pyramids) of the subiculum cornu ammonis. Two difficulties at once present themselves. (1) When the lateral filaments are not seen the trunk of the dendrite usually appears smooth and devoid of thorns. (2) In both the situations named (*a* and *b*) a vast quantity of tangential fibres cross the dendrites—in the cortex, the fibres of the laminæ

FIG. 6.

(1) A pyramid. (2) A cell of the nucleus lenticularis. Same animal and same method of preparation as 5.

medullares; in the cornu ammonis, the remarkable deep layer of "mossy" or rosette fibres. It is open to us, therefore, to reject the continuity of the dendrites and the lateral filaments on the plea that it is a delusive appearance, an instance of attractive staining, the colouration of heterologous and completely detached structures in the neighbourhood of coloured elements—a phenomenon with which we are not unacquainted when working with the chrome-silver method. On the other hand, the continuity is unmistakable, if we may trust

the method and the microscope, although we have to bear the reflection constantly in mind that it is impossible, when examining block structures, to distinguish between continuity and juxtaposition. It seems reasonable, however, to put the most obvious interpretation upon the preparations, and to accept their evidence that under certain conditions of staining the thorns may be replaced by filaments. As throwing light upon the connection of the thorn filaments with the trunk of the dendrite, it may be pointed out, that although this part of the dendrite is usually thornless, it has frequently a warty appearance. It may be that the filaments connected with the trunk of the dendrite are coarser than those connected with its twigs. Possibly they are derived from tangential fibres. The mossy fibres appear to be non-medullated. What becomes of the group of filaments given off at each rosette? No method of colouration has, as yet, shown their destination. They are usually figured as thick, short, and blunt, but often they are filamentous. Probably this is another illustration of the varicose accumulation of cell plasm, about to be given as an explanation of the dots at the ends of thorns.

These variations in form—taken in conjunction with the fact that in many mis-stained specimens the cell outline is invisible, owing to the mycelium of filaments by which it is surrounded, which makes it resemble a burr on a rose bush rather than the ordinarily well-defined nerve cell—lead me to believe that A THORN IS REALLY THE CELL-END OF AN UNSTAINABLE NERVE-FILAMENT SURROUNDED BY A FILM OF STAINING CELL-PLASM.

Nerve cells appear to me to consist, anatomically, of two substances, (1) the non-staining filaments and (2) the soft cell substance in which the filaments are embedded. This was the view of Max Schultze. It has been recently endorsed by Flemming, Rohde, and others. For the purposes of this paper, I will term the two constituents simply nerve fibrils and cell plasm, since at the present time I desire to express no opinion as to whether the fibrils are hollow or solid, or as to the constitution of the cell plasm. In the axis-cylinder the nerve fibrils are embedded in cell

plasm which is invested by some kind of sheath, hence the sharp outline of the axis-cylinder, by which it is always distinguished. The protoplasmic processes of the cell are naked.

Nerve fibrils brought to the association fields in grey matter are invested for a certain distance with the axis-cylinder sheath. Eventually the fibrils separate from one another, lose their sheaths, and, since they are not stained (by the chrome-silver or by the methylene-blue method), they become invisible in preparations made with the aid of these methods. They again come into view when they join the dendrites of nerve cells, owing to the overflow of the cell-plasm from the naked dendrite along the fibril. This portion of the fibril to which cell plasm adheres is the "thorn." Within the cell they are to be seen as filaments which traverse its substance on their road to its axis-cylinder process, by which they are collected into a bundle. It goes without saying that this transference of nerve fibrils occurs between heterologous, and not between homologous, neurons.

Cell bodies are often seen to give off filaments. It appears that the fibrils of afferent nerves enter the body of the cell as well as its processes. Probably the appearance of many forms of "basket" endings, described by Cajal, Held, Meyer and others, in which a cell body is grasped by thick, blunt, irregular fingers, is due to the varicose accumulation of cell plasm upon the brush of terminal fibrils into which the last branches of afferent nerves divide.

If my interpretation of the thorns is the right one, we are brought back to the theory of Gerlach, with certain modifications introduced by recent discoveries. It is tempting to speculate as to the mode of working of a central nervous system thus constituted, but in the present state of our knowledge, all hypotheses are mere speculations. It appears to me probable that the presence around the fibril of a film of cell plasm is necessary for the conduction of impulses. Possibly, the opening or closing of a nerve path depends upon the greater or less extension of this plasm film around the filament; an open reflex path being one in

which the film of cell plasm is permanently extended; inhibition being due to the withdrawal of the plasm into the cell.

The need which the psychologist constantly observes of a condition of "attention" on the part of an association field, if an afferent impulse is to produce its full effect, would appear to be due to the fact that the impulse (or rather sequence of impulses, for I imagine that all impulses are vibratory) has, on reaching the cell, to induce the overflow along the fibril of the cell plasm which favours its passage. One who adopts this theory of continuity, might easily amuse himself in formulating hypotheses which would constitute a complete system of nerve physiology; but it is hardly worth while until the anatomical basis of the theory has been established.

THE MUSCLE-SPINDLE[1] UNDER PATHOLOGICAL CONDITIONS.

BY FRED. E. BATTEN, M.D.

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ALTHOUGH the existence of the spindle in muscle has long been known, and a considerable amount of literature has been accumulated by continental writers, yet until a publication by Sherrington (30) in the *Journal of Physiology*, the subject had received but small attention in England. Beale (2), indeed, in 1862, gave an accurate description of the spindle, but regarded it as muscle and nerve in the course of development. It is my purpose, therefore, in the following paper :—

(I.) To give a short account of the work already done on the muscle-spindle.

(II.) To enumerate the various views that have been held with regard to the origin and function of the muscle-spindle.

(III.) To give a short account of the methods I have found to be useful.

(IV.) To give a description of the muscle-spindle as found in man.

(V.) To demonstrate the condition of the muscle-spindle under the following pathological conditions :—

(1) Infantile paralysis.
(2) Tabes.
(3) Myopathy.
(4) Progressive muscular atrophy.
(5) Peripheral neuritis.
(6) Injury to the Brachial plexus.
(7) After section of the sciatic nerve in cats.

[1] Synonyms Muskelknöspen, umschnürte Bündel, Faisceaux neuro-musculaires, Neuritis fascians, Neuro-musculaere Stämmchen.

(I.) History.

In 1843, Miescher (21) described, in the abdominal muscles of a house mouse, bodies to which he gave the name of *Schläuchen*. V. Siebold (31) figures these in a paper published in 1853, and states that the same bodies have been found in rats. Miescher thought they were pathological or a parasite, a view which v. Siebold, however, negatives. It seems to me not improbable that these were bodies which one now knows as muscle-spindles.

In 1861, Weismann (37), working with the muscle of frogs, described very fine muscle fibres in groups of six, eight, ten, or more. The diameter of these fibres varies considerably. They are bound together by a cord about the centre of their course, and, indeed, appear to be enveloped in a dull granular substance, through which one can but indistinctly trace each fibre; these fibres are equal in length and reach from tendon to tendon. Weismann considers these fibres as a stage in the development of muscle.

In 1862, Kölliker (18) described his *Muskelknöspen* which he found in the muscle of frogs, and he believed them to be muscle in the process of division. In his Croonian Lecture, 1862 (18), he says that though at first sight these bodies appear to be of the nature of tactile corpuscles or terminal nerve bulbs, they do not really belong to that class of body. They are nodular swellings marked by a profusion of rather roundish nuclei, and receiving a single very thick nerve fibre loosely surrounded by its comparatively wide membranous sheath. On further examination it is found that the nerve fibre, on reaching the nodules of the muscle, is wound up into a coil, in the meantime undergoing repeated division, and it may be seen to enter the muscle fibre.

On careful examination, however, it is found that the apparently simple muscular fibre is really a small bundle of three to seven fibres, and that the penetrating nerve fibres pass between these muscle fibres. Simultaneously with the increase of its terminal fibres, the dark-bordered parent nerve fibre doubtless augments in thickness, which explains the fact, otherwise difficult to understand, that it is invariably of

much larger size than the nerve fibres proceeding to the other muscular fibres.

In 1889 (18), Kölliker had studied the muscle-spindle not only in frogs, but also in rabbits and man, and after discussing the various views held with regard to these bodies, he still holds that they are muscle in the course of development.

In 1862, Beale (2), in a paper on the distribution of nerves to the elementary fibres of striped muscle, gives a detailed description of these bodies, and regards them as muscle and nerve in the course of development, and it is from observation of these that this author comes to the conclusion that the muscle fibre is supplied with nerves in its entire length.

In his Croonian Lectures, 1865 (2), he further considers these bodies, holding the same view and differing only from Kölliker in that he believes the development of the muscle-fibres takes place from the nuclei and not by division of the parent fibre.

Kühne (19), in 1863, working with the muscle of white rats, described a nerve dividing into two branches and entering a spindle-shaped muscle, where it ended in clear, transparent bodies, the nerve actually ending in a distinct hillock. In a more detailed paper on "Die Muskelspindeln" (19), he says that the above-described endings correspond with the broad nerve fibres. The proportion of spindles to muscle fibres is about 1-100 (in house mouse), and he further remarks that the abdominal and thoracic muscles seem to have more than the extremities. The nerve fibres passing to the spindles are three or four times as broad as those to motor nerve-endings. Each spindle is, as a rule, supplied by two nerves which come from a common trunk, the sheath of the nerve fibre passing into the sheath of the spindle. The muscle fibres of the spindle are smaller than the neighbouring fibres, and have well-marked striations at the ends, but lose these towards the centre; the striation of the muscle is broader than in the larger fibres, but this is usually the case with the smaller fibres. In conclusion he says: "It would seem from the above that a knowledge of the muscle-spindle is of great importance in

understanding the development, regeneration, and the growth of muscles and their nerves.

In a further paper (19), published in 1864, he says: "Are they an apparatus with a peculiar, still unknown physiological function for the *Zuckungsvorgang des Gesammtmuskels*, or are they only a stage in the not yet completely developed muscle fibre?"

Peremeschko (23), in 1863, in studying the development of striated muscle, discusses the spindle described by Kühne, denies that a nerve enters them, and agrees with Kölliker in calling them *Muskelknöspen*.

Sachs (28), in 1874, in a series of papers on the sensory nerves of muscles, shows that in frogs after section of the anterior roots for a period of six to eight weeks the motor fibres were completely degenerated, on the other hand the nerve in the muscle contained two well-formed, broad, medullated fibres, which after dividing gave off coarse nerve twigs; the further distribution of these differed entirely from the condition of a motor nerve.

He then discusses the result of section of the posterior roots peripherally to the ganglion. The degeneration of the sensory muscle nerves takes places very slowly, and after six weeks the appearance is by no means striking, and on this point he comes to no definite conclusion. He describes the nerve entering a spindle-shaped body and twisting round the muscle fibre in a spiral form.

Eisenlohr (9), in 1876, described, in a case of infantile paralysis, a small number of well-preserved fibres arranged within a sheath which they incompletely fill. He does not seem to have recognised them as spindles in cross section and would seem to regard them as pathological.

Fraenkel, in 1878 (13), in an exhaustive paper on the changes in the muscle of phthisical patients, describes "umschnürte Bündel" situated generally in the neighbourhood of vessels and nerves. He considers them pathological. He gives measurements of these bundles and there is no doubt that he is describing the muscle-spindle in transverse section. He states that he has never found them in the muscles of the eyes, never in the diaphragm or masseter, but they are

abundant in the muscles of the thumb. He found these "umschnürte Bündel" in one-third of all his preparations.

Ranvier (25), in 1878, discussing the spindles described by Kühne, says that they are cylindrical in shape. He notes that the muscle fibre loses its striation and becomes granular, shiny, and contains several nuclei at one part. He calls attention to the resemblance of the sheath to that of a Pacinian body. He says the nerve, after penetrating the sheath, divides into two; some fibres pass between the layers of the sheath, others either pass along a muscle fibre or curl spirally round it. He does not know whether they should be considered muscle fibres in the course of development or fibres undergoing atrophy.

Millbacher (22), in 1882, examined muscles in thirty cases of acute and chronic disease; he, like Fraenkel, found the "umschnürte Bündel" in about one-third of all his preparations; he, too, never found them in the eye muscles, diaphragm or masseter, being abundant, however, in the thumb. He describes three forms of "umschnürte Bündel":

(i.) Unvollständig umschnürte Bündel;
(ii.) Vollständig umschnürte Bündel, containing muscle fibres;
(iii.) Vollständig umschnürte Bündel, containing atrophied fibres;

and in these three forms he sees the process of degeneration. He found them also in four cases of apparently healthy individuals, and in these cases he is doubtful of their significance.

Golgi (15), in 1882, describes a spindle and gives drawings in cross and longitudinal sections. Why these fibres lie imprisoned and preserve, even in adult life, the characters of imperfectly developed fibres is not clear. He suggests that they have some special connection with the lymphatic system.

Bremer (4), in 1883, working with the muscles of frogs, mice and lizards, believes that muscle-spindles are a stage in the development of muscle, and he traces the various steps in development, not only of the muscle, but also of the end

plate. He notes the large size of the nerve fibre entering the spindle and its thick sheath ; he notes that the muscle fibre loses its striation near the centre of the spindle. He gives a detailed description of the spindle, and associates the variation in the spindle with various stages of development.

Babinski (1), in 1884, described, in the muscles from a case of chronic myelitis, circular rings having in their centre several atrophied fibres. He considered them as pathological. In a further paper, published in 1886, he gives a description of the spindle in transverse section, having a diameter of 100 to 200 μ; he says the spindle generally contains three to seven fibres ; he notes the sub-division of the intrafusal space, one part conveying the vessels, another the nerve, and the third the muscle fibres. He no longer considers them as pathological, but is doubtful of their real meaning. In a third paper, published in 1889, he criticises Eichhorst's " Neuritis fascians."

Mays, in 1884, studying the distribution of nerves in the muscles of frogs by means of the gold chloride method, describes certain appearances which he compares to the muscle-spindles of Kühne ; he believes they are in connection with the sensory nerve fibres.

In 1892, in a paper on the development of the motor nerve endings, he expresses the opinion that muscle-spindles take no part in the development of muscle.

Roth (26), in 1887, published, under the heading "Neuro-musculaire stämmchen," a description of a muscle-spindle. He had found them first in atrophied muscle, and soon after in healthy muscle. He considers them as physiological organs.

Westphal (39), in 1887, in two cases of pseudo-hypertrophy in sisters, describes small groups of muscles surrounded by connective tissue occurring in muscle which was completely atrophied ; he does not seem to recognise them as muscle-spindles.

Eichhorst (8), in 1888, describes, under the heading " Neuritis fascians," certain structures he has noted in the muscles from a case of alcoholic neuritis. He describes the onion-like sheath of the spindle, and believes that it comes from the thickened sheath of the nerve.

Felix (11), in 1888, working with the muscle of fœtuses from the fourth month to the thirty-sixth week, describes "primitive muscle bundles"; these are characterised by (1) greater breadth, (2) more intense staining, (3) wider striation, (4) marked Henle sheath. Nuclear increase is marked at one part of the muscle fibre, and tracing a primitive bundle one sees (1) an increase of irregularly-arranged nuclei, (2) nuclei arranged in rows; usually three rows of nuclei are found, the primitive bundle here beconing widened to nearly three times its former diameter. Only once did he find a nerve near, and he doubts whether they had any connection; he compares them to the muscle-spindles of the frog. In a further paper (11), in 1889, studying the growth of striated muscle in man, he points out that in the adult muscle-spindle there are usually three to six muscle fibres, whereas in the fœtal spindle there may be as many as twenty; this he attributes to the division of the daughter fibres. The sheath of the spindle is characteristic, and passes gradually into the perimysium of the muscle, so that on section towards the end of a spindle there is nothing to call attention to the presence of a spindle. He believes that spindles are most frequently found near the tendon, and, as may be inferred from the above, he considers them as a stage in the development of muscle.

Cattaneo (5), in 1888, studying the musculo-tendon nerve termination described by Golgi, both under normal conditions and after experimental lesions in guinea-pigs, rabbits, cats, and dogs, describes a body having a length of 80 to 800μ. situated generally where the muscle passes into the tendon, one extremity of the organ passes into the tendon the other into the muscle. The sheath enclosing these resembles the Henle sheath of a nerve. A nerve enters this body, and after passing through the sheath divides into two; the mode of termination is described. Blood vessels can also be seen entering this body. Among these musculo-tendon organs one can sometimes see a muscle-spindle, such as has been described by Kölliker, and after discussing various views, he is inclined to agree with him (Kölliker) rather than with Millbacher and Kraske, who regard them as pathological.

He has seen the muscle-spindle lie between two of the

musculo-tendon organs, but the nerve supplying the spindle arrives from a different direction. There is, however, a certain analogy between the two. With regard to the function of these musculo-tendon organs, after discussing the point at some length, he says that if they are the organs of muscular sense they ought to be independent of motor nerve fibres, and to be intimately connected to sensory fibres. To answer this question two paths are open: (1) the pathological, (2) the experimental. Not having the opportunity for the first, he turns to the second.

In dogs, after section of the posterior roots (fifteen days to four months), ataxia was found to be present, but he could find no alteration in the musculo-tendon organs nor in the muscle fibre.

In dogs in whom the anterior roots were cut the musculo-tendon organs remained normal; the muscle itself, however, showed advanced atrophy.

After complete section of the nerve, changes were found in the terminal organs 36 to 38 hours after section.

In conclusion, he says that the musculo-tendon organs are found sometimes in contact with muscle-spindles, sometimes with Pacinian bodies, but the position of the nerve fibre proves them to be independent of these bodies.

He considers the musculo-tendon organs above described as the true organs of muscular sense.

He further adds that, after section of the nerve, degeneration of the medullated portion of the nerve is slow, whereas in the non-medullated part it occurs in 20 hours, and disappears in three to four days.

Siemerling (32), in 1888, in a paper on a case of gumma of the base of the brain, describes what he considers to be a spindle in the inferior rectus of the eye, which muscle had undergone considerable atrophy.

In a second paper he criticises Eichhorst's "Neuritis fascians," and in a third (32) he reports a case of alcoholic neuritis, and says that the spindle seems to enjoy a certain immunity against pathological changes. This is not always so, however, for in two cases of phosphorus poisoning he found the muscle fibres of the spindle filled with fat globules.

Kerschner (17), in 1888, after describing the muscle-spindle, says that the spindle frequently ends in the connective tissue, and suggests that the function of the spindle might be to protect the nerve-ending; he comes, however, to the conclusion that they are a complicated sensory organ which may serve muscular sense. In a further paper (17) he compares muscle-spindles with other sensory end organs found in joints, tendons, conjunctiva, tongue, &c. In 1892, von Ebner (17) showed for Kerschner certain specimens to demonstrate the nerve-ending within the spindle stained with gold chloride; two nerve-endings are described, one a motor on the muscle fibre, another sensory, twined round the muscle fibre. In support of the sensory nature of these organs he puts forward the following points:—

(i.) The abundant nerve supply, far above the number of muscle fibres.

(ii.) The division of the nerve fibre after a type which reminds one more of a sensory than of a motor ending.

(iii.) The presence of nuclear groups like those of end-bulbs.

(iv.) The frequent ending of part of the same nerve in a tendon organ.

(v.) The analogy with a Golgi organ: (α) supply from the same nerve; (β) similar sheath.

(vi.) Their constant presence; their number and appearance in many muscles in which fine muscular sense is needed.

(vii.) The apparent vicariousness of the Golgi organ in similar muscles, as in the muscles of the eye.[1]

Kerschner then enters into the various points that have been advanced in proof of the embryological characters of the spindle, viz., the darker staining, the abundant nuclei, the variations in the sheath, and the presence of double and quadruple spindles. He says the darker staining frequently occurs with the smaller muscular fibres, the abundant nuclei occur at the nerve entrance, and the variations of the sheath are dependent on the point at which it is cut across. He points out that mitosis is absent. The one point which he

[1] Comp. Marchi, *Archiv für Ophthalmologie*, xxviii., 1882, and *Journal of Physiology*, vol. xvii., p. 248.

agrees looks like a developmental process is the division of the spindle into two and sometimes into four. In a further paper (17), 1893, he sums up the views held at that date by various authors.

Pilliet (24), in 1890, described the presence of muscle-spindles in the muscles from cases of alcoholic paraplegia, chronic rheumatism, and amyotrophic lateral sclerosis. He says it would seem that atrophy of the muscle makes them apparent.

Blocq and Marinesco (3), in 1890, showed that in a case of poliomyelitis and another of polyneuritis that the muscle spindles existed when there was total loss of all other muscle fibres.

Von Franque (14), in 1890, maintains the view that the muscle-spindle is a stage in the development of muscle.

Santesson (29), in 1890, describes the muscle-spindle in a case of myopathy (Leyden's form), and regards them as pathological appearances.

Dogiel (7), in 1890, studying the motor nerve-endings in the muscles of frogs, &c., by staining them with methylene blue, describes the entrance of the nerve into a muscle-spindle. The chief fibre to the spindle he describes as dividing into two—(i.) winds in the spindle in a spiral form and (ii.) distributes itself to the spindle sheath.

Christomanos and Strössner (6), in 1891, studying the spindle under normal circumstances in a fœtus, a new-born child, a child nine years old, and in an adult, found them in all of them, and they give a table showing the comparative size of the intra- and extrafusal muscle fibres. They describe an inner and outer sheath, and note that the spindle is larger in adults than in children, the increase being due chiefly to the thickness of the wall. They consider the spindle to be a sensory organ.

Trinchese (35), in 1891, sums up what is known of the muscle-spindle; he refers to his own work in 1888, when he demonstrated that the spindles had a different form and structure at the various periods of their development. He describes the nerve entering the nucleated part of the muscle fibre, and finishes by stating that the assertion of Bremer

that these fibres are muscle fibres in the course of development has not yet been demonstrated as being true. It would, indeed, seem probable, but until the phase of transition between the adult muscle fibre and the spindle has been demonstrated it cannot be asserted positively what the nature of these organs is.

Erb (10), in 1891, describes a bundle of small fibres surrounded by a nucleated, definitely striated band, which does not completely surround these fibres (p. 94), and in relation to the regeneration taking place in a degenerated muscle, he says that the foregoing curious condition stands in close relation to the *Muskelknöspen* (p. 147).

V. Thanhoffer (34), in 1892, asserts that the so-called muscle-spindles are nothing more than "building material" for the regeneration of muscle and nerve fibres.

Volkmann (36), in 1893, studying the regeneration of striated muscle, says he has found the muscle-spindle in four cases—(i.) the thumb muscle after injury to the arm, (ii.) diphtheritic paralysis, (iii.) in rectus abdominis of man aged 40, and (iv.) in the hand of a man who died of spinal disease. He believes that these bodies have to do with the growth of muscle, and not with regeneration of muscle.

Ruffini (27), in 1893, compares the muscle-spindles as found in cats with those found in man. He gives a description of the various terminations, and states that the annulospiral termination present in the cat is not found in man. The termination *à fleurs* is present in both, but is more numerous in man. In conclusion, he says that one may regard these *fuseaux musculaires* as special nerve organs, having a function unknown.

Forster (12), in 1894, working with the muscle from cases of muscular atrophy, transverse myelitis, &c., found that in muscles which were completely degenerated the muscle fibre inside the spindle remained well preserved—the nerve fibre passing to the spindle being also well preserved, as shown by Weigert's staining. It would seem that the trophic centre for the spindle does not lie in the spinal cord, and it is suggested that it lies in the spinal ganglion. Muscle-spindles are considered as a physiological apparatus,

and the suggestion of Kerschner is followed, viz., that they serve muscular sense.

Sherrington (30), in 1895, examined muscle in which all the motor fibres had been divided and the muscle fully degenerated; he found that the spindle remained intact, the nerve fibre passing to it was well-preserved and could be traced to the sensory roots. The intrafusal fibres were well preserved, the striation well marked.

Further, in a cat in whom the sciatic had been divided for 150 days, the muscle was completely degenerated; with regard to the spindle, the nerve fibres passing to it were degenerated, and the spindle itself contained globules of fat, but the intrafusal muscles were well preserved and their striation well marked. It would seem, therefore, that the intra-fusal muscle fibres are independent, in regard to their nutrition, both of afferent and efferent fibres.

Sihler (33), in 1895, published a method of staining spindles with hæmatoxylin. In the same year he published the results of his investigations in snakes and frogs. He gives a detailed account of the spindles, and considers them to be sensory organs.

Weiss and Dutil (38), in 1896, working with the muscle of rabbits, cats, &c., and using by preference the gold chloride method described by Ranvier, describe three modes of nerve termination within the spindle :

(i.) Two fine branches, one passing above, and the other below, a nucleus.

(ii.) A small eminence on the sarcolemma, where there is a group of two to three nuclei. This ending is generally found toward the pole of the spindle.

(iii.) Fibres spread themselves on the surface of the muscular bundles in tree-like form, deprived of all nuclear elements.

They arrive at the conclusion that spindles are sensory in function, and probably have the same function as Golgi's tendon organs.

Gudden (16), in 1896, says that in alcoholic neuritis the muscle fibres in the *neuro-musculaere Stämmchen* are degenerate, and that the nerves within the spindle do not stain normally.

(II.) Theories with regard to Origin and Function of the Muscle-spindle.

From the above abstract it will be seen that various views have been held as to the origin and function of the muscle-spindle, and I would here simply enumerate them.

(i.) That the muscle-spindle is muscle and nerve in a stage of development.

(ii.) That the muscle-spindle is muscle in the state of degeneration; (*a*) physiological; (*β*) pathological.

(iii.) That the muscle-spindle is a sensory nerve-ending in muscle.

(iv.) That the muscle-spindle is a protection to the nerve-ending during contraction of the muscle.

(v.) That the muscle-spindle has special connection with the lymphatic system.

I do not propose in this paper to enter into a discussion of these theories, all of which are dealt with at some length in the papers above quoted.

I believe, however, that all the evidence at present points to the fact that the muscle-spindle is the organ from which muscular afferent impulses are derived. Closely allied to the muscle-spindle are the musculo-tendon organs, and the tendon organs situated respectively between the muscle and the tendon and in the tendon.

What the exact nature of the impulses derived from these organs may be seems to me at present uncertain, but I think that the suggestion of Kerschner with regard to the muscle-spindle, by Cattaneo with regard to the musculo-tendon organ, that they are connected with the sense of position, may well receive consideration.

(III.) Methods.

I have used the following methods and stains in my work:

As often as possible portions of muscles have been hardened in Müller's fluid (some specimens which had been in spirit for some time were not put into Müller's fluid).

Small pieces were then placed in Marchi's solution for five to six days, washed in water, placed in alcohol and embedded in celloidin—some were, however, embedded in wax, cut and mounted in series.

Staining.

(i.) Specimens were mounted without further staining.
(ii.) Others were stained with hæmatoxylin and eosin.
(iii.) Others were stained by Pal's method.

The method, however, which I have found of most service is that described by Sihler (33).

A portion of fresh muscle is taken, and I find it best to select a part near the entrance, or slightly below the entrance, of a nerve. Divide this longitudinally into pieces the size of a small pencil, or, as I have found very convenient, freeze the tissue and cut *thick* sections in the direction of the muscle-fibre—put these into the following solution :

Acetic acid	1 part
Glycerine	1 part
Chloral hydrate solution, 1 per cent. in *distilled water*	6 parts

Let the tissue remain in this solution for twenty-four hours, then saturate with glycerine for two to three hours. The tissue is then swollen up, and can easily be pulled apart into fine strands about the size of a pin (if the tissue has been cut on a freezing microtome this is not necessary), which are placed in the following solution for three to ten days :

Ehrlich's hæmatoxylin	1 part
Glycerine	1 part
Chloral hydrate sol. 1 per cent. in *distilled water*	6 parts

Pieces of muscle are now removed, placed between two slides and squeezed ; on holding the specimen up to the light, or on looking at it under a low power of the microscope, a spindle-shaped body can often be seen stained darker than the surrounding muscle fibre. It will be noticed, too, that

the nerves, vessels, and lymphatics are also stained darker than muscle tissue. The spindle can now be teased out under the low power of the microscope, or even without the aid of any magnifying power.

In searching for the spindle it is always a good plan to follow a nerve fibre, as it frequently happens that such a fibre guides one to a spindle.

Having separated out a spindle, it can now either be mounted in the usual way or, if overstained, treated with dilute acetic acid—or after washing in water can be put to harden in Müller's fluid.

After hardening in Müller's fluid till all colour has disappeared from them, they can be placed in Marchi solution, treated in the usual way, and embedded in celloidin, and cut either longitudinally or transversely *in series*. The sections can then be stained by Pal's method, and a counter stain, dehydrated, and mounted in series. Specimens hardened in the above method, and mounted without further staining, show any recent degeneration.

Many of the series have been cut in wax, but I have experienced great difficulty in staining a spindle by Pal's method as a whole, and by far the most satisfactory results are obtained by cutting in celloidin and staining each section separately. The embedding in wax is, however, very useful for pieces of muscle stained with carmine or with Marchi solution, which do not require further staining.

(IV.) The Normal Muscle-spindle as Found in Man.

Distribution.—Muscle-spindles have been found in nearly all muscles of the body, but they are by no means evenly distributed. They are certainly numerous in the small muscles of the hand, and in the biceps of the arm (the latter muscle I have used more often than any other one muscle). Forster (12) states that they are frequent in the extrinsic muscle of the larynx. I have been unable to find them in the muscles of the eye, the intrinsic muscles of the tongue, or in the diaphragm, but my examination of these muscles

has been by no means exhaustive. The above statement agrees with that of other authors, Fraenkel (13), Millbacher (22), Kerschner (17), and Sherrington (30); but, with regard to the eye, is at variance with the statement of Siemerling (32), who has described a spindle in an atrophied inferior rectus muscle of the eye; and in regard to the tongue is at variance with a statement of Forster's (12), that they are to be found in the hinder part of the tongue.

Further, the muscle-spindles are more frequent in the belly of the muscle than near the tendon, but in relation to this statement it would seem to me that we should recognise in the muscle-spindle, in the musculo-tendon organ, and in the tendon organ the gradual transition from the one organ into the other, and I would regard them, not as distinct organs, but as variations of the same organs adapting themselves to the tissue in which they are situated, and in all probability serving the same function.

Age.—Muscle-spindles occur at all ages, from the fourth month of fœtal life onwards; this has been shown by Felix (11), Siemerling (32), and Christomanos and Strössner (6).

Size.—The size of a muscle-spindle varies considerably. One of the longest simple spindles I have found measured 11·7 mm., and the maximum breadth ·5 mm., but an average size is from 2 to 4 mm. in length, and ·15 to ·4 mm. in breadth. It would seem to me that the size varies both with regard to age and to the length of the muscle, being longer in the adult than in the child, and in the longer muscles.

Frequency.—I have made no attempt to estimate the number of spindles in any given muscle, but an estimate has been made by Felix (11), who found seventy-nine in one biceps. They are, however, much more easily found in wasted muscle than in well-developed muscle, and more easily in the muscle of a child than in that of an adult, hence the muscle of a child dying of a wasting disease is a muscle in which it is easy to demonstrate the muscle-spindle.

Description.—The muscle-spindle is, as its name implies, spindle in shape (fig. 1), varying considerably in size.

PLATE I.

Fig. 1.

Normal Spindle from Abductor Pollicis of Foot (Man).

Length	6·65 mm.
Breadth	·38 mm.

A nerve (N) can be seen entering the equatorial region and this nerve contains a fibre whose breadth is ·01 mm. A branch containing finer nerve fibres, ·004 mm., leaves the nerve and enters the spindle further to the right. Another nerve (N) enters the spindle at the opposite pole, this also is composed of fine fibres—one measuring ·006 mm.

An artery (A) and a vein (V) can also be seen giving branches to the spindle.

Teased Specimen. Sihler's Method.

Magnified 8 diameters.

Fig. 2.

Compound Spindle.

Length of complete spindle	1·5 mm.
Length between entrance of nerves	1·275 mm.
Breadth	·12 mm.

The pole of the second spindle enters the other spindle a little to the polar side of the equatorial region.

Compare figure 15, compound spindle in cross section.

Teased Specimens. Sihler's Method.

Magnified 24 diameters.

Fig. 3.

Double Spindle.

Length	5·5 mm.

The pole of one spindle passes into the pole of the next, thus forming one continuous spindle—the nerve, however, enters the equatorial region of each spindle.

Teased Specimen. Sihler's Method.

Magnified 7 diameters.

PLATE I.

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FIG. 1.

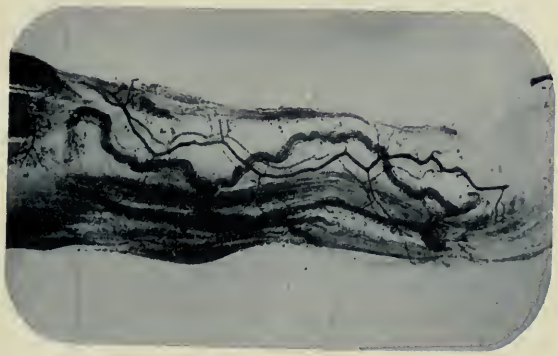

FIG. 2.

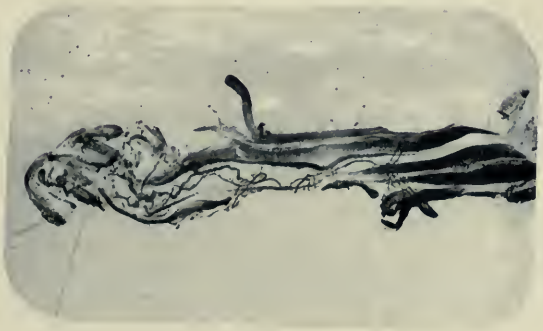

FIG. 3.

It is common to find several spindles in one plane of the muscles, and they are often found along the course of a nerve from which they receive their supply, and lie parallel to it (fig. 2). Spindles are sometimes found compounded of two simple spindles (fig. 2), but I have no teased specimen of more than two so compounded. Sherrington, however, describes a triple spindle, and Kerschner a quadruple (fig. 15). On the other hand, it is not uncommon to find one spindle joined at its end with another spindle (fig. 3), and that again may be joined to another; three in a row is the maximum number that I have found. In such a case each spindle receives its own nerve supply.

The relation which the muscle-spindle bears in position to the muscle fibre also varies. Firstly, the muscle-spindle may lie wholly in muscular tissue, the muscle fibre passing in at one end, and, after passing through the spindle, passing out at the other. Secondly, the spindle may lie partly in muscular tissue and partly in connective tissue. Thirdly, the muscle-spindle may lie wholly in the connective tissue, the contained muscle fibre being independent of external muscle fibres.

The essential portions of a muscle-spindle are—(i.) the muscle fibres, (ii.) the nerves and nerve-endings, (iii.) the sheath, (iv.) the blood vessels, (v.) the lymphatics and lymph spaces, (vi.) septa dividing the spindles into compartments.

The various parts will now be described in the above order.

The Muscle Fibre.—One or more muscle fibres enter one pole of a spindle; these fibres are smaller than the ordinary muscle fibres, being about ·02 mm. in diameter (the average extrafusal fibre measuring about ·06 mm.) (fig. 4). The muscle fibres are well striated, and their striation is generally slightly coarser than the extrafusal fibres, ten striations occupying the same space as thirteen striations of the extrafusal fibres. As the muscle fibre or fibres pass towards the equatorial region of the spindle they become divided into many smaller fibres, some of these measuring only ·008 mm. (fig. 4). At a certain point the muscle fibre appears to lose its striation, and nuclei appear in the

PLATE II.

Fig. 4.

Normal Spindle from Biceps of Man.

	Diameter.
Transverse section	·24 mm.
Extrafusal-muscle fibres	·04—·06 mm.
Intrafusal-muscle fibres	·008—·024 mm.

Magnified 160 diameters.

Fig. 5.

Normal Spindle in Longitudinal Section.

To show intramuscular nuclei, the nerve passing to this portion of the spindle is seen at N.

The sheath is well seen, and the comparative size of the intrafusal (Mi) and the extrafusal (Ne) muscle fibres is readily observable.

Intramuscular nuclei	·006 mm.
Intrafusal muscle fibres	·02 mm.
Extrafusal muscle fibres	·04 mm.
Nerve fibre	·004 mm.

Magnified 110 diameters.

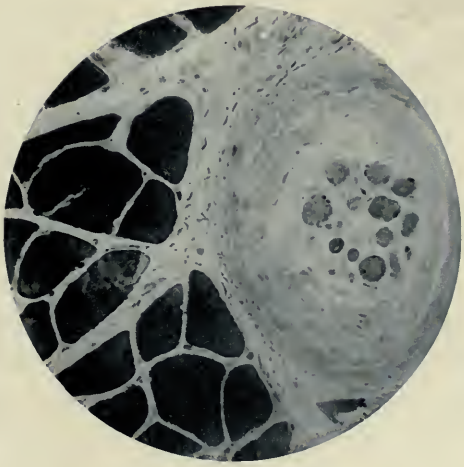

FIG. 4.

FIG. 5.

substance of the muscle fibres (fig. 5); these nuclei gradually increase in number till they completely fill the muscle fibre; then, after a short distance, they again become less numerous, and the muscle fibre again resumes its striation. The nuclei are rounded, have a diameter of about ·006 mm. (fig. 5), they stain well with hæmatoxylin, and generally occur about the equatorial region of the spindle.

In the equatorial region of the spindle the muscle fibres usually lie to one side, as they do not completely fill the space, whereas at the proximal and distal ends the sheath closely envelopes the muscle fibres. The muscle fibres, after passing through these changes in the equatorial region of the spindle, pass to the distal end, where they become joined again and pass out of the spindle. I do not believe that all fibres become so nucleated, for I have seen muscle fibres pass through a spindle without losing their striation at any point. The number of muscle fibres in a spindle varies according to the different regions seen in section.

Nerves and Nerve-endings.—The nerve supply is always abundant; as a rule, at least two nerves pass to a muscle-spindle, the one at the equatorial region, the other at the proximal or distal end. It is not uncommon, however, to find four nerves entering a spindle at various points, two or more arising from a common nerve trunk (figs. 1 and 10).

The largest nerve bundle usually enters the spindle at the equatorial region, and contains two or three fibres, the largest measuring about ·008 mm. The nerve bundles which enter toward the distal and proximal ends are generally composed of finer fibres, and sometimes of only a single fibre having a diameter of ·004 mm. or less.

These nerve fibres seem to terminate in various ways; as a rule, the large fibre which enters the equatorial region passes directly to the muscle fibre, and seems to lose itself in the nuclei of the muscle fibre above described (fig. 5); some fine fibres pass between the muscular fibres and terminate in such an organ as is figured in (fig. 7); others seem to have a spiral form. Others, again, form a fine plexus beneath or in the sheath of the spindle (fig. 6). So far

PLATE III.

Fig. 6.

Normal Spindle in Longitudinal Section.

Stained by modified Pal's Method to show nerve plexus toward the polar region of the spindle.

Letter N points to position of nerve termination, which is seen magnified in fig. 7.

Magnified 28 diameters.

Fig. 7.

Same as fig. 6.

Under higher magnification to show nature of nerve ending within the spindle.

Diameter of terminal end bulb (N)=·008 mm.

Magnified 375 diameters.

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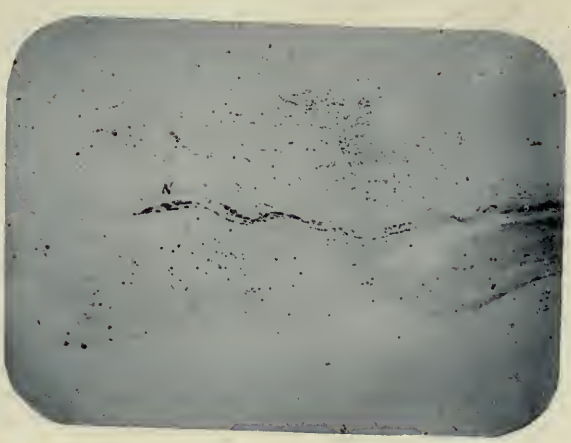

FIG. 6.

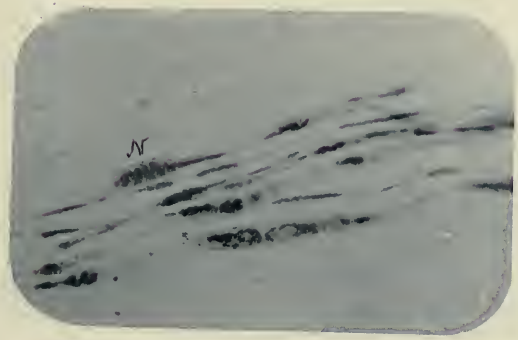

FIG. 7.

as my observations go, I have never seen a true motor end plate within a spindle. With regard to the number of nerve fibres within a spindle, one may see at times eight to ten in section. As a rule they are more numerous in the equatorial region than at the distal or proximal ends, but they are abundant in both situations.

Nerve Sheath.—Each nerve bundle passing to a spindle has a definite nerve sheath, composed of two or more layers. The nerve bundle passing to the equatorial region of the spindle has usually a sheath composed of eight or more laminæ, whilst the nerves entering at the distal or proximal ends have a sheath containing two to three layers. The sheath of the nerve passes directly into the sheath of the spindle as the nerve enters.

Spindle Sheath.—The sheath of the spindle resembles that of the nerve, and, as has been pointed out by certain authors, has the appearance of an onion in cross section. The laminæ are extremely fine, and possess elongated nuclei at intervals (fig. 14). At the equatorial region these laminæ are numerous, an average number being eight to ten; at the distal and proximal ends of the spindle the number of laminæ become less, and eventually pass into the muscle sheath. The characters of the sheath are best studied in cross section.

Blood Vessels.—The muscle-spindle is supplied with arteries and veins, which most frequently enter the spindle near the entrance of the central nerve, although vessels may be seen entering the spindle at various points (fig. 1).

Lymphatic and Lymphatic Spaces.—A lymph space is situated about the equatorial region of the spindle, and occupies about the middle one-third of the spindle. In some of my specimens the space exists as such, whilst in others it is filled up by a granular material, which does not stain readily. Sherrington has been able to inject this space by injecting the lymphatics of the leg.

Lymphatic vessels can also be seen in the teased specimens running parallel to the course of muscle fibres, and forming junctions with other lymphatics.

Septa.—Between the groups of muscle fibres and nerves within the spindle, septa are often seen dividing the spindle

PLATE IV.

Fig. 8.

Transverse Section from Tricep Muscle of Infantile Paralysis.

Two muscle spindles (S) are seen lying in completely atrophied areas.

Diameter of spindle { (i) = ·25 / (ii) = ·17

Magnified 24 diameters.

Fig. 9.

One of the above spindles under higher magnification, eight muscle fibres of varying sizes can be seen within the spindle, and two nerve fibres (N) which are well stained by Pal's method.

Size of nerve fibres	·01 and ·003
Size of muscle fibre	·024
Diameter of spindle	·25

Magnified 160 diameters.

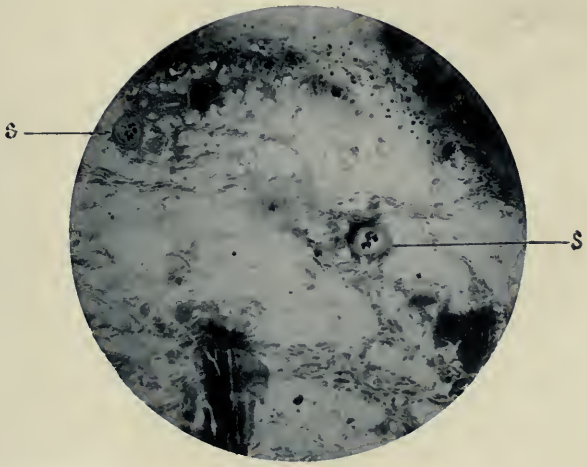

FIG. 8.

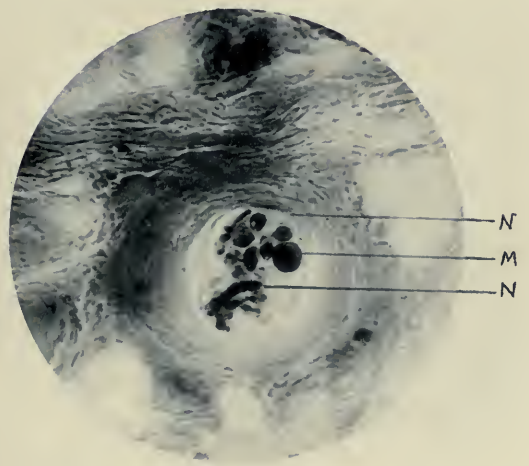

FIG. 9.

into two or more parts; these septa are extremely fine, and pass into the sheath of the spindle.

TABLE I.

Normal Muscle.

	Muscle.	Size of Extra-fusal Muscle Fibres.	Size of Spindle (outside).	Size of Intra-fusal Muscle Fibres.
Man.	Biceps.	·06 mm.	·16 mm.	·016.
Child.	Biceps.	·02 mm.	·16 mm.	·016.

Measurements in Millimeters.

(V.) THE CONDITION OF THE MUSCLE-SPINDLE UNDER THE FOLLOWING PATHOLOGICAL CONDITIONS.

(i.) Infantile paralysis; (ii.) tabes; (iii.) myopathy (Leyden's form); (iv.) progressive muscular atrophy; (v.) peripheral neuritis; (vi.) injury to brachial plexus; (vii.) after section of sciatic in cats.

I do not propose to enter into the clinical history of these various cases, or to describe the pathological changes in the various muscles under examination, except in so far as they concern the muscle-spindle.

(i.) *Infantile Paralysis.*—I have been able to examine the muscle from six cases of infantile paralysis; five of these were from museum specimens, and, having been preserved in spirit, could not be stained by Pal's method. In the sixth case I was able to obtain the arm fresh, and have been able to treat it by the methods above described. In all the cases the paralysis was of long standing. In four of the cases, thirteen muscles were examined; all these muscles were considerably atrophied; some had apparently no normal muscle fibres left, yet in all of these the muscle-spindles could be demonstrated, and the intrafusal fibres were about the usual size (fig. 8). In the other two cases

the whole substance of the muscle was replaced by fat, and in these two specimens I was unable to find any spindles (only small pieces of these muscles were obtainable). What is the condition of the nerves entering the muscle-spindle? In answer to this question I was able to examine the muscles of case No. 6 by Pal's method, and it is found that not only the nerve entering the equatorial region (fig. 9) of the spindle, but also the plexus of nerves towards the polar ends, remain intact. The teased specimens give the same result, and also show that the striation of the intrafusal fibres is well preserved. In regard to infantile paralysis, it would seem probable that the muscle-spindle remains absolutely intact, both in regard to the intrafusal muscle fibres and in regard to the contained nerves. Table II. gives details of measurements and muscle examined.

(ii.) *Tabes.*—I have examined the muscles from three cases of tabes. In two of these I had only portions of the muscle, which were given to me after being in Müller's fluid for some time. These I examined by the usual methods, staining them with hæmatoxylin and by Pal's method, and, so far as the examination goes, it shows that the spindles are normal, both in regard to the intrafusal muscle fibres and the nerve supply. With regard to the third case, I have been able to examine it with greater care. Spindles have been teased out in the usual manner from the rectus femoris, vastus internus, and biceps of the arm. So far as the shape of the spindle and the nerves entering it is concerned, no change can be seen (fig. 10). The spindle does not, however, stain so readily with hæmatoxylin as does the normal spindle, and especially the equatorial region, which, as a rule, stains very deeply. The intrafusal nerve fibres, however, stain well. The striation of the intrafusal muscle fibre is well preserved. Examining the specimens of a spindle cut in longitudinal section, stained by Marchi solution, one finds, situated in the muscular fibre in the same position as the round cells which have been above described, evidence of degeneration (fig. 11). If now one examines the section stained by Pal's method, in order to find out if any degeneration has occurred in the nerve fibre passing to this

TABLE II.

Infantile Paralysis.

Case.	Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibres.	Size of Spindle outside.	Size of Intrafusal Muscle Fibres.	Nerve Staining and Size within Spindle.
I. Man	Triceps	Extensive atrophy	·04 mm.	·12 mm.	·018	—
II. Child	Biceps femoris	Atrophied	·008	·1	·016	—
	Semi-membranous	Atrophied	·006	·12	·016	—
	Rectus femoris	Atrophied	·008	—	·015	—
III.	Anterior tibial	Some normal fibres	·07	·112	·013	—
	Gastrocnemius	Irregular atrophy	·014	·072	·011	—
	Permei	Irregular atrophy	·03	·056	·011	—
	Solens	Well developed	·057	·12	·013	—
IV.	Gastrocnemius	Fatty, with scattered fibres	·08	—	—	—
V.	Gastrocnemius	Fatty	—	—	—	—
VI. Woman	Deltoid	Partly atrophied	·04	·088	·015	Nerve well stained ·004
	Biceps	Partly atrophied	·035	·132	·013	Nerve well stained
	Supinator	Partly atrophied	·073	·14	·014	Nerve well stained
	Triceps	Completely atrophied	—	·168	·018	Nerve { ·008 mm. ·003 mm.
	Abductor pollicis	Completely atrophied	—	·18	·012	Nerve well stained

PLATE V.

Fig. 10.

Normal Spindle from Vastus Internus (Tabes).

	Teased Specimen.	Sihler's Method.
Length		11·9 mm.
Breadth		·51 mm.

Nerve bundles are seen entering at four points, 1, 2, 3, 4.

No. 1. contains the largest nerve fibre measuring ·008 mm. and terminates about the equatorial region of the spindle.

The other nerves entering towards the poles are finer, varying from ·003 to ·006 mm.

At the point marked 2 the nerve divides, one part can be seen passing to the motor nerve ending on the muscle fibres extrinsic to the spindle, the other enters the spindle.

Magnified 7 diameters.

Fig. 11.

Spindle from the Rectus Femoris (Tabes).

Cut in longitudinal sections to show degenerative change within the intrafusal muscle fibre. Stained in Marchi's solution.

The degeneration is manifest by the deposit of fat in that portion of the intrafusal muscle fibre when it has been shown that intrafusal muscle nuclei exist, the remaining portion of the muscle fibre remaining free.

Ms = Extrafusal muscle fibre.
M = Intrafusal muscle fibre.
Sh = Sheath.
D = Area of degeneration.

Breadth of spindle	·18
Breadth of muscle fibre	·024

Magnified 150 diameters.

Fig. 12.

Spindle from Rectus Femoris (Tabes).

Cut in longitudinal section stained by Marchi-Pal Method to show that the nerve passing to the degenerate area within the spindle remains normal. The degenerated area within the muscle-fibre is apparent.

Intrafusal muscle fibre	·028

Magnified 180 diameters.

FIG. 10.

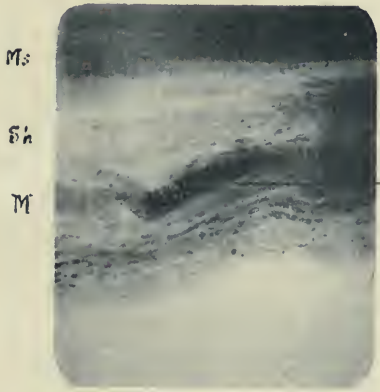

FIG. 11.

FIG. 12.

spot, one finds that the nerve fibre stains well, and shows no sign of degeneration (fig. 12). In a third specimen the same appearance is observable. It is difficult to prove that the above-described condition is truly pathological, but it is a condition which I have found in tabes, and not under any other circumstances. That changes should be found in the terminal organs, while the nerve lying between the ganglion cell and the terminal organ remains intact, seems to me not only a possible, but a probable lesion, for it has been shown by Cattaneo (5) that, after section of the whole nerve, changes take place in the nerve termination within thirty-six to thirty-eight hours, while changes in the medullated portion of the nerve are extremely slow. So that in tabes, where one has a gradual degeneration taking place in the cells of the posterior ganglion, it is not unreasonable to suppose that changes take place at the extreme end of the neuron, without any change being manifest in the nerve itself. With regard to the other nerve-endings within the spindle, I find no evidence of degeneration (see Tables III. and IV.).

(iii.) *Myopathy* (Leyden form).—I have had the opportunity of examining the muscles from one case of myopathy, and in this case the following muscles were examined:—Biceps (arm), pectoral major, pectoral minor, seratus, quadriceps, gluteus, and rectus abdominis. The sections have been prepared in the usual manner, except that there are no teased specimens. With regard to the condition of the spindle, it is remarkable, on examining the biceps, how numerous the spindles seem to be, as many as seventeen being found cut in one section. I have never found so many in any normal biceps, not even in the biceps from a boy of the same age, who died of phthisis. It was such an appearance which gave rise to the idea that these spindles were pathological bodies occurring in wasting muscles. This apparent increase is not so noticeable in the other muscles examined. With regard to the nerve supply, it is found that the nerves to the spindle all stain well by Pal's method, and the arrangement of the nerve fibres can be well studied. The striation of the muscle fibre is well marked within the spindle. In many of the spindles the intrafusal

TABLE III.

Tabes.

Case.	Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibre.	Size of Spindle outside.	Size of Intrafusal Muscle Fibre.	Nerve Staining within Spindle.
I. Man	Vastus Internus (left)	Normal	·048	·072	·015	Well stained
	Vastus Internus (right)	Normal	·048	—	—	—
II. Man	Vastus Internus	Normal	·06	—	—	—
III. Woman	Rectus femoris	Normal	·04	.1	·012	Well stained
	Vastus Internus	Normal	·04	·24	·018	Well stained
	Biceps	Normal	·036	·15	·02	Well stained

TABLE IV.

Tabes (Teased Specimens).

Case.	Muscle.	Length of Spindle.	Breadth.	Number of Nerves entering Spindle.	Size of Intrafusal Muscle Fibre.	Nerves.
III. Woman	Rectus femoris	—	—	3	·04	Well shown
	Vastus internus	11·9 mm.	·51 mm.*	3	·02	Well shown
	Biceps	8·5	·5 mm.	4	·028	Well shown

* Probably increased by pressure of cover glass.

fibre is as large, and often larger, than the extrafusal fibre, the same condition is met with in the muscles of young children. In myopathy, then, the muscle-spindle remains without alteration (see Table V.), and (fig. 13 and 14).

(iv.) *Progressive Muscular Atrophy.*—The condition of the muscle-spindle in progressive muscular atrophy has been worked at by more than one author, viz., Pilliet, Blocq and Marinesco and Forster, and they agree that the spindle does not undergo alteration. Forster has further shown that the nerve to the spindle remains intact. I have had the opportunity of examining muscles from three cases of progressive muscular atrophy. In the teased specimens from these cases it is noticeable how the large nerve fibre passing to the spindle stands out against the surrounding atrophied fibres in the nerve. The spindle itself remains unaltered, the nerve fibres within the spindle are natural, and the intrafusal fibres preserve their striation. My observations, then, would agree with the above authors, viz., that the muscle-spindle remains unaltered in progressive muscular atrophy (fig. 16 and Table VI.).

(v.) *Peripheral Neuritis.*—I have had the opportunity of examining the muscles from only one case of peripheral neuritis, and in this case the atrophy of the muscles examined is not extensive; therefore it is a case of no great value in regard to the condition of the spindle. Three muscles were examined—the extensors of the wrist, the supinator longus, and the extensor longus digitorum. On microscopical examination of the first two named muscles, very little change is noted in the muscular structure. In the extensor longus digitorum there is a considerable amount of fat, some fibres considerably atrophied, others normal in appearance and size. In the extensor longus digitorum a muscle-spindle is seen in the middle of a completely atrophied area, the walls and muscle fibres appear to be normal, sections from Marchi's solution show no change, and staining with Pal's method shows the nerve fibres are normal. The other muscles stained in a similar manner also show the normal spindle. In the teased specimens the intrafusal muscle fibres preserve their striation, the extrafusal muscle

PLATE VI.

Fig. 13.

Transverse Section of Biceps from a Case of Myopathy.

To show the prominence given to the muscle spindle by atrophy of the muscle—three spindles in transverse section are seen.

Diameter of spindle .. .. ·18 mm.

Magnified 44 diameters.

Fig. 14.

One of the above spindles under a higher power. The sheath is well shown, the intrinsic muscle fibres are equal in size to the extrinsic (compare fig. 4). The varying size of the intrafusal muscle fibres is well shown.

Diameter of spindle	·18
Intrafusal muscle fibres	·024

Magnified 180 diameters.

PLATE VI.

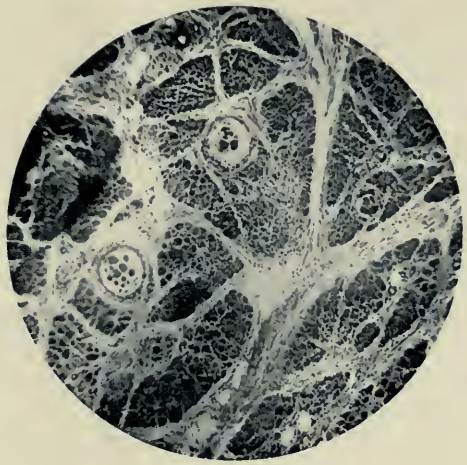

FIG. 13.

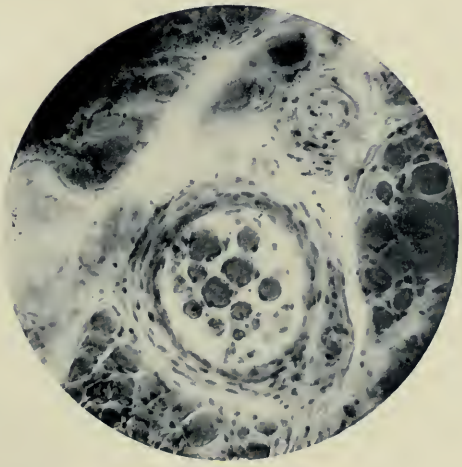

FIG. 14.

TABLE V.

Myopathy.

Boy aged 5.

Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibres.	Size of Spindle.	Size of Intrafusal Muscle Fibres.	Nerve Staining within Spindle.
Biceps	Atrophied	·024 ·008 ·048	·180 mm	·032	Nerve well stained
Pectoralis major	Atrophied	·04	·160 mm	·02	Nerve well stained
Serratus	Atrophied	·054	·12 mm	·024	—
Pectoralis minor	Atrophied	·036	—	—	—
Quadriceps	Atrophied	·024	·1 mm	·024	Nerve well stained
Gluteus	Very fatty	·016	—	—	—
Rectus abdominis	Normal	·04	—	—	—

TABLE VI.

Progressive Muscular Atrophy.

Case.	Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibre.	Size of Spindle.	Size of Intrafusal Muscle Fibre.	Nerve Staining.
I. Man	Biceps	Atrophied	·044	·14	·02	Well stained
II. Man	Abductor minimi digiti	Atrophied	·04-·008	·09	·02	Stained
III. Man	Abductor minimi digiti	Atrophied	·012	·14	·016	Well stained
	Flexor brevis	Atrophied	·012	·16	·024	Feebly stained

PLATE VII.

Fig. 15.

Compound Spindle from Biceps of Myopathy.

Seen in Transverse Section. Stained by Pal's Method.

This is probably an instance of a quadruple spindle, it is noticeable that the spindles are cut in varying parts of their course. Six divisions are seen, two of these (NN) contain nerve fibres as shown by the staining method, the other four contain muscle fibres. It is obvious that the poles of these spindles cannot lie on the same plane.

Diameter	·34 mm.
Nerve fibre	·004 mm.

Magnified 47 diameters.

170'

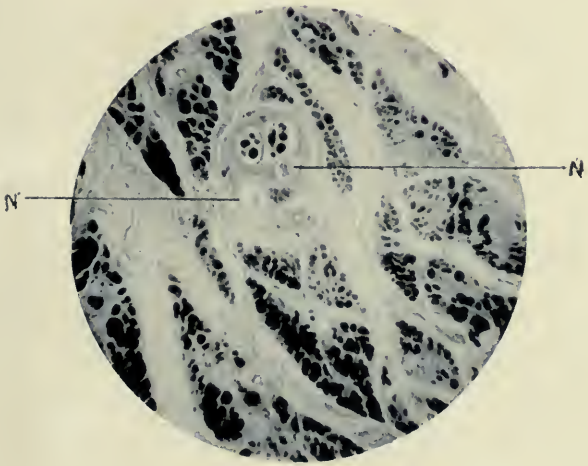

FIG. 15.

fibres have, in many fibres, lost their striation, and have a granular appearance. So far as one may judge from this case, one may say that in peripheral neuritis the spindle and its nerve remain intact after the muscle has become extensively atrophied; this is not, however, in agreement with Gudden's (16) statement (Table VII.).

(vi.) *Injury to the Brachial Plexus, with Complete Loss of Motion and Sensations of a year's standing.*—In this case, I have examined the biceps, triceps, muscles of the thumb, muscles of the little fingers, flexors and extensors of the wrist, and the ulnar and median nerves. All these muscles show most extensive atrophy; in many there are no recognisable fibres present, but in the biceps and triceps a few remain.

The ulnar and median nerves both show a very few fibres stained by Pal's method, which appear normal (these fibres measure about ·004 mm., and one or two ·008 mm.).

What is the condition of the muscle-spindle under these conditions? The spindle can be recognised in most muscles on careful examination, but it does not stand out in contrast to the surrounding tissue; all the spindles are small, the intrafusal muscle fibres atrophied, and the nerve fibres to the spindle do not stain by Pal's method. (There is one exception in a section of the triceps, one nerve fibre having taken the stain.)

In this case I have had considerable difficulty in teasing out the muscle-spindle; this I attribute partly to the fibrous increase in the muscle, and partly to the atrophy of the spindle. In the spindles which I have, however, teased out, the nerve is atrophied, the shape of the intrafusal muscle fibres can still be seen, but the transverse striation is almost completely lost and the fibres have a granular appearance; the usually darkly-stained portion in the equatorial region is absent, pointing to degeneration occurring in the intramuscular nuclei in this region. The spindle sheath can still be distinguished, but is not so distinct as normally. (Fig. 17).

It would seem, therefore, from the examination of the above, that atrophy of the spindle and its contained muscle fibres does take place, but at a much later period than the ordinary muscle fibre.

PLATE VIII.

Fig. 16.

Muscle Spindle from Hypothenar Muscles of Progressive Muscular Atrophy.

To show unaltered condition of muscle spindle in this disease.

The intrinsic muscle fibres retain their normal size and striation, while the surrounding muscle fibres have undergone extensive atrophy, being reduced to $\frac{1}{10}$ of their normal size.

Diameter of spindle .. .. ·14 mm.

Magnified 180 diameters.

Fig. 17.

Muscle Spindle from Hypothenar Muscles of a Case of Injury to Brachial Plexus.

Transverse section.

Two spindles are seen in transverse section lying in areas where extensive atrophy has taken place. The muscle fibres are smaller than normal, and appear shrunken.

Magnified 120 diameters.

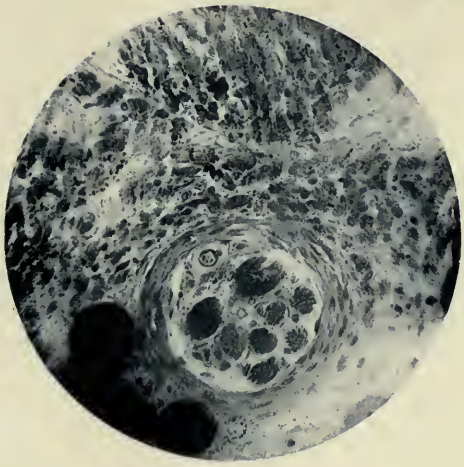

FIG. 16.

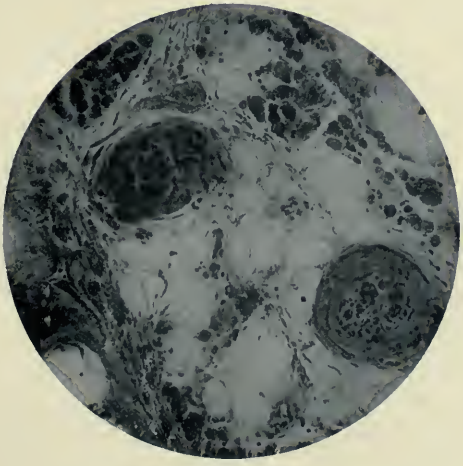

FIG. 17.

TABLE VII.

Peripheral Neuritis.

Case, Woman.

Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibre.	Size of Spindle.	Size of Intrafusal Muscle Fibre.	Nerve Staining.
Extensors of wrist	Not much atrophied	·048	·2	·02	Well stained
Supinator longus	Irregular in size	·08—·02	·2	·016	Well stained
Extensor longus digitorum (leg)	Atrophied	·1—·008	·18	·02	Well stained

TABLE VIII.

Injury to Brachial Plexus.

Muscle.	Condition of Muscle.	Size of Extrafusal Muscle Fibre.	Size of Spindle.	Size of Intrafusal Muscle Fibre.	Nerve Staining.
Biceps	Atrophied	·06– ·004 mm	·072	·02	Absent
Triceps	Atrophied	·048—·004	·08	·02	Present (one fibre)
Thumb muscles	Atrophied	·02—·004	·08	·012	Absent
Hypothenar	Atrophied	·02—·004	·16	·012	Absent
Extensors of wrist	Atrophied	·02—·004	·052	·008	Absent
Flexors of wrist	Atrophied	·02—·004	·01	·016	Absent

(xii.) *Section of Sciatic in Cats*[1]—three weeks, one month, two months, three months.—I have (thanks to the kindness of Dr. Risien Russell) had the opportunity of examining the gastrocnemius of four cats, at various times after complete section of the sciatic. The gastrocnemius of the cat is composed of two parts, one being white muscle fibres and the other red. I have examined muscles at the following times, after section of the sciatic: three weeks, one month, two months, three months, and have also examined

Table IX.

Cats—Section of Sciatic.

Muscle Gastrocnemius.

Cat.	Red Muscle.	White Muscle.	Size of Spindle outside.	Size of Spindle inside.	Intrafusal Muscle Fibre.	No. of Fibres in Spindle.
Normal	·052	·047	·12	·08	·0216	5
Cat I. (3 weeks)	·0412	·036	—	—	—	—
Cat II. (1 month)	·0505	·034	·16	·08	·018	7
Cat III. (2 months)	·0256	·0228	·1 ·096	·064 ·056	·0143 ·019	7 5
Cat IV. (3 months)	·0184	·0152	·048	·032	·019	7

the normal muscle for comparison. In the normal muscle, the extrafusal fibres measure between ·04 to ·06 mm. The white fibres are somewhat smaller than the red. The intrafusal fibres measure about ·02 mm. Muscle-spindles occur in both forms of muscle, and to an equal extent, so far as I can judge. Table No. IX. shows the comparative size of the red and white muscle fibres at the various periods after section of the nerve, and also the size of the intrafusal muscle fibres. The

[1] From Experiments performed at University College Laboratory.

table tends to show that atrophy takes place rather more rapidly in the white muscle fibres than in the red. I cannot say there is any definite atrophy in the intrafusal fibres. Sections stained by Pal's method show that the nerve to the spindle is degenerate in all cases. Striation is certainly well-marked in the intrafusal fibre three months after section of the nerve, and Sherrington has shown that striation still persists 150 days after section of the nerve. I believe, however (as the case above related tends to prove), that degeneration does take place in the intrafusal muscle fibres if sufficient time is allowed to elapse.

Summary.

Historical.—Although certain observers, fifty years ago, seem to have described in muscle, bodies which we now recognise as muscle-spindles, Kölliker was, however, the first who gave a detailed description of these bodies, and soon afterwards Kühne added considerably to the description of Kölliker, and introduced the name "muscle-spindle"; both these authors regarded the spindle as a stage in the development of muscle. Then followed a series of authors who regarded the spindle as a pathological condition occurring in atrophied muscle, and even as late as 1890 one author still regards them as pathological. Sachs, in 1874, fully recognised the sensory termination in muscle, but it was not till 1888 that Kerschner argued this sensory nature of the muscle-spindle, and suggested that their function might be connected with the sense of position. In the same year Cattaneo suggested that the muscle-tendon organs described by Golgi had a similar function, and proved experimentally that these bodies were connected with the posterior nerve roots. Then follow a series of authors working with normal and pathological material, some of whom adopted Keschner's view, others again adopting the view of Kölliker. Sherrington definitely proved, by degenerative experiments, that the nerve fibre from the muscle-spindle passes up in the posterior nerve root. All the later evidence points to the sensory

nature of the muscle-spindle. The muscle-spindle has been shown to be a spindle-shaped body, composed of a sheath resembling the Henle sheath of a nerve; within this sheath are contained small muscle fibres, which at one point become densely nucleated, and lose their striation. Two or more nerve bundles enter the muscle-spindle, and terminate —(i.) in the muscle fibres, (ii.) between the muscle fibres, and (iii.) in the sheath. The spindle also contains blood-vessels and lymphatics. These spindles are found in nearly all muscles of the body, except the eye muscles, the intrinsic muscles of the tongue, and the diaphragm. It has been shown, in the present paper, that in infantile paralysis the spindle remains absolutely normal, although the surrounding muscle tissue undergoes complete atrophy. In tabes it is shown that certain changes take place in the termination of the nerves, the general structure of the spindle remaining normal. In myopathy, the spindle and its contained nerve are normal. In progressive muscular atrophy the spindle remains unaltered, and the same is probably true with regard to peripheral neuritis. Section or injury of nerve trunk leads to atrophy of the muscle fibre within the spindle, though it is probable that it takes a considerable length of time for changes to take place in the muscle fibre within the spindle.

In conclusion, I would wish to take this opportunity of thanking the pathologists of the National Hospital, Dr. Colman and Dr. Risien Russell, for the kind manner in which they have assisted me in my work. To many others I would also tender my thanks for having allowed me to make use of material which I needed for my work, and especially to Dr. Barlow, Mr. Willett, and Dr. Kanthack.

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ON A METHOD OF DEMONSTRATING SECONDARY DEGENERATIONS OF THE NERVOUS SYSTEM BY MEANS OF PEROSMIC ACID.

BY D. J. HAMILTON, M.B.

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ALL who have had practical experience of Marchi's perosmic acid method of demonstrating secondary degenerations of the central nervous system, must have found that there are certain drawbacks to its use which render it almost completely inapplicable for entire brains or large masses of the same. When it succeeds, no doubt, the result is highly to be commended. The clearness with which the oil globules can be shown in the degenerated tracts is beyond all praise.

The reaction is always most successful in small pieces of tissue, such as short segments of the spinal cord, a nerve trunk, or a piece of brain substance of cubic capacity not greater than one or two centimètres. Even in the case of a large spinal cord, such as that of man, only a limited length, amounting to something like a couple of centimètres, can be stained successfully by means of it. When larger pieces of tissue are immersed in the solution, the perosmic acid often fails to penetrate to the interior. So great are the hardening properties of the solution, and so rapidly does it act, that the cord or other piece of nerve tissue placed in it is apt to be, so to speak, "case hardened." The outside becomes quite horny in consistence, and perfectly black, while the interior remains uninfluenced. When this has happened, no amount of renewal of the solution or increase of its strength will cause the perosmic acid to penetrate

further. Indeed, with the use of these expedients the tendency is rather to increase than to decrease the evil. The exterior becomes so brittle that it is found to be impossible to cut sections, even after the most careful imbedding in celloidin. In the case of an entire brain, even of a small animal like a cat or monkey, no reliance can be placed on the solution penetrating to the interior. The stain of the surroundings, moreover, is apt to be too dark, so that there is difficulty in seeing the oil globules. Then the quantity of perosmic acid necessary is large, and involves very considerable outlay. With experimental brains these difficulties are almost insuperable, and it was after the bitter experience gained by a succession of failures in such cases that I resolved to start an inquiry, now many months ago, with the view of finding out whether there was not some means of applying the perosmic acid more to be relied upon than that comprised in this otherwise estimable method.

Did the degenerated tissue stain with Marchi's liquid when cut into sections, the difficulties would be in great part overcome, but one of the most evident facts connected with the process is that neither by the ordinary method nor by any modification of it is it possible to call forth the reaction after the sections have once been cut. For months I worked at every conceivable means whereby this much-to-be-desired result might be attained, but in all instances with signal failure. A spinal cord, for instance, which gave an exquisite reaction in the degenerated tracts when stained *en bloc*, utterly failed to show a single oil globule when treated in section by the reagent.

It was only lately that I discovered what appears to be the explanation of this most remarkable phenomenon. When the Müller's fluid is entirely removed from such a cord by prolonged soaking in running water, the reaction with Marchi's solution is no longer forthcoming. A piece of the above cord, which gave a brilliant reaction when transferred to the Marchi's solution directly from the Müller's fluid in which it had been hardened, failed to do so when the Müller's fluid had been removed from it.

It struck me, therefore, that there must be something contained in the tissue hardened in Müller's fluid which is soluble in water, and which is the means of fixing the perosmic acid upon the globules of oil. If this theory be correct, the substance contained in the hardened tissue ought, when separated, to have the same properties. The substance in question may probably be soluble also in spirit, ether, &c., and the cause of the section not staining with the Marchi's reagent may be that it is washed out in the process of imbedding, cutting, &c. The substance must be a product formed slowly in the hardening of the part, for it is those tissues which have been longest immersed in the Müller's fluid which give Marchi's reaction best.

Acting upon this observation, I pounded up in a mortar a piece of brain which had been for many months in Müller's fluid, with some of the Müller's fluid in which it had been hardened, and filtered off the liquid after reducing the mass to a thin cream-like consistence. The liquid which came through the filter was perfectly transparent, and, with the exception of being a little darker, did not differ from ordinary Müller's fluid. Two parts of this liquid were mixed with one part of a 1 to 100 solution of perosmic acid, as in the making of Marchi's reagent. To my delight, after I had kept sections of the cord above referred to in the mixture at a body temperature for twenty-four hours, a decided reaction was visible in the oil globules in the degenerated parts. So decided was it that even in unclarified preparations the globules could be readily enough detected. The reaction, however, was not so good as when brought about by Marchi's method. Neither were the blackened oil globules so numerous, nor were they so black as when the parts were treated by the ordinary means. Still there was evidently something contained in the solution which had the property of fixing the perosmic acid upon the oil globules in a manner which I had not been able to obtain by any previous means.

It struck me that, by placing the sections, after being stained in the solution, in a reducing liquid, the blackening of the oil globules might be much intensified. I used

for this purpose a mixture of pyrogallic acid, glycerine, spirit, and water, but found that when the preparations had been in this at a body temperature for twenty-four hours they were so intensely black all over that the oil globules were lost in the universally black background. On treating the sections, however, with solution of permanganate of potash, so as again to oxidize the reduced perosmic acid, and subsequently decolorizing in sulphurous acid, the blackening left the surroundings and remained upon the oil globules.[1]

The latter stood out in a manner comparable to the best results obtained by Marchi's method.

The solution of perosmic acid, however, seemed to me too strong. It took too long to remove the stain from the surroundings, and I, accordingly, went on diluting it, and studying the results. As the sum of the various trials made, I found that a mixture of one part of a 1 to 100 solution of perosmic acid in distilled water to *two hundred* of the filtered brain liquid is best.

Employing such a diluted solution of perosmic acid (1 to 20,000), the degenerated fibres do not blacken until acted upon by the reducing agent. The section, when taken out of the liquid, has a universal brown colour, without any black points being visible upon it. But in some unaccountable manner the liquid has the property of fixing the perosmic acid upon them more than upon the surroundings.

In order to test the staining powers of this brain liquid solution of perosmic acid as compared with other solutions of perosmic acid, I cut a number of sections of a human spinal cord secondarily degenerated after six weeks' com-

[1] Heller, I found, had also used permanganate for the purpose of removing the reduced perosmic acid from the surroundings of stained, medullated, healthy nerve fibres. He employs oxalic acid for the purpose of destroying the brown colour imparted by it to the tissue. I prefer sulphurous acid. My own observations on the employment of a reducing agent followed by permanganate were made independently of, and before his were published. The sulphurous acid removes the colour imparted to the tissue by the permanganate in virtue of its strong affinity for oxygen, converting, as it does, the permanganic acid into the colourless protoxide of manganese. A ferrous salt has the same action.

pression, and placed some of them in each of the following solutions: (*a*) Brain liquid 200 parts, and perosmic acid solution in distilled water (1 to 100) 1 part; (*b*) Müller's fluid 200 parts, and perosmic acid solution in distilled water (1 to 100) 1 part; and (*c*) distilled water 200 parts, and perosmic acid solution in distilled water (1 to 100) 1 part. They were all treated subsequently in exactly the same way. In the case of *a* the staining of the degenerated tracts was intense; in that of *b* only a fibre here and there was stained; and in that of *c* the degenerated tracts were practically unstained.

On comparing sections of this cord stained by Marchi's method with those of the same stained by my own, I was struck by the fact that the reaction in the latter was more delicate than in the former. Long before oil globules had made their appearance in the degenerated fibres, at the time when the myeline sheath was breaking up and giving rise to a varicose aspect of the fibre, a decided reaction was obtained. In a case of ascending degeneration of the cord, for instance, over and above the intense blackening in the recognised ascending tracts, odd fibres, having a disintegrated appearance, were found scattered over the section, which gave an intense black reaction. These were only faintly touched by Marchi's method, and many of them were not indicated at all. What was also most striking in this same cord was that the crossed pyramidal tracts in the part above the point of compression were devoid of any blackened fibres, and the fibres within them were seen to have a perfectly normal aspect. Axis-cylinder and myeline sheath were each undisturbed.

It might be asked, however, what is the effect upon a normal cord or brain? Does the black stain not remain upon the normal nerve fibres? In order to test this, I used a human medulla oblongata from an adult, the brain of a kitten, the cords from a monkey and rabbit, and the brain, cord, and nerves from a hedgehog. They were all hardened in Müller's fluid and treated as described subsequently in detail. In none of them, after being the prescribed length of time in the permanganate of potash, could I find a single

nerve fibre which had given the black reaction. A black precipitate occasionally formed here and there, probably some compound of the iron of the blood with the pyrogallic acid, but the colour of the section, otherwise, was a universal greenish-grey. The irregular varicose aspect of those fibres which stained black in degenerated cords, together with the breaking up of the myeline sheath, was additional proof of their being abnormal.

Such are briefly the outlines of the method. Its success seems to depend upon something being contained in the brain hardened in Müller's fluid, which is soluble in various media, and which has the property of fixing the perosmic acid upon the fattily degenerated parts more than upon those which are normal. When, consequently, the reduced perosmic acid is oxidised by the permanganate, the fattily degenerated parts lose their colour less readily than the surroundings.

After many trials and modifications of the method, I find the following procedure gives the best results :—

The parts must be hardened in Müller's fluid. The human brain, or brains of large animals, should be *injected* with the same, after the manner described in my "Text-book of Pathology," vol. i., p. 56. From three to four months' time is requisite to complete the hardening. The pieces of tissue to be cut, without being washed, are dehydrated and imbedded in celloidin. It is a remarkable fact that, even when they are thoroughly dehydrated with absolute alcohol, subsequently treated with ether and alcohol, and left, it may be for months, in an ethereal solution of celloidin, the oil globules are not washed out. Indeed, the staining capacity of the part does not seem to be altered in any way.

As the sections are cut they are placed in a capsule containing dilute alcohol, one above the other in serial order, and, if necessary, separated by pieces of thin paper.

They are next enclosed in a double film of collodion. Some thin collodion is poured over a slide and allowed to become firm on the surface, not too dry. The slide is then immersed in methylated spirit in a flat dish, and a section,

or several sections, as the case may be, are placed upon it. A *printed number* of small size, corresponding to the place of the section in the series, is then fixed on the film of collodion close to the margin of the section. The spirit is dried from the slide by covering it with a piece of white tissue paper and pressing over this with a sheet of blotting paper. The tissue paper prevents the fibre of the blotting paper from adhering to the collodion. A second layer of collodion is now poured over the surface so as to encapsule the section and its attached number. When this again has become firm, not too dry, the slide is plunged for a few seconds or longer into methylated spirit, and the film of collodion with the section included stripped off. The margins are next clipped so as to leave a border of about 1 cm. all round, inclusive, of course, of the printed number. The sections can be kept permanently in pure methylated spirit or other form of dilute alcohol.

A piece of brain which has become quite brown by being kept in Müller's fluid for many months, it may be years, is pounded up in a mortar, and converted into a thin cream-like consistence by the addition of Müller's fluid. The Müller's fluid in which the brain employed has been hardened is best, but ordinary Müller does well enough. About half a kilo-weight of brain to the litre of Müller is sufficient. When the two are thoroughly incorporated, the liquid is filtered off through paper, and of this 200 cc. are mixed with 1 cc. of a 1 to 100 solution of perosmic acid in distilled water. The mixture is placed in a wide-mouthed stopper bottle, and the stopper is anointed with glycerine to render it air-tight. If necessary, the stopper should be tied down to prevent it becoming displaced. The incapsuled sections are immersed in a considerable excess of the reagent, and the bottle is kept in an incubator at a body temperature. The use of the incubator seems to be essential for success. They are left in the solution for twenty-four hours, and thereafter are taken out and washed for a few seconds in water. The bottle must next be thoroughly cleansed, and sufficient of the following reducing fluid introduced to cover the sections :—

Pyrogallic acid	1 grm.
Glycerine...	50 cc.
Rectified spirit or absolute alcohol	50 cc.
Water to...	400 cc.

The stopper is anointed with glycerine, replaced in the bottle, and the bottle put into the incubating chamber for twenty-four hours.

The sections and the bottle are again thoroughly washed in water. Particular attention must be given to seeing that this is done effectually. If any of the reducing fluid remains, the permanganate solution becomes decomposed and precipitated. Indeed the sections should be left overnight in water. The bottle is now filled with $\frac{1}{4}$ per cent. permanganate of potash solution, and the sections are immersed in this. The stopper is replaced, but the bottle is *not* put in the incubator. It is left at an ordinary sitting-room temperature. After a day's immersion, the sections are taken out, washed in water, and treated with sulphurous acid. The strength of the sulphurous acid does not make much difference; that of the British Pharmacopœia acts very quickly. Weaker solutions, however, may be employed, a longer time being allowed for decoloration. When thoroughly decolorised, the sections are again washed, and placed for twenty-four hours in the permanganate solution, and so on for three days, the decoloration with sulphurous acid being practised after every twenty-four hours—that is to say, three times in all. After the three days' treatment with permanganate and sulphurous acid, they are again subjected to the reducing fluid in the incubator for twenty-four hours. The object of this last proceeding is two-fold. The decoloration of the healthy parts of the section by the permanganate is so complete that, when clarified, they become almost invisible. The immersion in the reducing liquid over-night gives them a greenish-grey colour, and so renders them apparent. A second effect is that the degenerated fibres, blackened by the process up to this point, are rendered literally jet-black by the night's immersion in the reducing fluid.

The last part of the procedure is to wash thoroughly in water, to dehydrate by immersing in several relays of dehydrated spirit followed by absolute alcohol, to clarify in oil of cloves or carbol-xylol, and to mount in dammar lac. The dehydration must be perfect, and to this end it is necessary to place the sections in excess of alcohol. For the first dehydration I use ordinary dehydrated methylated spirit, pouring an ounce or so into a capsule and immersing the sections in it for five to ten minutes. They are afterwards placed on a slide, and the excess of spirit removed by application of tissue and blotting paper as before described. Thereafter absolute alcohol is poured twice over the section, and allowed to remain in contact with it for a few minutes. The excess is removed from the margins with a soft cloth, not by using blotting paper. The most satisfactory method of clarifying is to plunge the slide, with the section upon it, into the clarifying agent, and to leave it in this until clarification is completed. The superfluity of the clarifying medium is removed with a soft cloth, and the section mounted in dammar lac.

The oil globules blackened by Marchi's method are liable to fade after being mounted for some time. So far as I have yet seen, after several months, those stained by my own method, if thoroughly washed after use of the sulphurous acid, are not.

The infinitesimally small quantity of perosmic acid necessary for my process does away with the objection of expense. The solution, moreover, does not require to be renewed, for by twenty-four hours the impregnation with perosmic acid is completed. I do not recommend using the solution more than once. A quantity sufficient to cover the sections will generally be found enough, but I always allow a considerable excess.

CASE OF BRAIN TUMOUR WITH AUTOPSY.[1]

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THE chief interest in this case lies in the question whether operation might have been successfully performed This question has become of vital moment since the reports of successful removal of extensive infiltrating gliomata, especially those of Beevor and of Wood.

Case.—Mr. H——, of Salem, a patient of Dr. Phippen, was seen by me in consultation with Dr. Phippen and Dr. Johnson, October 29, 1894. The history up to that time was as follows: He was well up to the preceding July, when he complained of difficulty in figuring and trouble with the trial balance. He seemed indifferent about his vacation, and said that he did not feel well. Those in the office think that the symptoms had been coming on for six months. He was a man 53 years of age, of sedentary occupation.

September 12.—An attack with loss of speech and twitching of the face occurred, lasting perhaps ten or fifteen minutes. It was not noted on which side the spasm occurred. This attack was followed by indistinctness of speech and apparently by some aphasia. On September 13, a queer sensation in the throat suddenly appeared, lasting only a minute. On September 19, he had an attack of loss of speech. This was quickly over, but for the next ten days he had a peculiar feeling in his throat.

September 30.—A severe general convulsion occurred, with a long warning. Deep coma lasted ten to fifteen minutes, followed by confusion for some hours.

[1] Shown at a Meeting of the American Neurological Association, June, 1895.

October 9.—A convulsion occurred on the street, with some warning. His sister thinks the eyes turned to the right in this attack. He did not return again to work. The pulse was poor and the recovery slow after the deep coma of this last attack passed away, and aphasia continued up to the date of this examination.

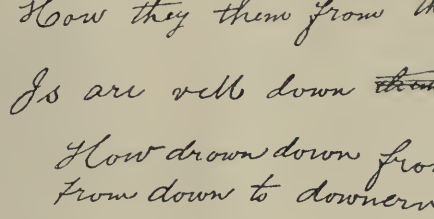
October 14.—A slight attack with loss of consciousness occurred, with twitching in the neck and spasmodic drawing of the jaw to the right. After this attack there was repeated twitching of the right pectoralis-major muscle. The aphasia rapidly increased from this time. On October 27 the patient began to drag his right foot, and to find difficulty in holding a spoon. During the day his face became drawn to the left. The next day (October 28) he first complained of headache, indicating the frontal and left parietal regions. There was no dysphagia. The same day he had a dizzy turn in the garden. The pupils have been dilated during attacks. There has been no complaint of numbness.

Physical Examination.—The patient is not confined to his bed. He walks about with some difficulty, with toe drop on the right side. He stands very well with his eyes closed and feet together. The pupils are alike and react to light. His grasp is good on the left, but weak on the right. He cannot button his vest with the right hand. There seems to be also slight numbness in the right hand, but it is impossible to make an accurate test. There is complete loss of visual field for objects on the right (right hemianopsia). Both knee-jerks are normal. The left plantar cremaster and abdominal reflexes are lively on the left, absent on the right. The epigastric is moderate on the left, very slight on the right. The tongue is deviated to the right. The nutrition is fair.

The patient appeared to understand all questions put to him, but to be unable to reply. He had the appearance of being quite intelligent, indicated the left frontal region when asked if he had headaches, opened the mouth, protruded the tongue, and shut the eyes without hesitation when requested, but when asked questions requiring an answer, hardly brought out a word, though apparently making a great effort to express himself. When asked to name objects he nodded the head, or said "yes," when the object was rightly named by another, but could not repeat, for example, the words "book" or "handkerchief." He could not say the alphabet, partly apparently through aphasia, and partly through difficult enunciation. The difficulty of testing his power of

speech was somewhat complicated by the fact that he stammered previously to the onset of the disease.

A facsimile of his writing is given :—



The chirography is perfect, but sense is lacking, the writing consisting of an endless repetition of the same words. It will be noted that in two words only incorrect letters were used.

Examination of the fundus showed commencing double optic neuritis.

The comparative absence of headache, and the character of the writing, led to the consideration of general paralysis as well as of tumour. The apparent intelligence of the patient, and the comparative stability of paralysis, rendered the former diagnosis improbable, while the presence of optic neuritis, and of hemianopsia absolutely established the presence of new growth. This case illustrates the importance of testing the field of vision in case of suspected tumour of one of the hemispheres. Glioma was regarded as one of the most probable pathological diagnosis, though the patient was given the benefit of every doubt in treatment.

The question of operation was discussed, but the family were averse unless definite prospect of relief was offered. They were informed that on account of the probable size (determined by the combination, hemiplegia, aphasia and hemianopsia), and on account of the probable infiltrating character of the growth, surgical interference offered only a forlorn hope. We did not even venture to assume that the temporal lobe and angular gyrus were spared, though no word blindness nor word deafness was detected, the patient's inability to express himself increased so far the difficulty of discovering a moderate degree of these defects. In a case reported by Dr. M. H. Richardson and myself not long since,[1] the entire angular gyrus and posterior temporal region were

[1] *American Journal of Medical Science*, December, 1893.

extensively involved in the growth, though only moderate indications of word blindness and word deafness were present.

The patient grew steadily worse, and died about one month later. Dr. Phippen removed the brain, and forwarded me the left hemisphere, which presented the following character:

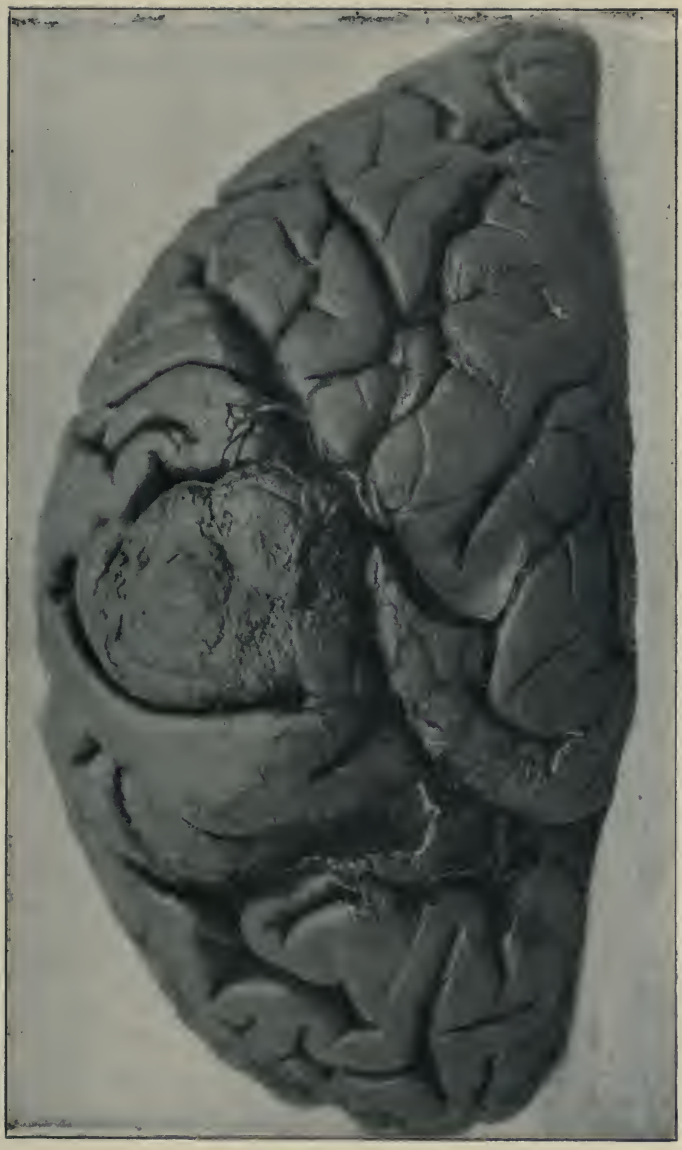
Autopsy.—The entire surface of the brain appeared normal, excepting at the junction of the Rolandic and Sylvian fissures. At this point a round tumour appeared, level with the surrounding surface, and quite sharply defined. Its consistency was approximately that of the brain substance, it was dark grey in colour. It measured two by one and a half inches. A vertical section through the growth showed a fairly sharp gradation from the tumour substance to the surrounding, apparently healthy, tissue of the centrum ovale, though no distinct capsule. Its centre was composed of white matter of softer consistency and containing hæmorrhagic spots. This was apparently the result of necrotic growth.

Section just behind the ascending portion of the interparietal sulcus showed normal brain substance. Successive sections anteriorly showed extension of the growth under the healthy cortex, beyond the transverse frontal sulcus, the termination being quite sharply marked at a point between the second and third frontal convolutions.

The following is the report of the microscopical examination by Dr. Taylor:

"The tumour given me for examination consists of a highly cellular new growth, infiltrating the surrounding brain tissue. The nuclei vary considerably in size and to a certain extent in shape, but never approach the spindle-cell variety. The transition from normal brain tissue to the pathological area is a fairly abrupt one, but without the slightest evidence at any point of encapsulation. The tumour is highly vascular, the vessels filled with blood and in certain instances containing thrombi. There is a slight amount of free hæmorrhage. In many parts of the new growth are areas of necrosis in varying stages. The tumour probably belongs to the class of gliomata.

"In the light of our present knowledge it should be stated that an absolute definite diagnosis is possible only through the use of the recently published 'Weigert-Mallory methods of differential neuroglia staining, methods which had not yet appeared when the above examination was made. There can be but little doubt that these methods are to throw the first really clear light on the many disputed points connected with the histological structure of the gliomata as distinguished from the sarcomata.'"



The result of the autopsy, together with the fact that since that time cases have been reported of successful removal of extensive infiltrating gliomata in this region, to say nothing of a case of Dr. Putnam's (not yet reported) in which Dr. M. H. Richardson has recently removed from this locality a gliosarcomatous growth, weighing at least a pound,[1] would lead me in a case presenting similar symptoms to-day, to regard operation somewhat more hopefully than I did at that time, though experience leads us to be far from sanguine regarding the final outcome of such operations, notwithstanding individual successes, however laudable and even urgent this procedure may become as a last resort in otherwise hopeless cases.

[1] In this case the recovery, Dr. Putnam informs me, was comparatively temporary, the patient succumbing about a year later, from extension of the process.

THE DIET TREATMENT OF HEADACHE, EPILEPSY, AND MENTAL DEPRESSION.

BY ALEXANDER HAIG, M.A. AND M.D.OXON., F.R.C.P.

In several previous articles[1] I have dealt with the etiology and pathology of these troubles, and as the treatment by diet is the direct outcome of this pathology, and is now, I believe, likely to be used more extensively by others than has hitherto been the case, I propose to record shortly the more important points in my practical results up to the present time.

The above-mentioned pathology makes these troubles due to the disturbing influence of high blood pressure on the circulation of the brain. The troubles are associated, as I have shown, in a definite and distinct manner with the presence of excess of uric acid in the urine, and this is due to the simple fact that a similar and contemporaneous excess of uric acid in the blood is the direct, and probably the mechanical, cause of obstructed capillary circulation throughout the body, and so of high blood pressure.[2]

It follows from this that uric acid is the cause of all these troubles, in so far, at least, as they are not due to other obvious mechanical causes, which I need not mention; and the treatment by diet aims at clearing the blood of uric acid, and keeping it clear. Now as uric acid, or xanthin and its compounds, which are equivalent to it, are found in all kinds of fish, meat, fowl, game, their extracts and decoctions, also in eggs, and in tea, coffee, and cocoa, and similar vegetable

[1] Brain, Spring Numbers of 1891, 1893, and 1896.

[2] "Uric Acid as a Factor in the Causation of Disease," Third Edition, pp. 147, 172, 174, 344.

alkaloid-containing substances, all these things must be cut out of the diet. But as nitrogen is a necessary of life, these foods must be replaced by other foods of about the same nitrogen values, such as cheese and pulses; also by milk, cereal foods, and fruit, which contain little or no uric acid or xanthin.

The quantities required are not difficult to calculate, if we allow, in accordance with physiology, for an adult leading an active life 3½ grains of urea per pound, and for one who is sedentary 3 grains of urea per pound of body weight per day. Thus an adult weighing 140 pounds will require, if he is sedentary, 420 grains of urea per day, and if he is active 490 grains per day. Then, roughly speaking, urea, multiplied by 3, will give the albumen required to produce it, so that 1,260 grains of albumen would be required in the one case and 1,470 grains in the other. And as the percentages of albumen in the above foods are given in most works on food and hygiene, there is not much difficulty in calculating, with quite sufficient accuracy, how much cheese, pulses, milk, and cereal foods a given patient should have.

These preliminaries being settled, it is only necessary to add that nutrition, strength, and power of endurance should be quite as good on the one diet as on the other (as soon as the initial difficulties of taste and habit have been adjusted), and that, speaking generally, the diet does good in very many directions, and, as far as my experience goes, harm in none.

As regards the uric acid headache properly diagnosed and carefully treated by the above diet, my results have been most satisfactory. The headaches may improve at once, and, as the stores of uric acid previously in the body are eliminated, the attacks fall to one-eighth or one-tenth of their original frequency within twelve to eighteen months; the attacks also become at the same time much shorter and less severe. This improvement may set in at once almost from the day the diet is altered, or, on the other hand, the attacks may be decidedly more frequent during the first few weeks of the altered diet. It is necessary to be on one's guard against this, and to tell the patient beforehand that it is liable to occur; as, with those who do not understand its

causation, it may have a most disheartening effect, and lead to the treatment being abandoned.

Its causation is simply as follows. The excretion of uric acid in the urine is, other things equal, dependent upon its solubility in the blood, and its solubility is dependent on the alkalinity of the blood. With high urea and good nutrition the blood is less alkaline than with low urea and feeble nutrition. It follows from this that in conditions of low nutrition the blood will be flooded with uric acid from all the places of deposit in the body in which it has been previously stored. Therefore the blood will be flooded with uric acid if, in changing diet, the patient, either from habit as to the old diet, or positive dislike of the new diet, or simple want of appetite, takes it badly, and lets nutrition down.

Then, again, many vegetable foods introduce more alkali and less acid into the body than did the animal foods left off, and this is another cause for increased alkalinity of the blood and its consequent flooding with uric acid. From one of these causes, or both combined, the headaches may be decidedly worse during the first few weeks of treatment; but this is by no means an unfavourable sign as regards ultimate results, and is only one more proof, if any is needed, that the functional trouble is absolutely dependent on uric acid.

Where these headaches are sufficiently frequent and severe to call for treatment, I generally give a mixture containing bromide of ammonium and salicylate of ammonium, to be taken for a few days, from time to time as may be necessary to help them over the first rush of uric acid.

The same will apply almost word for word to epilepsy and mental depression, in so far as they are due to uric acid.

In mental depression there is almost always some bodily depression as well, and even if there is no dyspepsia the appetite is very poor indeed. Under these circumstances, a sudden change of diet is almost certain to produce some failure of nutrition, and the depression will be worse instead of better at the first start. Here it is very little use to pre-

scribe a diet, and leave it to the patient and friends to carry it out to the best of their ability, which is often very small.

I believe that the best plan is to put the patient to bed, so as to economise force when there is none coming in; to feed by rule carried out by a trained nurse, and to increase the quantities and add massage and passive movements as nutrition improves till sufficient force has been accumulated to allow of his being up and about once more.

Such mental depression has often seemed to me to be uric acid headache, with absence of appetite and failure of nutrition; nutrition being so bad that blood pressure falls (from heart failure) below the point at which headache is possible, and depression then takes its place; feed up such patients on meat and wine diet they will, perhaps, have a good bilious attack—headache, vomiting and all—and then be fairly well for a time; but they are now storing uric acid and are certain to relapse later on.

With mental depression, also, I have got many very good results, and most of my headache cases have suffered also from depression, and the two have cleared up together. Mental depression, however, rather tends to linger after the headache, and is apt to give trouble from time to time if nutrition is allowed to fail, especially in hot weather (spring and summer).

I have purposely left epilepsy till the last, because my results with it have been not nearly so good as in the case of the other troubles, and because it presents many serious difficulties to those who would attempt to estimate the real effects of any treatment. Of late years I have prescribed the diet for a considerable number of out-patients, and have got many reports of very decided improvement as to number, and still more as to severity of the fits. On the other hand, cases treated even for many weeks in hospital, and with great care outside, as to regulation of diet, have given only most disappointing results.

In some cases of epilepsy, just as in headache, the attacks may be more frequent and severe soon after the diet is altered. I was especially struck with this in the case of a boy who was under my care at the Metropolitan

Hospital a little more than a year ago. When he came under my care the friends were getting desperate, and ready to try anything; I accordingly put him on a very strict diet, and kept him in bed. He had previously been under observation for many weeks, and his fits had been frequent, generally two or three each day; then on my diet they soon became distinctly both more frequent and more severe, so that the resident medical officer and the sister of the ward became decidedly anxious about him, and a few doses of bromide were given, but otherwise he had no drugs.

However, I persevered with the diet, and in a week or two more the fits began to get less frequent and severe, falling to one a day and two in a day, and eventually a day, or even two, occurred here and there without any at all. At this point, however, he got scarlet fever, which had been introduced into the hospital, and going to one of the fever hospitals was lost sight of.

I have seen several other cases of epilepsy in which the fits, just as in this case, were distinctly worse when the diet was first begun, and my experience leads me to look on this as rather a favourable sign as to the possibility of future improvement, and one which strongly indicates perseverance with the diet.

Though my results with epilepsy cannot be called very favourable, they have been by no means entirely negative, and they furnish us, I think, as in the cases mentioned above, with one more parallel to be added to the very extensive series[1] which connect epilepsy with the uric acid headache, which is so extremely amenable to diet treatment. We must remember, however, that the uric acid headache is not, strictly speaking, cured; it is more commonly reduced from forty to fifty attacks a year down to three or four. The patient is always liable to suffer if he transgresses the diet, and he may suffer at any time after exposure to great cold or any unfavourable circumstance affecting importantly the solubility of the uric acid, which he still always has with him, though in lessened quantity. Regarded from this point

[1] See BRAIN, Spring Number, 1896.

of view, the results in some cases of epilepsy are not so very far behind, and I believe that in future, with more care and strictness in carrying out the diet, and in selecting early and suitable cases, we may hope for a considerable number of decided improvements, and here and there some cures, comparable at least to those of headache.

There is one other trouble due to excess of uric acid in the blood, which presents us with an exact parallel in its reaction to diet. I refer to cases of anæmia and chlorosis, and I have for several years been pointing out that my own blood actually alters its quality from day to day, in accordance with the amount of uric acid passing through it.

In cases of anæmia put on diet for headache or depression there is very often a fall in the value of the blood decimal during the first few weeks of treatment, followed by a rise; and at the end of six months, and still more at the end of twelve, it is considerably (often as much as 20 to 30 per cent.) above the point at which it stood when the diet was begun.

I mention this here because the blood is easily examined, and I believe its alterations afford us a very reliable index of the quantity of uric acid passing through it. In my own case the correspondence with daily observations of blood and urine is so absurdly close, that I often think I might give up estimating the excretion of uric acid in the urine, and trust to the changes in the blood to tell me whether it has gone up or down. An examination of the blood at the time the diet treatment is begun, and again after one month, three months, six, nine, and twelve months, will give you a very fair idea of the amount of uric acid that has been passing through the blood, and the effects of the diet upon it. And, speaking generally, if the blood decimal falls at the end of the first month it will probably have risen by the end of the third, and still more at the end of the year. Any very decided fall in the early weeks is an indication to look carefully after nutrition, and perhaps to give tonics to moderate a little the rush of uric acid.

The above facts, by the way, probably explain the common treatment of anæmia with excess of animal food,

because the primary fall of the blood decimal on other diets frightens those who do not understand its meaning and causation. But here again, just as in the diet treatment of headache, epilepsy and mental depression, the only road to complete success may run through this belt of apparent failure.

Reviews and Abstracts.

The Present Evolution of Man. By G. Archdall Reid. Chapman and Hall, 1896. Demy 8vo., pp. 370.

Reflex Action, Instinct and Reason.

The old time controversy as to whether animals can reason as long been settled in the affirmative, but the preliminary question of what the difference is between instinct and reason still remains an unsolved problem. Mr. Archdall Reid's offer of a solution in his recent book on Psychology appears to me unacceptable, because the basis of his definitions is unsound, their form unsatisfactory, and they do not stand the test of application to actual cases.

Mr. Reid defines instinct as "the faculty which is concerned in the conscious adaptation of means to ends, by virtue of inborn inherited knowledge and ways of thinking and acting," and reason as "the faculty which is concerned in the conscious adaptation of means to ends by virtue of acquired non-inherited knowledge and ways of thinking and acting." To regard instinct and reason as "faculties"—as separate entities—is a lingering remnant of the discarded "metaphysical" mode of treating psychological questions, which is out of harmony with current methods of thought, and is a cause of inevitable confusion. It is not possible to compare things so fundamentally different as the purely physical process of reflex action and a so-called "faculty" of reason, which is presumably mental. There is no possible basis of comparison between them. The mechanism of reflex action is as purely physical as the swing of a pendulum. It is no more possible to compare reflex action with a faculty of reason than to compare the swing of a pendulum with the working out of an equation. We may, indeed, if we can get a clear idea of what is meant by a faculty, compare the faculty of instinct with the faculty of reason, but we cannot compare either with the process of reflex action; and even if we term that process also a

faculty, we shall be no nearer to securing a basis of comparison. Mr. Reid appears to feel this difficulty, for though his chapter is devoted to reflex action, instinct and reason, and though he defines instinct and reason, he does not attempt to define reflex action. To define instinct and reason as faculties is, it is submitted, to define the unknown by the still more unknown, and so to sin against the primary canon of definition.

What we actually observe in men and animals, and can compare upon a common basis and by a common standard, are, not faculties, but acts. While we cannot compare a reflex act with a faculty of reason or of instinct, we can very easily compare reflex acts with instinctive acts and with reasoned acts; and to these acts our definitions, like our comparisons, must be limited.

No less unsatisfactory than the basis is the form of Mr. Reid's definition. A definition to be adequate must be clear; it must be intelligible. Mr. Reids definitions are neither. Apart from the term "faculty," which conveys no clear connotation, the way in which the faculty is "concerned" in the adaptation of means to ends is left unexplained. How is the faculty concerned? The term carries the implication that there are other factors also "concerned" in the process. If so, what are these factors? If not, the definition is faulty in not stating explicitly that instinct or reason is the faculty which *actuates* the adaptation, &c. Again, the expression "by virtue of" is by no means clear, either in itself or its connections. Is it the adaptation of means to ends which is effected "by virtue of" inborn knowledge, &c., or is it the faculty which is concerned "by virtue of" this knowledge, &c.? In either case, what is the influence which this knowledge, &c., exerts on the process? Not to appear captious, I will pass over the absence of any distinction being drawn between knowledge and ways of thinking, and will note a very serious defect in the definition of reason. The term is used in the colloquial sense in which it is commonly employed when "reason" is contrasted with "instinct," and though there is much "virtue" in the term "faculty," it appears that what Mr. Reid is really dealing with under the name of reason is a certain "way of acting." If reason is to be regarded as a faculty, in accordance with the old metaphysical method of speaking of the subject, we may pass this use of the term; but in a book on Evolution, which aims at presenting the results of modern psychology, it is expedient that the term should be used in the sense recognised among modern psychologists, as connoting a mental process. Lastly, it is extremely difficult to understand

what is meant by "a faculty which is concerned with the adaptation of means to ends by virtue of inherited . . . ways of action." Is not the adaptation of means to ends "a way of acting?" and if it be, what is the meaning of a way of acting by virtue of a way of acting? The definitions appear so faulty in form as, when critically examined, to be almost unintelligible; but let us accept them as they are, and apply their provisions to actual cases, and see whether they will stand the test of application; and in order to be quite fair, let us take the instances offered by Mr. Reid himself.

He adduces the case of the young alligator or turtle instinctively seeking the water on emerging from the egg. Is it seriously maintained that the new-born turtle or alligator has a *conscious knowledge* of the difference between land and water, of the direction in which water lies, and of the fact that progression in one direction will take him towards, and in the other direction away from water? If this is not asserted, the definition falls to the ground. If it is asserted, it can be denied, and of neither assertion nor denial can proof be adduced. It might similarly be asserted that the sunflower turns to the sun by virtue of a conscious knowledge of the sun's direction.

"By what term," says Mr. Reid, "shall we designate the action of the spider when he builds his web? Does the animal not know for what purpose he constructs it?" His definition of instinct (as the faculty which is concerned in the *conscious* adaptation of means to ends, &c.) depends upon the affirmative answer to this question. But the question cannot be certainly answered in the affirmative. We know nothing about the consciousness of the spider. We can only guess what it may be by analogy from our own consciousness, and while this analogy allows us to suppose that, on the second or third occasion of constructing a web, the spider knows the purpose for which the web is being constructed, it positively forbids us to suppose that he is so aware when, without previous experience, he constructs his first web. If we say that he must know, because he constructs the web instinctively, and instinct is the conscious adaptation of means to ends, we are arguing in a circle; and there is no other ground for the statement. It is evident that when the factor of consciousness is brought into the definition, a factor is brought in of which we are almost entirely ignorant, a factor about which contradictory assertions may be made without the possibility of either being proved or refuted, and, therefore, a factor which renders the definition valueless. Mr. Reid compares the action of the spider in running

along a thread to secure his prey with that of a man running to secure a snared bird; and that of the former cutting loose a dangerous captive with that of the latter in building a golden bridge for a flying enemy; and asks in what respect the actions of the spider differ from those of the man. "Only," he says, "in that the actions of the spider result from knowledge that is inborn and inherited, whereas the actions of the man result from knowledge which is neither inborn nor transmissible." In this statement there are many assumptions, and if any one of them fails, the distinction is invalid. Take two of them only—first, that the spider knows what the effect of his action will be; second, that no other difference between the acts can be found. Both are unwarranted. There is no warrant for the statement that the spider knows that the result of cutting loose a too-powerful captive will be to save his web from destruction, any more than that the ant knows, when she is hiding a pupa-sister, that she is helping to preserve the formicine body politic. We know nothing certainly about the knowledge of the spider in the circumstances, and to postulate that his act is the result of knowledge is a gratuitous assumption, opposed to what analogy we have to guide us. That there is another distinction between the acts of the spider and the man, a distinction that is not open to this objection, will presently be shown.

Precisely the same criticism applies to the stated cases of the alligator which seeks the water and snaps at an opposing walking stick; of the nesting bird; and of the bird which utters cries of warning; as compared with the man who seeks a refuge and strikes an intervening enemy; who builds a hut; and who warns his fellow of danger. Mr. Reid's distinction fails unless we assume that the alligator and the bird respectively foresee the results of their acts, and, since he brings it forward as the only one possible, it fails also if we can find another and clearer distinction.

Almost every sentence in Mr. Reid's chapter on this subject presents obvious points for criticism, but to notice them all would be tedious. It is necessary to notice a few which bear directly upon his definitions. "Appropriate stimulation," he says, "does not cause variations and developments in reflexes and instincts," and, again, on the same page, "instinct, which, though undeviating in its promptings, is associated with consciousness, and controlled to some extent by volition, whereby the element of choice is introduced." But if instinct is controlled to any extent by volition, and if the element of choice is introduced, then appropriate stimulation *does* cause variation in instinct. For if the element of

choice is introduced, the instinct is varied, and varied to better suit the circumstances, or what is the advantage of choice? And, if varied to better suit the circumstances, then varied in obedience to the "appropriate stimulation" which the circumstances furnish.

"There is no vestige of proof that instincts are increased by stimulation, *i.e.*, that they are not only called into activity by stimulation, but sharpened by it." On the contrary, there is abundant proof that the bird with practice makes a better nest; the spider with practice makes a more perfect web. But Mr. Reid says this improvement is no longer instinct; it is reason. Then, we ask, what becomes of the absolute distinction between instinct and reason? "Instinct does not merge into reason, but is sharply divided from it. Reflex action, instinct and reason are not derived the one from any other, but each one is distinct from, and has arisen independently of, the others—is not a more or less complex form of the others." "Instinct and reason do not merge at any point."

Mr. Reid quotes Spencer as regarding instinct as arising out of reflex action, and being evolved into intelligence, and Lewes as regarding instinct as the result of intelligence, and sets them off the one against the other, and disagrees with both. No doubt, as he puts the case, the two positions are irreconcilable; and if instinct arises out of reason it seems impossible that reason can arise out of instinct; but if we alter the wording very slightly it will appear not at all inconsistent to speak of reason arising out of instinct, and sinking into instinct again.

It appears evident that such a mass of inconsistencies and contradictions in the argument points directly to a fundamental fault in the definitions upon which the argument is based; and the test of their application to actual cases confirms the impression derived from the ambiguity of their form and the unsoundness of their basis—that they do not fulfil the requirements of scientific definitions, and do not define the subjects to which they are applied.

It has been pointed out that there is no common basis for comparison between a purely physical process such as reflex action, a quasi-psychological "faculty" such as instinct, and a psychological "faculty" such as reason. By reflex action is meant the physical reaction of a living body to physical stimulation. Reason is employed sometimes to connote purely mental processes, which can be observed by introspection of our own minds only; sometimes to imply the combination of this mental process with other mental processes and with conduct. It is in this latter sense that it is

understood when used in contradistinction to instinct, but the mischief is that, when so employed, the other more restricted meaning is continually creeping in and vitiating the argument. In whichever way it is employed, it can no more be compared with reflex action than a resolution passed at a public meeting can be compared with a sausage-machine. Before any mutually exclusive definition of any two or more things can be made, there must be some common standard of comparison to which they can all be reduced. In the present case, we can directly observe reflex action. Neither instinct nor reason can we directly observe. We cannot in any way compare reflex action with either instinct or reason, but we can very easily compare a reflex act with an instinctive act, and both with a reasoned act; and to these acts our definitions, like our comparisons, must be limited.

What, then, are the characteristics of a reflex act? If we touch the expanded tentacles of a sea-anemone they retract. They do not retract until touched, and when touched they retract. However long we watch the animal, we do not see its tentacles retract until they are touched, and when they are touched they never fail to retract. If we lightly touch the conjunctiva of a human being, the eyelid is convulsively closed. However long we watch the man, the lid is not so closed unless the conjunctiva is touched; and when the conjunctiva is lightly touched the lids never fail to close convulsively. If a foreign substance gets upon the sensitive surface of the air-passages, a cough is excited. The individual does not cough unless a foreign substance impinges upon the air-passages, and when such a substance touches the sensitive surface the cough never fails to occur. The two characters common to them are common to all reflex actions. Every reflex action is what its name implies—it is reflex. That is to say, it is the reaction of the living organism to a stimulus applied. No stimulus, no reflex action. And when the stimulus is applied it never fails to evoke that particular reaction and no other. When the tentacles of the sea-anemone are touched, they retract. They do not retract unless touched; and however often they may be touched they still retract and do nothing but retract. When the conjunctiva is touched, the lids close convulsively. However often it may be touched they still close convulsively. And the convulsive closure of the lids is the only action that is evoked by the stimulus. It is true that if a foreign body remains in contact with the conjunctiva the man may get up and go to a surgeon, but this act is manifestly no part of the reflex. So, too, however often the air-passages are stimulated by the contact of a foreign body, the cough never fails

to follow, and the action that follows is never anything but a cough. Allowance must be made for the differences in intensity of stimulus and for diffusion of stimulus. If the conjunctiva is wiped instead of being tickled, convulsive contraction of the lids does not follow; but then the stimulus is different, and the contention is that the same stimulus provokes always the same reaction. So, if the irritation of the air-passages is great and persistent, the cough will be reinforced by vomiting. But these examples evidently do not vitiate the rule. A moderate irritation of the air-passages will evoke a cough, always a cough, and nothing but a cough. A more violent irritation will evoke coughing and retching, always coughing and retching, and nothing but coughing and retching. Reflex action is, then, a reaction; it never occurs except in response to stimulus. It is unfailing; upon application of the stimulus it never fails to occur. It is unvarying; the same stimulus always evokes the same reaction. We may, therefore, sum up the nature of reflex action by defining it as an unfailing, unvarying reaction to stimulus; or, better, we may combine the two first qualities in a more comprehensive term, and define reflex action as *determinate reaction to stimulus*.

Such being the character of reflex action, how is instinctive action distinguishable from it? Like reflex action, instinctive action is determinate—that is to say, in given circumstances it is unfailing and unvarying. Under given circumstances of nutrition and warmth, the bee builds comb. Every working bee without fail takes its part in comb-building, and every bee builds comb in the same unvarying way. The garden spider never fails to make a web, and always makes a geometrical web. The trap-door spiders never fail to make their webs, and always make them to line their nests and to form a trap-door over the mouth of them. The house spiders never fail to make webs, and always make their webs in sheets and festoons. Every bird, when the nesting season comes round, makes its nest; and every species chooses a similar locality, similar materials, and a similar style of architecture. The rook always builds at the top of a tall tree, and makes its nest of live sticks. The thrush always builds in a bush, and makes its nest of grass lined with cow-dung. The kingfisher and the sandmartin always make a tunnel in the ground; the woodpecker a hole in a tree. When the season for migration comes round, the bird never fails to migrate, and migrates to the same country, the same locality, nests even in the same tree that it had sought before. When the spawning season comes round, the salmon unfailingly seeks the river. The newly-hatched turtle and alli-

gator unfailingly seek the water; the egg-laden turtle and alligator unfailingly seek the land. The beaver never fails to make its lodge and its dam, and makes them of the same materials and in the same way as other beavers do. The rabbit always makes a burrow, the hare always sleeps in the open country, and so with all other instinctive actions. When the circumstances occur, the acts occur, and occur with unfailing, unvarying regularity. So that instinctive actions, equally with reflex actions, are *determinate*.

But they differ from reflex actions in being spontaneous. They are not reactions to stimulus. They are the natural spontaneous activity of the organism seeking expression in definite, stereotyped directions. They need no stimulus to evoke them. They occur under certain circumstances, but the circumstances are not necessary to evoke the actions, and although certain circumstances may be necessary to render the action possible, as, for instance, a running stream for the dam of the beaver, yet in the absence of the circumstances an effort to perform the action is still made, as when the beaver, confined in a room, makes in the corner a caricature of a dam out of boots and hair brushes; as when the dog who never wants a meal, buries his bones for a future need which will never arise. So with all instinctive actions; when the time for their performance comes round, the animal spontaneously attempts to perform them. If the necessary stimulus is not applied, no reflex action will take place. If the stimulus is withheld during the whole life of the organism, still the reflex action will never occur. But the instinctive action needs no stimulus. The young turtle turns spontaneously to the water. The bee spontaneously seeks its way out of the hive, seeks the flowers, and returns laden with honey and pollen. The spider needs no external stimulus to induce him to make his web. The stimulus comes from within. The action is spontaneous. So when the nesting season arrives, the bird is impelled by irresistible urging from within to set about building; and later is impelled by a similar craving to start on its weary migration. The changing season supplies the occasion, but no stimulus from without is needed. The stimulus arises from within. So the spawn-laden salmon seeks the river, the egg-laden turtle and alligator the land, urged thereto by no stimulus from without, but by a craving which arises within.

Instinctive actions, then, while they resemble reflex actions in their determinate character, differ from them in being, not reactions to stimulus, but spontaneous actions. They are *determinate spontaneous actions*. The difference between reasoned actions and

the two classes of actions that we have already considered is manifest at a glance. Reasoned acts are *indeterminate* acts. They may occur in response to stimulus, or they may arise out of the spontaneously-generated activities of the organism, but in either case they are indeterminate. They are uncertain. They vary. They are unpredictable. To take Mr. Reid's illustration: In what respect, he asks, does the action of the spider in running to secure his prey differ from that of the man running to secure a snared bird? In the origin, he says, of the knowledge by virtue of which the adaptation of means to ends is made—inherited in the one case, acquired in the other. But, apart from the uncertainty as to whether the action of the spider is prompted by knowledge at all, there is another and much more relevant difference. The action of the spider is unfailing and unvarying, undivertible, predictable with certainty. The action of the man is indeterminate. When a fly touches the spider's web, and by the action of its wings communicates a vibration to the threads, the spider rushes out. When another fly does the same thing, the spider rushes out again. If we imitate the vibration of the fly's wing by touching the web with a vibrating tuning fork, again the spider rushes out. As often as the web is made to vibrate, so often is the spider "drawn." Repeated disappointment does not check his promptness of action. The presence of a formidable enemy does not scare him into quiescence. His reaction to stimulus is strictly determinate. But the reasoned action of the man has no such determinate quality. When he has snared a sufficient number of birds, he sets his snares no more. In presence of a formidable enemy, he does not rush out to capture his birds, but remains concealed. And, in any case, when a bird is in the snare his attention may be diverted, he may be called off his task by an urgent message; he may follow some bigger game within his reach; he may wait, so as to refrain from scaring birds from a neighbouring snare. His action is indeterminate. So when a spider has a dangerous captive, he cuts him loose. However often a too powerful fly engages his web, he deals with it instantly and in precisely the same way. His action is determinate. But the man does not necessarily build a golden bridge for his flying enemy. He may prefer to attack, on the chance of exterminating him; he may hang upon his rear and harass him. His action is not determinate. So with the action of the alligator that seeks the water and snaps at an opposing walking-stick, as contrasted with the man seeking refuge and striking at an intervening adversary. To accept Mr. Reid's explanation we must assume that the alligator

knows that he is going towards water, and knows that his action in snapping is minatory and deterrent, which, for the first occasion on which he does so, are large assumptions. The more essential distinction is that the alligator's action is determinate, the man's indeterminate. On every occasion on which the alligator is disturbed, he makes straight for the water. Even if he has previously experienced the futility of his effort at escape, the insecurity of his refuge, he still makes the same effort, seeks the same refuge. His action is certain and unvarying. But the man may seek refuge or may determine to fight; may seek one refuge rather than another; may have recourse to various stratagems; and if he has already found the refuge to be insecure he will not seek it again. The alligator attacks the foe who intervenes between him and the water. The man may attack or may avoid him. His action is variable. The same distinction may be traced between the instinctive or reflex action and the reasoned action in the other examples given in Mr. Reid's chapter.

If the distinction here drawn between reasoned acts on the one hand, and instinctive and reflex acts on the other, be the true distinction, then two consequences follow. First, that so long as any action falls completely within the definition of either, so long the distinction between them is absolute; a determinate act is instinctive or reflex, an indeterminate act is reasoned. Second, that any element of uncertainty or variability that is introduced into an instinctive or reflex act imparts into that act some element of reason; and, contrariwise, any element of certainty, or, let us say, *predictability*, that exists in a reasoned act renders that act in so far instinctive or reflex. Thus it will be seen that very few acts are purely instinctive or purely reflex, and very few acts are purely reasoned; but that the two kinds of action merge into each other by an infinite series of gradations. Nevertheless, although Mr. Reid is unquestionably wrong when he states that instinctive action is sharply divided from reasoning action, and that there is no border space where the one merges into the other, yet his statement contains an approximation to the truth; for, undoubtedly, the instinctive *factor* in any act is sharply divided from the reasoned *factor* in that act—as sharply as certainty is divided from uncertainty, and sameness from variability.

The action of breathing is adduced by Mr. Reid as an example of reflex action. According to our definition, it may more properly be regarded as instinctive, since the action is provoked, not by

stimulus from without, but by spontaneous impulse from within; but if we allow it to be a reflex action, it is still not completely determinate. It is certain that the rate of breathing will be within narrow limits, the same so long as the body is at rest in the same position; that it will be increased by exercise, greatly increased by violent exercise. But, to some extent, it is modifiable. We can at will hold our breath for a season, breathe slowly or rapidly, take deep or shallow breaths. In these cases the action of breathing, usually determinate, becomes to some extent indeterminate. An element of reason is imported into it. It becomes partly reasoned. It merges into reasoned action. Another example of reflex action is coughing, and of this the same is true. Though coughing is certainly excited by irritation of the air-passages, provided the irritation is sufficient, yet, if the irritation be only slight, the cough can be suppressed. If it cannot be wholly suppressed, it may be minimised; or it may be maximised—exaggerated. Lastly, a cough may be produced spontaneously, without the stimulus of irritation. In each of these cases the ordinarily purely reflex action may have an element of reason engrafted upon it—may become in part a reasoned action. In the last case it is purely reasoned. Yet in all cases the reflex element in the action can be sharply distinguished from the reasoned element, so that, although reflex acts may and do graduate and merge into reasoned actions, reflex action and reason remain distinct.

The action of the spider in making its web is commonly adduced as a type and example of instinctive action. Yet it is manifest that although there is a general and very striking sameness in the webs of any two spiders of the same species, no two webs, even of the same spider, are precisely alike. Each individual web has certain distinctive features, which adapt it to the particular position in which it is placed, and to the relative positions and distances of its supports. Though it is certain that an *epeira diadema* will build a geometrical web, composed, first, of main boundaries attached to supports, and enclosing an irregular polygonal space; second, of radii stretching from the centre of the polygonal space to its boundaries; and, third, of two ranges of spirals, one closely set round the centre of the space, and one more widely set external to the first; yet, when an *epeira* is placed in an enclosed space, it is quite uncertain to what points it will attach the main supports of its web, or what will be the shape of the polygonal area. Although the impulse, or, if we please, the craving, to construct the web is purely instinctive,

and although the attachment of the spiral threads is a combination of pure instinct and pure reflex action, yet the construction of the main supports is only partly instinctive and is partly reasoned. The general action of making supports, of making them stronger than the rest of the web, and of enclosing a polygonal area within them, are instinctive. They are certain and unvarying. But since no two webs are made in precisely the same position, the particular action of attaching these supports to particular objects is a reasoned action, and an action involving sometimes a high degree of reason. Moreover, whenever reason is exhibited in the construction of a web, it is exhibited in the attachment of the supports, and not in any other part of the process. Thus the means by which the supports are attached to distant points are various, uncertain, and ingenious. The spider may hang by a thread, and allow itself to be swung by the wind to the desired spot; or it may run round, having first attached one end of a thread to the point of departure, and pull it taut on arriving at the destination; or it may float a thread in the air and allow it to be carried by the wind. In windy weather, spiders will attach the lowest part of the web to a hanging weight, instead of to a fixed object, and thus preserve their web from destruction. A better example of a reasoned action it would be difficult to find; yet it is a part of the action, considered typically instinctive, of web-spinning. In this instance, therefore, instinctive action does merge into reasoned action, that portion of the action which is certain and unvarying being instinctive, and that portion which is uncertain and varying being reasoned.

Another action usually selected as the type and example of instinctive actions is the comb-building of the bee. The domestic bee never fails to make its comb, and the comb is invariably made of hexagonal cells with pyramidal bases, composed of three rhombic plates. In an apiary of a hundred hives, each containing tens of thousands of cells, every cell answers this description. In so far, the action of the bee in building its comb is purely instinctive and reflex. But this action is subject to much variety. It is varied abundantly and continually to meet special cases, and to overcome special difficulties. Where the drone comb, built of larger cells, meets the worker comb, built of smaller cells, intermediate cells are interposed to afford a transition from the one to the other. Where a corner has to be rounded, the cells on the convex side are made with mouths larger than their bases, and those on the concave side with bases larger than their mouths. When the comb becomes heavy with its contents, its attachments

are strengthened. If a comb is torn down by its own weight, temporary buttresses are constructed from comb to comb, to hold it up until it is permanently fixed by new comb. "In places where special conditions of the situation do not otherwise permit, it may be observed that the bees, far from clinging obstinately to their plan, very well understand how to accommodate themselves to circumstances, not only in cell-building, but also in making their combs." They will pull down and rebuild repeatedly the same cell in different ways, until they have it to their satisfaction. So that the typically instinctive acts of comb-building merge into and blend with highly-reasoned acts. And the instinctive element in the action is still the determinate element, and the action is reasoned in so far as it is indeterminate.

A third typically instinctive action is the nidification of birds. The impulse to build is purely instinctive. The choice of position, of materials, of pattern, of construction, are all instinctive. That is to say, they are determinate. They are in most respects certain and unvarying, and therefore almost purely instinctive. But in some respects they are uncertain and variable, and in so far as they are thus indeterminate they lose their instinctive character and become reasoned. Thus the thrush builds its nest in a bush, and builds it of grass lined with cow-dung ; in so far the action is determinate and is instinctive. But the particular bush, and the particular branch of the bush, are not determinate. These depend upon the choice of each individual bird, and of course varies with each bird, and with the same bird from year to year. That part of the action is not instinctive, it is reasoned. It is, as already said, proved beyond doubt that the first essay of the bird at nest-building is less successful, its first nest less perfectly constructed, than its subsequent nests. It learns and improves by experience and practice, and, so improving, its action varies, and thus becomes reasoned action. In this instance, again, instinctive action merges into reasoned action. An instinctive factor and a reasoned factor enter into the composition of the act, which thus becomes composite ; and, although the two factors can be distinguished from one another upon analysis, yet in the doing they are combined and merged the one into the other.

The real nature and relationships of reflex action, instinctive action, and reasoned action cannot, however, be thoroughly understood without a consideration of the nervous mechanism by which they are severally actuated. Reflex action being determinate, is actuated by nervous structure which is fully and completely organised ; so that the ingoing current provoked by the stimulus

is restricted to certain definite paths, acts upon and discharges certain determinate cells, whose discharge flows through definite and restricted paths to the muscles, and produces the definite, determinate action. The process is determined by the physical conditions under which it occurs. The stimulus can no more produce a different action than water can leap over a bank. The action can no more occur without the provocation of the stimulus than gunpowder can explode without the spark. If, however, the stimulus is unusually intense, the more powerful current that it produces will evoke a more powerful discharge, which, being of too great volume to wholly escape by its accustomed channels, forces its way into neighbouring areas, and produces additional and allied actions. Thus an intense irritation of the air-passages will produce, not only cough, but vomiting.

Similarly, instinctive action is actuated by a fixed arrangement of nerve tissue, an arrangement which is inherited just as the arrangement of muscles and bones in a limb is inherited. Just as a bird inherits an arrangement of muscles attached to the bones of its wing—an arrangement such that the action of the muscles on the bones will produce the movement of flying—so it inherits a certain arrangement of nerve tissue, such that the action of these cells and fibres will cause the alternate contractions of the muscles necessary to produce the movements of flying. And so, too, it inherits a certain structure of nerve tissue such that it serves as a storehouse of energy, and, when replete, discharges the energy into the nervous apparatus which actuates the movements of flying. Thus the action of flying is spontaneous, because the nerve tissue become replete with energy; and this energy finds its way, through to channels already formed, in certain definite directions, from which the movements of flying result. The arrangement and nature of the tissues which determine this result are inherited by the bird, just as the nature and arrangement of the rest of its tissues are inherited; just as the shape of its beak, the number and form of its claws, and the colour of its feathers are inherited. The important thing to observe is, that what is inherited is not a "faculty," nor knowledge, nor a way of thinking, but an arrangement of tissues and organs.

What is true of the instinctive action of flying is true of other instinctive actions. The impulse to perform the acts arises from the repletion of certain nerve tissue with the energy which it is its function to store. The form of the action depends upon the arrangement and connections of the nerve tissue into which the liberated energy is discharged. The reason why the

repletion of tissue impels to one instinct rather than another is because the replete tissue is in relation with one set of viscera and nervo-muscular apparatus and not with another.

We have seen that, although the distinctive quality of instinctive action, as compared with reasoned action, is its determinate character; yet no instinctive action is wholly determinate. There is, in every such action, some variable factor, some element of reasoning. Even in an action to all appearance so wholly instinctive as flying or walking the direction is variable, the time of starting and arrest, the direction and speed are variable. No action, however greatly instinctive, is wholly instinctive. So, too, the arrangement of nerve tissue which underlies instinctive actions, definitely and fixedly organised though it be for the most part, yet is always to some extent plastic, modifiable, subject to disturbance and rearrangement. It is inherited with its main features, indeed, organised, but it is never completely organised throughout, or instinctive action would be as unvarying as pure reflex action.

Lastly, together with the wholly fixed, definite, stereotyped, unchangeable, completely-organised nervous arrangements that actuate reflex action, and the, in great part, fixed, and nearly completely-organised nervous arrangements that actuate instinctive action, there is inherited a quantity of nervous tissue which is still in plastic condition, still modifiable, and subject to rearrangement under the action of incident forces and of its own escaping energy. It is this portion of nervous tissue that actuates reasoned actions—actions whose indeterminate character represents the as yet incomplete organisation of the nervous arrangements to whose activity they are due. This portion of nervous tissue is, of course, inherited just as much as are the portions which actuate instinctive reflex action. The distinction is that in the last case the arrangements are inherited ready made, completely fixed; in the second they are in great part ready made, but in part subject to modification by the experience of the individual, and in the first case they are only "roughed out," and although the individual derives much assistance, and is saved much labour by the inherited tendency of this tissue to develop and organise in certain definite ways, yet, in the absence of the guidance of experience, that is, of stimulation by circumstances, it will only slowly, slightly and imperfectly so develop.

By regarding the inherited factor in conduct, not as faculty, but as arrangement of nervous tissue, we have a trustworthy guide through the perplexing tangle of the genetic relationships of the three sets of actions with which we are dealing.

When it is stated that instinct and reason do not merge at any point, as they would were the one derived from the other, there is a confusion of thought involved in the statement, which becomes obvious when we deal, not with instinct and reason as faculties, but with instinctive action and reasoned action. Obviously an act cannot be, in the same respects, both determinate and indeterminate, and if we call the determinate portions of the act instinct, and the indeterminate portions reason, then, of course, instinct and reason do not and cannot merge into one another. But if we regard the action as a whole, and find that certain portions of it are determinate and certain other portions are indeterminate, that indeterminate action may become determinate and *vice versâ*, as in the cases examined above of the web-making of the spider, the comb-building of the bee, and the nest-building of birds; then, undoubtedly, an action may partake of the nature of both instinct and reason, and in this sense the two do most undoubtedly merge into each other; so that this argument against the origin of one from the other falls to the ground.

But, says Mr. Reid, I define instinct as depending on inheritance, reason as wholly acquired, and, since acquired traits are never inherited, instinct cannot be derived from reason; nor, since reason contains no element of inheritance save the power to be acquired, can reason be derived from instinct. If reason and instinct be regarded as faculties, and if acquired traits are never inherited, and if Mr. Reid's definitions are correct, this reasoning is no doubt sound. But if we deal with instinct and reason in terms of the acts which display them, and if we regard these acts as depending on nervous structure, we shall see reason to doubt his conclusions. Pure reflex action is actuated by a nervo-muscular mechanism, which is completely organised, that is to say, which has its parts disposed in an arrangement so fixed and determined, so shut off from the possibility of interference from without, that the action follows the stimulus as surely as the discharge of the gun follows the pull of the trigger. But it is quite in accordance with our knowledge of the arrangement of the nervous system to suppose cases in which, although the mechanism is completely organised throughout most of its extent, yet there is in some part of the circuit a breach through which modifying currents can find admission; and, correspondingly, we find that there are reflex actions, which are not purely reflex, but admit of modification and inhibition. Thus, although we cannot check or interfere with the convulsive closure of the eyes that follows a

light touch on the conjunctiva, we can modify and check the cough that follows a slight irritation of the air passages. Once allow that reflex actions can be modified—that the nervo-muscular circuit, well and deeply organised, as for the most part it is, yet in part of its course is incompletely organised, and retains some plasticity—and it is immediately apparent that the degree to which the fixity of organisation may be imperfect, and plasticity of action may be retained, is indefinite. It may be, in some cases, little or none, in others much; and hence we shall find actions, such as that of the eyelids, or the contraction of the pupils on the stimulus of light, that are purely reflex; others, like that of coughing, that are slightly modifiable; others, like that of withdrawing an injured limb, that are more modifiable, until we arrive at actions like that of undressing, or winding the watch, or crocheting when the materials are placed in the hands, or writing when a pen is put between the fingers, whose reflex character is so little apparent, is so swamped by the intelligence with which they are usually guided, that it only becomes apparent in certain morbid states of the nervous system, when the normal interference of parts of the nervous system outside of the special nervo-muscular circuit is abolished by disease.

Purely instinctive action is similarly actuated by a fixed, definite, organised arrangement of nerve tissue. But, as in the case of reflex action, the whole complexus of this arrangement is seldom organised with such completeness as to prevent the incursion, at some point, of interfering currents from without its own area. Whenever such incompleteness of organisation exists, the actions admit of modification. They become indeterminate. They partake of the nature of reason. They exhibit intelligence. Now, it is certain that incompletely organised arrangements of tissue are as freely transmitted from parent to offspring as completely organised arrangements. For instance, the majority of bones are more completely organised cartilage, yet not only bones but cartilages are inherited. Many inherited tissues are partly bone and partly cartilage, and some such tissues become in the course of life completely organised into bone. Hence there is nothing unusual, or incongruous with other instances of inheritance, in the transmission from parent to offspring of either the completely organised arrangement of tissue that actuates purely reflex action, or the well-organised, but in part incompletely organised, arrangements that actuate impure reflex action and instinct; and the point is, that not only is that part of the arrangement inherited which actuates the instinctive portion of

the action; but that part also of the arrangement which actuates the reasoned portion of the action is likewise inherited. This latter part of the nervous arrangement is, it is true, plastic, modifiable, educable. It is inherited, not as a completely organised arrangement, but as an arrangement incipiently organised, and thus in some sense justifies Mr. Reid's description of reason that what is inherited is not reason itself, but only the power of acquiring reason. It is obvious, however, that to regard what is transmitted as nervous tissue rather than as "power of acquiring reason" is a great advance in the definiteness and clearness of knowledge.

As with reflex acts, so with instinctive acts, the proportion of unorganised or incompletely organised tissue that enters into the construction of the nervous arrangements that actuate them varies greatly in different cases, and, correspondingly, the amount of the reasoned element that is combined with the instinctive action varies, some actions being almost purely instinctive, others being almost purely reasoned, and having their motive only in instinct.

This being so, the final question, whether instinct can be evolved into reason, or whether reason can sink into instinct must be stated anew in the terms, not of faculty, but of action. It becomes the question whether reflex or instinctive action admits of modification so as to become reasoned, and whether reasoned action can become stereotyped so as to become instinctive or reflex. That the first of these questions can be answered in the affirmative we have already seen. In every instinctive action, and in nearly all reflex actions there is an element of reason. The question becomes whether this element can increase and take a larger and larger share in the action. Without adducing examples, it will be evident to those who are acquainted with the facts of animal psychology, and even from the facts already adduced, that this does occur, and that instinctive actions may and do become modified into reasoned actions.

As to the reverse change, the attainment of fixity and invariability by reasoned actions, we have the universal experience of mankind in its favour. Of the nature and mode of working of nervous processes we know little, but this fact is established beyond doubt, that nervous processes are established and organised by repetition. When a novel process occurs in the plastic, and comparatively unorganised nervous tissue, it brings about a partial and tentative re-arrangement of the tissue elements. If the process is not repeated, the new arrangement becomes partially or completely dissipated, and a mere trace, or no trace, is left.

But if the process be repeated, the disturbed nervous elements are thereby confirmed in their new disposition; and with every repetition they become more firmly fixed, more stably compacted, more completely organised. So that it is a matter of common, of universal experience, that actions become easier by being practised. At first purely reasoned, they gradually lose their reasoned character and become more and more determinate. Although, in the lifetime of an individual, reasoned acts rarely become purely reflex, yet they often become so largely determinate that the application to them of the term "reasoned" becomes inappropriate, and they are called, in the less advanced stage "habitual," and in the more advanced stage "automatic." The response to command may become so fixed in the nervous organisation of the old soldier, that if he is suddenly called to "attention" when carrying his dinner, he will jerk his arms to his sides and let his meat and potatoes go rolling in the street. In this case, the transformation of a reasoned action into a reflex has become complete in the lifetime of a single individual. To examine the question whether the reflex thus acquired may be wholly or in part transmitted to the offspring is beside the question.

Although, therefore, we cannot refute the position that "reason and instinct do not merge into one another," since the instant an act becomes instinctive it ceases to be reasoned, and the instant it becomes reasoned it ceases to be instinctive; yet, if we deal with acts, we find that, beyond the shadow of a doubt, instinctive and reflex actions do merge into reasoned actions, and reasoned actions do merge into instinctive and into reflex actions.

CHARLES MERCIER.

SOME WORKS ON PSYCHOLOGY.

(1) *Mental Physiology, especially in its relation to Mental Disorders.* By T. B. Hyslop, M.D., Lecturer on Mental Diseases to St. Mary's Hospital Medical School, &c. London : Churchill, 1895.

(2) *Outlines of Psychology.* By Oswald Külpe, Professor of Philosophy in the University of Würzburg. Translated by E. B. Titchener, Sage Professor of Psychology in the Cornell University. London: Sonnenschein, 1895.

(3) *Analytic Psychology.* By G. F. Stout, Fellow of St. John's College, Cambridge, &c. 2 vols. London: Sonnenschein, 1896.

(4) *An Outline of Psychology.* By E. B. Titchener, Sage Professor of Psychology in the Cornell University. New York and London: Macmillan, 1896.

The English student of psychology is to be congratulated just now on the number and variety of the text-books of the science which are appearing in his language. Some of us can remember the time when the reader was confined to the works of two writers, Herbert Spencer and Alexander Bain. Now he has a choice of English and American productions, as well as translations of German works. The publication of so many books might seem to be regrettable on the ground that the student will not know what to read. But in truth it is to be welcomed, on the one hand, as showing the greater quantity of activity now thrown into psychology, and, on the other hand, as securing the student against a one-sided and individual conception of the subject. Psychology, which deals with a multitude of diverse human minds as its subject-matter, can only be developed by the co-operation of diverse minds.

The books whose titles appear at the head of this notice have somewhat dissimilar aims, and markedly unlike characters. A word or two on these differences will help to illustrate the many-sidedness of contemporary psychological work.

Dr. Hyslop's manual of "Mental Physiology" follows, in the main, the lines of the well-known works of Dr. Maudsley and others. It is, as its title indicates, especially written for medical students. At the outset it gives a fairly full and clear account of what is known at present respecting the structure and functions of the human brain. The results of recent research into the localisation of what Dr. Hyslop not too happily calls "mental faculties" are adequately brought out. After this follow chapters on "Mind," "Sensation," "Perception," "Attention," "Memory," "Feeling," and so forth. The deviations from the normal type of mental process are dealt with in close connection with this last; and this is in many ways an advantage to the student.

The author has evidently read widely, and makes on the whole a judicious use of his material. Perhaps the most noticeable defect in the volume is the scanty reference made to the new and large field of experimental psychology. Hence, it will probably be found necessary to supplement the reading of this manual by that of Professor Ziehen "Introduction to Physiological Psychology," or some similar work.

Professor Külpe's work is an important contribution to psychology. A pupil of Wundt, he not unnaturally follows, in the main, the lines of his teacher's great work. But it is much more than an epitome of the Wundtian psychology. Külpe thinks out his subject with a good deal of fresh vigour. As a text-book it will be of real service to those who desire to be abreast of the latest results of experimental work. In this respect it is decidedly in advance of Professor Ladd's well-known works. Külpe, judiciously perhaps, omits all account of the nervous system. He everywhere assumes a knowledge of the physiological processes involved, as may be seen by a glance at his chapters on sensation. The various lines of experimental work developed at Leipzig and elsewhere are carefully summarised, and their value critically estimated. It is here that the writer is at his best. Külpe's book will long remain a valuable work of reference.

In the new treatise by Mr. Stout, the editor of *Mind*, and now Lecturer on Comparative Psychology in the University of Aberdeen, we have an examination of mental processes, written with as little reference as possible to nervous processes. It is pre-eminently a work of introspective analysis, aiming at unravelling more carefully and completely the complex processes of mind, such as perception, belief, and so forth. Here lies its value. Mr. Stout has a particularly acute eye for the subtle

complexities of our mental life, and he has, in some important respects, carried the work of analytical psychology to a further point than had been reached by his predecessors. The writer's originality and force are illustrated in dealing, among other subjects, with that aspect of perception which he calls "the apprehension of form," *e.g.*, the rhythmic form of a series of sounds (a subject at which the Germans have of late been vigorously working), the "process of attention," and "apperception" and "apperceptive systems." In discussing this last he seeks to show that the laws of association, as ordinarily stated, do not fully explain the way in which our ideas are grouped into organic wholes. While, however, Mr. Stout keeps for the most part to the introspective point of view, he by no means despises the standpoint of physiological psychology. On the contrary, he emphasises the fact that what are called by other psychologists "psychical dispositions" are in reality psycho-physical. His use of this conception of a psycho-physical disposition is most suggestive. Other fresh and suggestive discussions concern the connection of thought and language, where he shows a careful study of the earlier forms of gesture-language, and belief and imagination. Enough has been said, perhaps, to show that Mr. Stout's work is not one to be neglected by the physiological psychologist, as written from the old "metaphysical" point of view. It is eminently scientific, both in its aim and in its method.

Professor Titchener's little manual is carefully planned and well written. He also, like Külpe, is a pupil of Wundt, and follows to some extent his teacher's mode of treatment. Yet he keeps throughout in touch with the traditional English psychology. This enables him to write clearly, and makes his manual better fitted for the average English and American student than Külpe's more elaborate text-book. Titchener, as he tells us in his preface, makes the exposition of the methods and results of experimental psychology his main object. This feature gives to the book its chief value. It may be noted that Titchener follows Külpe in his tendency to assimilate sensations and images. That is surely far enough away from the tradition of English psychology since Hume, and seems of very doubtful value. It may be added that Külpe and Titchener further agree in not recognising any simple third "quality" of mind, after sensation and feeling, answering to conation or volition. Their treatment of this subject may well be contrasted with that of Stout (book i., chap. vi., and following chapters).

James Sully.

Des Dégénérescences secondaires du Système Nerveux. Dégénérescence Wallérienne et rétrograde. By Dr. GUSTAVE DURANTE. Paris, 1895.

THE scope of this work is sufficiently indicated in the title. It consists of an extensive critical summary of the chief recorded facts bearing upon secondary degeneration, and more especially upon the so-called retrograde degeneration, the importance of which has been accentuated by the development of the "neuron theory" of Waldeyer, based upon the important histological observations of Golgi.

After a preliminary discussion of the Wallerian degeneration, illustrated by the characteristic changes following lesions of the efferent and afferent tracts in the spinal cord and the peripheral nerves, the remainder of the book is given over to examples and criticisms of retrograde alterations following various lesions. As it is unnecessary to follow the author through the numerous instances which he has given from among recorded cases and several original observations, a summary of the conclusions at which he has arrived may be sufficient.

This retrograde change, which is characterised chiefly by an attenuation of the myeline sheath of the nerve fibre with preservation of the axis-cylinder, follows not only section of the nerve, but also injuries involving the peripheral terminations of the nerve fibres. Depending upon the rapidity and intensity of the process, its appearance varies slightly. Sometimes the myeline is broken up into fine bullæ; at other times absorption takes place, so as to exhibit a progressive atrophy; changes which are clearly shown by Marchi's method. It does not invade the whole neuron simultaneously, but follows an ascending course towards the trophic centre of the nerve, which it generally, but not necessarily, implicates. The affection of these centres is not always histologically apparent, even when the degeneration passes from one neuron to another. The chief change observed here is a simple atrophy of the nerve cells without any structural alteration. For example, several instances are given of degeneration of the posterior columns of the spinal cord following cerebral lesions. The appearance presented by the posterior columns in these cases varies somewhat from that due to Wallerian degeneration in the same structures; in the retrograde form

the degeneration is much less compact. Of similar nature to the above are the alterations observed in the spinal cord after amputation of the limbs, and numerous other examples might be cited by way of illustration.

The pathogeny of this degeneration is still obscure. Although the nerve fibre remains attached to its trophic cell, yet it is deprived of the normal impulses from adjoining structures through its peripheral terminations. The recognition of this retrograde change permits the study of a number of degenerations which were at one time believed to be of a primary nature. It shows, in fact, that the determining cause may not only be an affection of the nucleus of origin of a nerve fibre, but also of the peripheral ramification. Its acceptance indeed establishes the possibility of degeneration propagated from neuron to neuron, although a trophic centre is interposed. Such may be several forms of muscular atrophy previously regarded as of "reflex" origin; and to such also may be due the incurable nature of some peripheral paralyses, and mention has been already made of the changes in the spinal cord following amputation. It may also explain the mode of development of some forms of combined sclerosis, as well as a certain number of cases of tabes dorsalis.

There is an extensive bibliography at the end giving references to the whole subject of secondary degeneration.

W. A. Turner.

Neurologische Beiträge. Von P. J. Möbius. Heft I., "Über den Begriff der Hysterie und andere Vorwürfe vorwiegend psychologischer Art," pp. 210. Heft II., "Über Akinesia Algera. Zur Lehre von der Nervosität. Über Seelenstörungen bei Chorea." *Verlag von Ambr. Abel* (*Arthur Meiner*) in Leipzig. 1894.

The author has, in these two volumes, reprinted a number of his writings on neurological subjects; many of these papers are of much value, and are now accessible to the reader in a convenient form.

The first three papers, which deal with hysteria, have been already shortly considered in Brain, 1894, p. 142. One of them

treats of astasia-abasia. In addition the author makes some further contributions to the theory upheld by him of the psychical origin of hysteria, quoting recent observations, chiefly from the Salpêtrière School, in support of this view. With regard to hysterical anæsthesia, he agrees with Onanoff that it *may* come through suggestion; but thinks that it more commonly originates as a direct consequence of morbid brain action. The stigmata and the symptoms of the convulsions bear a similar relation to the emotions in the hysterical that their physical concomitants bear to emotions in the healthy. Just as a healthy person may turn pale and sweat from a fright, so the hysterical may become anæsthetic, or pass into a fit. Hysterical symptoms thus fall into two groups: those which are called out through some emotion, but which had a previous existence; and those directly connected with a suggestion, and which correspond to it in the form they take. One source of confusion in the literature of hysteria has been the tendency of authors to exaggerate one of these sets of phenomena at the expense of the other.

The author is in agreement with Prof. Janet's views, founded on experiment, that the most striking hysterical symptoms, especially anæsthesia, are of psychical origin, and to be assigned to a division of consciousness. He has himself expressed the same view in the aphorism that the anæsthetic hysterical patient feels but does not know it. Janet regards narrowing or restriction of the field of consciousness as the primary change, and the division of consciousness (double consciousness) as secondary to it. Möbius would regard the latter as the primary phenomenon, and points out that such a division can only be regarded as hysterical if that part which fails the waking consciousness is not entirely lost to it, but can be recovered under suitable conditions.

His own view is that the essential change in hysteria consists in that, temporarily or permanently, the psychical condition of the hysterical is similar to that of the hypnotised person. Hysteria is not, however, simply identical with increased amenability to suggestion, but is a *morbid* suggestibility. Suggestion is possible in healthy persons to a varying degree, but the resulting action corresponds to the idea suggested, whereas in the hysterical the reaction is a morbid one; painful or terrifying emotions are called out and give rise to morbid symptoms. For instance, unless hemi-anæsthesia is directly suggested, no healthy person will develop it, whereas it may occur in the hysterical, without their being in the least aware of it, as the result of a fright. Briefly, all hysterical symptoms are in form

suggestions, but a portion of them are only indirectly due to suggestion, being a morbid reaction to emotion.

The papers immediately succeeding are occupied by an argument with Wagner as to the origin of the psychical disturbances that occur in persons revived after attempted suicide by hanging. In such cases, convulsive attacks occur, and are followed by a period of mental confusion. Amnesia is present for all events from a period a little before, to some time after, the strangulation. Wagner argues that these symptoms are to be attributed to mechanical causes, *i.e.*, sudden anæmia and subsequent great flushing of the brain with blood, due, respectively, to occlusion of the carotid and relief of the pressure. Möbius would explain them as hysterical; and in this he relies chiefly on the close resemblance clinically of the symptoms to those met with in certain phases of hysteria. This applies especially to the peculiar retro-active amnesia which occurs in precisely the same form in traumatic hysteria. This explanation Möbius does not apply to all cases, but only to those in which no structural damage to the brain has occurred. The question is dealt with at considerable length, Wagner relying on the results of experiments in animals, and on the rather meagre facts of morbid anatomy in persons that have subsequently succumbed, and Möbius on clinical observations, chiefly recent researches and methods of psychological experiment in hysteria.

Other papers in this volume deal with the value of electricity in the treatment of paralysis, and with the dangers and uses of hypnotism. In the last paper, which deals with the principles of psychology, Dr. Möbius has adopted the admirable plan of defining the psychological terms used by him, and of stating the sense in which they are used. He says that he has taken the opportunity of stating over again the opinions which form the groundwork of all his statements and the starting-point for his consideration of hysterical symptoms.

The first four papers in the second volume deal with the neurosis originally described by the author under the title of *Akinesia Algera*. The first is the original communication, of which an abstract, giving a more or less full account of the disease, will be found in BRAIN, 1894, pp. 173, 174. In the others Dr. Möbius gives the sequels of the cases previously published, with an autobiographical account of Fechner's illness, who suffered from a similar affection, also short abstracts of cases reported by other observers, which in general support his own statements, and then proceeds to a

further description of the neurosis. The patients are "degenerates," partly from hereditary, partly from acquired, conditions. He notes the frequency with which other members of the family suffer from various nerve disorders, especially milder forms of the psychoses. The exciting cause is overstrain—mental, emotional, or intellectual, the latter less commonly. The cardinal symptoms of *Akinesia Algera* are (1) the pains and other abnormal sensations which result from the performance of any action or work and (2) the consequent restriction of functional capacity. These symptoms tend to be complicated in some patients by those of neurasthenia, and in others by hysterical manifestations, and from both of these they have to be distinguished. That the pains are of psychical origin is inferred from the absence of organic lesions, and from the possibility of modifying them through psychical influences. Other symptoms besides the subjective ones and the incapacity for volitional actions are increase of the tendon reflexes, localised œdemas, and wasting of muscles. Moreover, the patients are not amenable to suggestion in any form, and, unlike normal persons, cannot be influenced by feelings of hope or fear. Affections of general sensation and of the special senses are to be regarded as secondary symptoms (hysterical). All activities, such as moving, reading, writing, seeing, hearing, and mental efforts, give rise to painful sensations in the head. But these pains, which resemble those of neurasthenia, are distinguised from the latter by their close relation to functional activity of some kind. Persistent (constant) pains in the head, apart from such activity, are at least rarely met with. One typical feature of the disease is that movements of the head itself cause no pain. Patients who are otherwise unable to make any movement can raise and move the head without trouble; similarly, movements of the oculo-motor and facial muscles are not interfered with, and the patients speak without effort or ill result. Insomnia is, as a rule, a marked feature. The sufferers thus pass into a state of absolute incapacity. (See Brain, *loc. cit.*)

According to the author, the prognosis is extremely bad. In only one case, that of Fechner, which differed in some respects from the ordinary type, was there a relative cure. In time a tendency to psychical disturbance, ending in insanity, shows itself. Of his own cases, one died after seven years' illness; one died insane after three years' illness; one committed suicide; and one died of tuberculosis after fifteen years' illness. Other patients observed are steadily getting worse, after illnesses of varying

length. So far no form of treatment has been found effectual. Active treatment of any kind aggravates the disease. In time he thinks it quite possible that milder forms of the neurosis may come under observation. For the present, the author thinks it advisable to class, at any rate provisionally, *Akinesia Algera* as a distinct disease. It is most nearly allied to hysteria, and might, perhaps, be regarded as a special form of hysteria; but, on the other hand, the customary features of hysteria are absent, and the peculiar course and grouping of the symptoms make Akinesia Algera a sufficiently well defined clinical entity.

The next section of this volume consists of several papers on neurasthenia, and contains a full and most useful bibliography of the numerous writings on this disease. The author gives an interesting historical sketch of the work done on this subject. That until recently so little advance was made in our knowledge of the neuroses he attributes to the concentration of attention on the analysis of symptoms and determining of functions of organs in the light of researches into pathological anatomy and physiology, to the neglect of psychology and also of sociology, the latter arising from the limiting of medical study to the sick; whereas, if nervous diseases are to be properly understood, man must be studied in his social relations, and under normal conditions of activity. The value of a name is strikingly exemplified in the case of neurasthenia. If Beard had written on "nervousness," his observations might, like those of his predecessors on the same subject, have fallen unheeded. No doubt the increase in nervous complaints, due to the complex conditions of modern civilised life, has also been a most important factor in directing attention to them.

Dr. Möbius emphasises the need of a more exact definition of neurasthenia. He believes that clearness is gained by starting from the conception of fatigue. The symptoms of neurasthenia are those of fatigue; and the neurosis consists in an increased susceptibility to fatigue from exertion; the greater the innate tendency the slighter the exertion necessary to give rise to the symptoms. It may thus be defined as chronic fatigue, and should be spoken of, not as a disease, but as a group of symptoms.

Neurasthenia is not to be regarded as synonymous with nervousness. A man may throughout life be nervous, and have a tendency to hypochondriacal ideas, and yet show no sign of neurasthenia, *i.e.*, of abnormal fatigue from mental or bodily exertion. A sharp distinction is to be made between hysteria and neurasthenia. In hysteria the symptoms are dependent

upon idea; the essence of hysterical conditions being that ideas too easily excite abnormal changes in the organism. Hysterical states are most strikingly distinguished from neurasthenic ones by the fact that they may disappear quite suddenly, even when they have been present for a long time.

The author points out that work which involves much strain on the attention, either from its novelty or difficulty, is the kind of work which is most apt to lead to neurasthenia. In this essay there is an excellent section on treatment. The author thinks that residence at health resorts has been overdone; he divides these patients into three groups from this point of view: (1) The smallest number, who are cured and greatly improved by residence at a health resort; (2) the largest—comprising two sub-groups: the (*a*) "degenerate," and (*b*) those in whose case the exciting cause is not removed—in whom there is temporary cure, followed by a relapse; and (3) not a small group, those who return worse than they went. Therapeutic measures should be directed both to mind and to body. For the former, suggestion may be useful in a very limited number of cases; but the chief measure is the systematic guidance to a well-regulated activity, designed to call into action the organs unaffected; counsel and judicious advice as to the avoidance of injurious influences. For the body, the best methods are those which entail exercise and strengthening of the muscular system. That is the most suitable work which employs mind and body, is suited to the individual capacity, and, in the right way, alternates with sufficient rest. If possible the work enjoined should have a tangible result, otherwise it is apt to be unsatisfactory. The kind of treatment thus generally indicated is more likely to give good results than that by climate, baths, or gymnastics, which do good at first, but, as the patient becomes accustomed to them, fail of their effect.

There is also an interesting paper on the influence of heredity in nervous diseases. Several instructive family histories are recorded, especially with regard to the occurrence of neuroses and milder forms of nervous disease in the same family. Such histories show that hereditary factors play a larger part than is generally known; the more carefully such genealogies are investigated the more weight is to be attached to the milder forms of disease, and the more clearly it is seen that even the members of such families that pass as normal individuals, present evidence of hereditary taint. As regards particular points, he thinks that the moral degeneration accompanying good

intelligence, which is described by some authors in those hereditarily nervous, is rarely to be met with.

The concluding essays deal with the psychical disturbances present in some cases of chorea.

It will thus be seen that these essays cover a wide range of subjects; and the marked ability and originality, which throughout mark their treatment, render them worthy of careful study.

J. Michell Clarke.

Manuel Pratique des méthodes d'Enseignement spéciales aux Enfants Anormaux, par les Docteurs Hamon du Fougeray et L. Couëtoux, avec une Préface du Dr. Bourneville. Paris, 1896.

The book, as Dr. Bourneville says in his preface, is addressed to all physicians, educationalists, professors, and instructors who are interested in teaching. Considerable efforts have been made by the French Government in educating normal children; but, unfortunately, the same enthusiasm has not been displayed in instructing all classes of abnormal children. This book shows that all categories of the latter class, viz., the blind, stammerers, deaf and dumb, and idiot and backward children are capable of being educated. A fair amount of good work has been done with respect to the blind, the stammerers, and the deaf and dumb; but people generally are not so convinced of the necessity of teaching idiot and backward children. Dr. Bourneville is of opinion that the special establishments for these abnormal children should receive them with less restrictions than at present, that the education should be begun as soon as possible, that as instruction is slow, parents should have as much patience as possible, that the task of education should be confided to competent persons, and that the instruction should be collective in character. When the children arrive at adult age, those who have least inproved should remain in establishments for adults, in which they can work at trades, and so diminish the cost of their maintenance; while those who have made good progress, as soon as they are discharged, should be aided by philanthropic societies, so that places may be obtained for them, in order to prevent them

from falling into habits of mendicity. It is only since the commencement of the nineteenth century, the authors say, that the question of educating the classes of children here referred to has undergone (thanks to scientific progress) its full development. This good work is, however, still unknown in great part, not only to the general, but the medical public; and the object of this book is to enlighten them on what has been done, to elicit their interest, and to gain their support. Several special treatises have been published with respect to one or other of the classes of abnormal children, but no book up to now has dealt with all classes. The work is divided into five parts: the first four deal with the study of each infirmity in particular; the fifth gives the results of teaching combinations of each defect in the same individual. Each infirmity is studied first from the physiological point of view; and the authors have endeavoured to show this as clearly as possible, and to demonstrate the physical, moral, and intellectual consequences. Then the history of each defect is described, and the methods which are in operation for alleviating it are pointed out. Chapters have been added giving statistics, lists of institutions in which special instruction is imparted, and the formalities required for admission. Lastly, the authors give a condensed account of the legislation which is applied in France to these diverse maladies. In describing the deaf and dumb, some chapters have been added on deafness, and on oral teaching, in individuals who have become deaf, but are not dumb. In the chapter on the treatment of stammering, the method employed in cases of perforation of the palate is related. The chapter on idiocy is chiefly a reproduction of a report by Dr. Bourneville to the Congress at Lyons, in 1894, on the assistance, treatment, and education of idiot children. The fifth chapter treats of the following combinations in the same individual, viz., deaf dumbness and blindness; deaf dumbness and idiocy; blindness and idiocy; deaf dumbness, blindness, and idiocy. Allusion to the well-known case of Laura Bridgman, and the less known case of Helen Keller, is made. The illustrations add considerably to the interest of a book, which will be of use to all who are concerned with the education of these abnormal children.

FLETCHER BEACH, M.B.

Theory of the Functions in Living Matter.—By Professor E. Hering, Prague, 1888.[1]

Metabolism is, physiologically speaking, the essential distinction between living and dead matter. It signifies the chemical processes in living substance, by which, on the one hand, certain products are excreted as foreign bodies, and either accumulate *in situ*, or pass out into the circulating fluids; while, on the other, there is a simultaneous intake of nutritive matters to form new constituents. This last function is known as assimilation; the first may be termed dissimilation.

In distinguishing these functions, we must not fall into the error of regarding them as two intrinsically separate, parallel processes, and the living matter itself as a quiescent mass, used up on one side, and replaced on the other—as a copper wire dipping into copper sulphate, and traversed by an electrical current, loses copper on the one hand by decomposition, while on the other it takes up new copper. Assimilation and dissimilation must rather be conceived as two closely interwoven processes, which constitute the metabolism (unknown to us in its intrinsic nature) of the living substance, and are active in its smallest particles,—since living matter is neither permanent nor quiescent, but is in more or less constant internal motion.

To assimilate and dissimilate is a fundamental property of living matter, engrained deeply in its nature, and these functions continue—provided the essential conditions of life are present—without assistance from external stimuli; we are thus free to regard what are here termed vital conditions as being in part "internal" stimuli.

In so far as living matter is wholly unaffected by adventitious external stimuli, its assimilation (A), and dissimilation (D), may be denoted *autonomous*.

[1 Though published long ago, this very important article does not seem to have received from students of physiology the amount of attention it deserves. It has been thought desirable to make its substance known to English readers through the medium of BRAIN, but the impossibility of reducing it to an intelligible abstract has made necessary its full reproduction as a translation. —ED.]

So long as the autonomous D and A are equal, the state of the living matter cannot alter; qualitatively and quantitatively it remains the same. Such a state of perfect equilibrium between the autonomous D and A may be designated *autonomous equilibrium*.

This state of living matter is altered when any stimulus excites it to active dissimilation, no longer balanced by equal assimilation. Such dissimilation is no longer exclusively autonomous; it is reinforced by external factors, and must be denoted *allonomous*, in contradistinction from the pure autonomous process. The increased formation of D-products, and corresponding loss of elements that were formerly an integral part of the living matter, and included in its chemical structure, produces internal alteration in the substance, in proportion with the intensity and duration of the stimulus. Hence at the close of excitation the living matter is quantitatively and qualitatively (*infra*) altered.

If the process of dissimilation is regarded as a function of living matter, the latter must at this stage be designated less capable of functioning. Since the substance is altered, not merely qualitatively, but quantitatively also, the present as compared with the former state may be termed "below par" (*unterwerthig*). From this condition it tends to return to the earlier "at par" (*mittelwerthig*) of autonomous equilibrium. In proportion *i.e.* as living matter is excited by external stimuli to more active dissimilation, its disposition to dissimilation falls, while its disposition to assimilation rises.

Other conditions of A and D being constant, the autonomous D is weaker, the autonomous A greater, than before excitation. And living matter tends to return to its former state the more energetically, in proportion with the deficit from the preceding stimulus, *i.e.*, the amount to which it remains below par at the close of excitation. At the same time the greater disposition to A, and corresponding increase of assimilation, immediately after excitation, diminish with the neutralisation of the changes produced by excitation, and thus the expenditure of substance is checked again. This augments the lesser D-disposition, and correspondingly feeble dissimilation, until finally the A- and D-dispositions, as well as the autonomous assimilation and dissimilation, are once more equal, and the earlier "mean state" (*mittelwerthig*) and autonomous equilibrium between D and A are recovered.

Whereas this last state is characterised by equality of A- and D-dispositions in the substance, the state of deficit entails excess of A- over D-disposition.

Obviously, therefore, the depreciation of living matter develops and proceeds *pari passu* with the impact and duration of a *D*-stimulus. The *D*-disposition falls in the same ratio, since substance below par exhibits less disposition to *D*, along with a simultaneous rise of *A*-disposition. This implies a corresponding loss of excitability towards the constant *D*-stimulus.

The excitability of living matter to *D*-stimulation, briefly *D-excitability*, thus declines in ratio with the effect of the *D*-stimulus, while the activity of the dissimilation itself is correspondingly diminished. But we have seen that the *A*-disposition (along with the simultaneous autonomous assimilation, which is not directly altered by the *D*-stimulus) rises, as *D*-excitability falls. The "down" change and expenditure of substance are thus delayed, since restoration is more vigorous than the normal; excitability to the sustained *D*-excitation also sinks more slowly. Finally, however, with constant stimulation, a new equilibrium must be reached between *A* and *D*, due on the one hand to the continuously declining *D*-excitability with declining magnitude of allonomous dissimilation, on the other to the continuously rising *A*-disposition and autonomous assimilation. From this moment, other *D*- and *A*-conditions being constant, there is no further alteration of substance; it remains at this lower level, consumption and supply being once more balanced.

Yet this reinstated equilibrium between *A* and *D* is essentially different from the state of autonomous equilibrium described above. For the balance is now maintained only through the action of the constant *D*-stimulus, which, owing to the diminution of *D*-excitability, now elicits less activity of allonomous *D* than in the former case—so that the purely autonomous *A* suffices to hold it in equilibrium. This state of equilibrium between *D* and *A*, in which the substance falls below par proportionately with the intensity of *D*-excitation, may be denoted *allonomous equilibrium*.

The down change in living matter caused by the *D*-stimulus comes, therefore, to an end, in spite of persistent excitation: a limit is reached that cannot be passed, by reason of the prevailing *A*-conditions. The substance has *adapted* itself to the constant action of the stimulus —↓ .

Just as we may conceive of external stimuli which compel the living substance to vigorous dissimilation, so others are conceivable which enforce greater activity of assimilation. This increase of assimilation, which is no longer purely autonomous, and is not balanced by corresponding activity of dissimilation, modifies the living matter in a direction contrary to that described above as

below par, and therefore to be denoted above par. At the close of such excitation the living matter is over-nourished; its disposition to assimilation is less than before, in proportion with the intensity and duration of the stimulus, and consequent preponderance of allonomous assimilation over autonomous dissimilation. The disposition to dissimilation is proportionately greater. Hence, at close of excitation, autonomous dissimilation preponderates over autonomous assimilation, and the living matter, owing to its gradual depreciation, returns to par.

As soon as the *A*-stimulus begins to act, a *plus* state *(ueberwerthigkeit)* of substance develops, in consequence of the preponderance of allonomous assimilation over autonomous dissimilation. This development implies depression of *A*-disposition and *A*-excitability, so that allonomous assimilation (notwithstanding constant stimulation) declines, while autonomous dissimilation increases with the increasing *D*-disposition in the substance. The disparity between the more active allonomous assimilation and less active autonomous dissimilation is accordingly lessened, and the *plus* state develops more slowly than would otherwise be the case. But since allonomous assimilation decreases steadily, while there is a constant increase of autonomous dissimilation, both must finally be equal in magnitude. The substance is now above par, and *A*-excitability is proportionately depressed, so that the constant *A*-stimulus can only effect an allonomous assimilation equal to the raised autonomous dissimilation from the rise of *D*-disposition.

Equilibrium may therefore obtain between *A* and *D* in three cases :—(1) when the living matter is at par, both *A* and *D* being purely autonomous; (2) with action of a constant *D*-stimulus, when dissimilation is allonomous, assimilation autonomous; (3) with action of a constant *A*-stimulus, when assimilation is allonomous, dissimilation autonomous. The first state is therefore *autonomous*, the other two are *allonomous* in equilibrium —— .

Descending and ascending alteration are two opposite functions, and mutually exclusive, as are also the states of below and above par which result from the 'down' or 'up' changes. The two processes of dissimilation and assimilation are also antagonistic, inasmuch as their consequences are opposite; but they are not mutually exclusive, and occur simultaneously. Other stimuli are thus conceivable which excite the living matter to an augmentation of the one process, with simultaneous reduction of the other. In proportion as such a stimulus tends to promote *D* and to reduce *A*, or *vice versa*, the down or up change in the substance

occurs the more rapidily. If, when a stimulus favourable to *D* and inimical to *A* is acting upon living matter, we could assume that the acceleration of *A*-disposition, concomitant with the rapid expenditure, tended to neutralise the *A*-depressing effect of the stimulus, the final restoration of a new allonomous equilibrium between *A* and *D* would be conceivable. But it is obvious that such a state would only be reached with a deficit much more pronounced than in the above case of the sustained action of a *D*-promoting stimulus. The same considerations apply to stimuli that are favourable to *A* and unfavourable to *D*.

While with stimuli that act in one direction only, *e.g.*, that are only favourable to *D*, the rise of autonomous *A*, which is a protection against too rapid expenditure, begins immediately after the commencement of the down change, along with retardation of the sinking of excitability to the persistent *D*-stimulus, so that the activity of allonomous dissimilation also sinks slowly; while further the new allonomous equilibrium, (by which a limit of expenditure is reached in spite of the sustained excitation) appears at a comparatively early stage of depreciation —these favourable conditions are lost so soon as a simultaneously *D*-promoting and *A*-reducing stimulus acts persistently upon the substance. Similar considerations apply to a stimulus that favours *A* and reduces *D*. This last class of stimuli, in common with that first described, induce a preponderance of dissimilation over assimilation, or *vice versa*, in living matter that is in autonomous equilibrium. Hence they might, *à fortiori*, be denoted as *D*- or as *A*-stimuli. They may further be characterised as producing an effect in both directions—in contra-distinction to the first class, which act in one direction only.

The fourth conceivable case, viz., such a modification of the living matter that *A* and *D* are simultaneously promoted or reduced, will be discussed below ——— .

We are thus acquainted with two phases of alteration in living matter, descending and ascending. The former appears under the action of a *D*-, the latter under that of an *A*-stimulus, taking the state of autonomous equilibrium between *A* and *D* as point of departure. The change effected by a *D*-stimulus, *i.e.*, *allonomous descending alteration*, reduces the substance more and more below par. If the stimulus ceases, the living matter returns, in virtue of its internal energy, to the mean state of autonomous equilibrium. It alters, therefore, in an inverse sense to the change during action of the stimulus is, *i.e.*, ascending. This is therefore an *autonomous ascending alteration*, by which the inherent energy of the substance compensates its expenditure, and returns to par.

If, on the other hand, the living matter in autonomous equilibrium is attacked by an *A*-stimulus, the substance undergoes *allonomous ascending alteration*, and rises more and more above par. At close of excitation the substance itself sets up the opposite process, *i.e.*, *autonomous descending alteration*, and thus returns finally to the state of autonomous equilibrium.

If the living matter undergoing autonomous descending or ascending alteration is submitted to an *A*- or *D*-stimulus, the process will be more or less delayed, or even arrested (with adequate strength of stimulus), if the excitation be sufficient to produce immediate allonomous equilibrium between *D* and *A*. With a still stronger stimulus, the opposite process appears, and so forth —— .

Up to this point we have not taken into consideration that the actual magnitude of assimilation, whether purely autonomous or allonomous, depends not merely upon the actual state of the living matter and its consequent disposition to assimilation or excitability to *A*-stimuli, on the one hand, and upon the intensity of a just effective *A*-stimulus on the other; but that further assimilatory conditions must also be given, *e.g.*, a certain intrinsic temperature of the substance, presence of adequate nutritive materials, &c. It follows that when consumption of the nutritive matters exceeds supply, assimilation cannot reach the same proportions as it otherwise would, at the given *A*-disposition of substance, or strength of just effective *A*-stimulus. So, too, in dissimilation. The physiologist has grounds for assuming that an excessive accumulation of dissimilation products obstructs the functions of living matter, although it is impossible to say whether the disturbance affects the processes of dissimilation and assimilation equally, or one more than the other. It is, however, conceivable that the formation of dissimilation products may occur more rapidly than the breaking down.

Deficit of *A*-materials, or accumulation of *D*-products, may be represented on the one hand as an indirect effect of excessive *A*- or *D*-excitation; on the other, as the consequence of quite different factors, *e.g.*, altered constitution of blood, disturbance of nutritive fluids, &c.

To take the first case. Supposing the *D*-products to have accumulated in consequence of an active *D*-process, which is thus obstructed, so that the living matter, although distinctly below par, with a marked increase of *D*-disposition, cannot dissimilate to the same extent as under normal conditions.—In such a case a *D*-stimulus will fail to call out the same activity of dissimila-

tion as under normal relations. This can only signify that the *D*-excitability of the substance no longer corresponds with the sum of the expenditure. We have thus strictly to determine between the *D*-disposition corresponding with the actual value of the living matter, and the other conditions of the normal process of dissimilation; and both the decline of the former, and any disturbance of *D*-conditions, may reduce the so-called excitability to *D*-stimuli.

A consumption of *A*-materials in excess of the simultaneous supply, with similar consequences during active *A*-excitation, is equally conceivable.

In illustration of the second case cited above,—let it be assumed that the supply of *A*-materials is from some cause defective, and no longer adequate to balance the autonomous assimilation. Autonomous dissimilation will then be maintained in the substance; but since assimilation, notwithstanding a sufficient *A*-disposition, cannot keep pace with it, the living matter undergoes descending alteration, until equilibrium with the diminished *A*-supply is finally re-established. If the *A*-supply fails altogether, while dissimilation continues, the store of *A*-materials will be quite used up, assimilation eventually ceases, and the living matter, in consequence of progressive dissimilation, undergoes radical and fatal alteration, exhausts itself, and perishes. The scantiest supply of *A*-materials, will, however, maintain a correspondingly low metabolism in the substance, as soon as equilibrium has been restored between the feeble assimilation and decreasing dissimilation. Each increment of *A*-materials will then bring back the living matter, in virtue of its augmented *A*-disposition (corresponding with the marked deficit), to energetic vital activity.

Let us further assume that the intrinsic heat essential to the normal process of metabolism cannot be maintained, from external causes, and sinks lower and lower. Then the living matter may be conceived as affected in both its processes in the same degree by this diminution of the proper heat. For in such a case dissimilation and assimilation decline, *pari passu*, without alteration of value in the substance, since the equilibrium between *D* and *A* is unaltered. And supposing dissimilation and assimilation to become minimal, the substance will not be dead, but dead *in appearance* only. Additional warmth at once restores it to its former activity.

When the depression of intrinsic heat has a more pronounced depreciatory effect upon the one process than upon the other, a new autonomous equilibrium between *A* and *D* is set up, *i.e.*, the

living matter adapts itself to the lowered intrinsic temperature in the same way as to a constant external stimulus.

The heat evolved, *e.g.*, in the process of dissimilation, must also be reckoned among the *D*-products, although not material, and must be given off again, if the substance is not to become overheated. Accumulation of heat affects the *D*- and *A*-processes equally, and is beneficial, or the reverse, according to the absolute height of the specific temperature. In this connection, much could also be said about the *internal self-adjustment of metabolism in living matter* (as already illustrated by many examples), but we must limit ourselves to a few salient instances.

We have distinguished the *D*- and *A-stimuli* which act upon dissimilation and assimilation—briefly, upon living matter—from other circumstances affecting metabolism, which we termed *D*- and *A-conditions*. This distinction is fundamental, although no absolute separation can be predicated. The external stimuli are adventitious factors; *D*- and *A*-conditions are, generally speaking, constant. As the metabolism of the living matter adapts itself to a *constant* stimulus (*supra*), so it is always adapted to what we have termed *D*- and *A*-conditions, so far as these are constant. When living substance is exposed to new *D*- or *A*-conditions—shortly speaking, to new vital conditions—these, as we have seen, act upon it at first as stimuli, until it is completely adapted to them. Thus the given intrinsic heat of any living substance may be reckoned among its vital conditions. A sudden rise or fall of internal temperature at first acts as a stimulus to living matter; but if the temperature remains constant, the substance adapts itself to it, and once this has occurred, the altered temperature ceases to be a stimulus.

It is, as we have seen, conceivable that the rise or fall of intrinsic temperature in any nervous substance may cause an equal alteration of *A* and *D*. We have already considered the simultaneous and co-extensive depression of both processes—when the value of the substance is not altered, but only the intensity of its metabolism. So, too, when *A* and *D* are reinforced in equal proportions, the living matter remains qualitatively and quantitatively the same, this is once more increased metabolism of substance, while the value is not altered.

Where, from any cause, there is a constant fall of assimilation while dissimilation continues, the latter also declines gradually, and metabolism is much reduced. The living matter then sinks more and more, and finally perishes; while if *D* and *A* are simultaneously and equally depressed, the value does not alter, and

energy is maintained. We have, therefore, to distinguish between the value of living matter and the intensity of its metabolism at any given moment, since intensity may vary under different vital conditions, value remaining constant.

If a part of the living substance were so broken up in dissimilation that its primitive constituents were completely transformed into *D*-products, the living matter would merely alter *quantitatively*, and assimilation too would affect *quantity* of substance only. The difference in bulk between *A*-materials taken up, and *D*-products given off, in the unit of time, would not only express the quantitative increment or decrement of the living matter under investigation, but would also be the measure of descending or ascending alteration in the substance. Hence there would be merely a *waxing* or *waning* of the living matter without alteration of its internal activities; the fluctuating value would be expressed in the increasing or decreasing bulk of the substance.

But the alterations in living matter cannot be thus simply represented, nor are the phenomena of its intrinsic waxing and waning the subject of the present discussion. The dissimilatory process modifies not only bulk, but also quality of the internal activity. When the substance has been protected for a long time from external stimuli, and left to itself, the normal conditions of metabolism being present, it arrives, as we have seen, sooner or later, at the state of autonomous equilibrium between *D* and *A*, denoted above the mean state. The amount in which it is present in the tissues is in the first instance taken for granted. It is determined by the sum of all the vital conditions that constitute the environment of the substance in these tissues. If the internal movements of the constant flow of living matter could be suddenly arrested, the existing chemical relations of its primitive elements would be spatially represented in definite quantitative proportions and arrangement. Thus the absolute volume, relative bulk of the primitive elements, and actual chemical composition of the substance conceived to be in autonomous equilibrium could be determined.

If the equilibrium between *D* and *A* were in any way disturbed, *e.g.*, in favour of dissimilation (the substance thereby undergoing descending alteration), and if its movements were then suddenly brought to a standstill, we should find not merely a deficit in the absolute bulk of the substance, but alteration of its internal chemical structure also. The partial loss of primitive matters, as converted into *D* products, implies an alteration of the whole remaining complex; the substance is no longer chemically the

same,—in so far as it is legitimate to speak of chemical constitution in a substance that is in perpetual, internal, chemical movement. For not merely is there partial abstraction in dissimilation (without alteration of the permanent remainder), but the living matter also forms *D*-products internally, at the expense of that activity which characterises the mean state. And this internal, chemical, and therefore qualitative, alteration is the index of the state of deficit. Through it the substance acquires the augmented tendency to assimilation, and diminished tendency to dissimilation, denoted above as rise of *A*-disposition, and fall of *D*-disposition. If preponderance of assimilation subsequently brings the substance back to the mean state, the acquired products are once more taken up into the chemical structure, the living matter becomes again what it was, and exhibits the chemical activity characteristic of the mean state.

When the living matter in autonomous equilibrium undergoes ascending alteration, and rises above par, this is no mere quantitative increase, without change of constitution, but signifies that what is taken in, in excess of the average, enters into the chemical structure of the substance, and qualitatively alters the whole of the living matter. This is characteristic of the *plus* state, and brings about the lowered disposition to further assimilation, as well as increased disposition to dissimilation.

We have therefore to distinguish between this qualitative, as well as quantitative, up and down change, and the specific formation of new living matter (increment of substance), together with its partial destruction or consumption, which are due to pure quantitative alteration. These changes do not come into the category of the processes now under consideration. We are assuming from the outset that the living matter in the part of the organism involved is already at the maximum possible to it under the given vital conditions.

Since the actual magnitude of assimilation and dissimilation is determined, on the one hand, by the circumstances which we have designated *A*- and *D*-conditions, or conditions of metabolism in general; and, on the other, by the factors denoted as stimuli; since further, alteration of *A*- or *D*-conditions may act as a stimulus on the living matter, while a new but constant *A*- or *D*-stimulus, to which the substance has adapted itself, may also be regarded as a new *A*- or *D*-condition,—it may be asked why we should distinguish between *A*- and *D*-stimuli, and *A*- and *D*-conditions,—between allonomous, and autonomous, alteration and equilibrium of substance. Even if the distinction is accepted *ab initio* in animal

physiology, it appeals less to the botanist. It is more particularly in regard to the nervous substance of the sense-organs, and to muscle and nerve physiology, that the distinction is called for. With no sharp line of division, the adventitious sense-stimuli are yet marked off intrinsically from other vital conditions of the excitable substance of the sense-organs; and the so-called artificial stimuli applied to the living matter of nerve and muscle, are still more distinct from the internal conditions of metabolism in these tissues.

The case is different for living substance, in which the so-called vegetative side of life predominates. The living matter of the contractile tissues, from the substrate from which our concepts of excitation, excitability, fatigue, &c., have mainly been developed, is fundamentally distinguished from most other living tissues, by the fact that the products of its chemical activity are also, generally speaking end-products of metabolism, the so-called *excreta*. This is not equally true of the living matter of the more vegetative organs in the animal organism. The products of their chemical activity are, in large part, matters which may be still further broken up in the same or other organisms, in part again such as serve immediately as *A*-materials for other living substances in the same or other animals.

We must not enter upon the question of how far the development of chemical products of vital activity in these last living substances (*e.g.*, manufacture of glycogen, fat, specific constituents of secretion, &c.), should be viewed as identical with the formation of *D*-products as described above; or if the internal chemical movements of living substance merely effect the conditions for another grouping of primitive matters,—not intrinsic to the complex of elements formed by the living tissue itself, but in direct contact with it, forming new combinations internal. In nerve and muscle substance, moreover, the parallel question recedes into the background, in comparison with the stand-point from which we have reviewed the functions of living matter. One point only claims attention. I see no reason why the pronounced development of vital energy manifested in muscular activity is to be predicated of the dissimilatory process in living matter in general. It would be easy to show that a tendency to this assumption exists in many quarters, and finds application in departments where it seems to be wholly unjustifiable.

If I have myself in this brief discussion dwelt mainly upon the processes in such living substances as it is customary to term irritable, or excitable, in the narrower sense, I am fully aware

that in other living substances, much would be prominent that is here only touched upon, or left untouched. On the other hand, the theory I have advanced seems to me broad enough to find application *mutatis mutandis* to other kinds of living matter also.

These excitable substances in the narrower sense are characterised by great lability of equilibrium, and comparatively rapid alterations of value. But since the changes in value are quantitative as well as qualitative, and the magnitude of actual *A* or *D* must be referred to quantity of substance, as standard to measure them by, we have next to seek some measure of quantity in the living substance.

When living matter has fully adapted itself to the last given *A*- and *D*-conditions (assumed to be constant), and has been sufficiently protected from the action of adventitious stimuli, the quantity of substance then present in the tissues may be taken as the standard to which to refer the intensity of the *D*- and *A*-process, could this be expressed in numbers. The quantity of *A*-matters consumed in the unit of time, referred to quantity of substance, would then measure the magnitude or intensity of the *A*-process, and the *D*-process may be similarly gauged, even where, in consequence of *A*- or *D*-excitation, the two are no longer in equilibrium, so that the substance has undergone descending or ascending alteration. The sum of *A*-matters consumed, and of resulting *D*-products, must still be referred to quantity of living matter present in the mean state, although the substance is now above or below par, *i.e.*, has increased or diminished in bulk. The same is true of the difference between *A*- and *D*-intensity, or between bulk of *A*-matters consumed, and resulting *D*-products, in the time unit; which difference, as referred to quantity in the living matter still at par, expresses *the rapidity of descending or ascending alteration* in the substance. This is the rapidity with which living matter allonomously or autonomously alters from, or returns to, the mean state. The magnitude of the whole change effected in a given time by the action of an *A*- or *D*-stimulus, in matter that was previously in autonomous equilibrium, together with the degree of *minus* or *plus* state of the substance, is, however, expressed by the difference in weight between the total consumption of *A*-materials, and the simultaneous yield of *D*-products in this time, as referred to quantity of substance in the state of autonomous equilibrium.

The quantity of living matter to which actual strength of assimilation or dissimilation is to be referred, in order to find a

measure of the alterations due to the action of an *A*- or *D*-stimulus (for rapidity of alteration as well as for degree of resulting *minus* or *plus*) is thus a varying magnitude, inasmuch as it is dependent upon the sum of *A*- or *D*-conditions acting at the moment, and taken as constant. It follows that the degree of increase or decrease in *A*- or *D*-disposition in the living matter is also dependent upon the same conditions. But wherever the action of a stimulus upon any excitable substance is to be investigated, the inevitable point of departure must be that state of living matter which obtains after long protection from external stimuli, under otherwise normal conditions—admitting that altered conditions might bring about a different equilibrium. Whether, when the vital conditions are permanently altered, the living matter can finally become qualitatively quite the same as before, cannot here be entered upon.

In the foregoing we have briefly considered the relations that obtain between the metabolism of living matter and adventitious external stimuli on the one hand, and the so-called *A*- and *D*-conditions on the other.

A third and essential relation has not, however, been touched upon, *viz.*, the reciprocal inter-dependence of the individual elements in living matter. We know that given alterations in state at any part of the living matter can produce alterations in the state of adjacent parts in vital connection with it—alterations of state in one part may act upon adjacent parts as a stimulus. Setting aside the hypothesis that sees a merely physical process in the propagation of excitation through a nerve, the transmission of "excitation" in living matter belongs, in the first degree, to those processes which prove that alterations of metabolism in one part of the living substance may excite the same in adjacent parts. Another salient illustration of the fact that function in any individual part of a nervous organ is in coördinated relation with the simultaneous processes in other parts, is offered by the facts of simultaneous contrast. But while in those processes, which are termed transmission of excitation, the adjacent parts of the altered substance appear to undergo exactly the same kind of alteration, the living substance of the visual organ manifests another kind of interaction, *i.e.*, the alteration of one part augments the disposition to the opposite kind of alteration, or even induces the correlative change, in the adjacent parts. Many other phenomena of nerve-life illustrate the one as well as the other kind of reciprocal action between the individual parts in a larger or smaller complex of living and coherent excitable

substance. The nature of this reciprocal dependence is unknown to us; it is only discovered in its manifestations. Yet we must be on our guard, in reference to the process of conductivity, against too hasty generalisations, founded almost exclusively on observations of the motor nerve-paths to striated muscle, and of peripheral nerves alone —— .

These considerations as to function in living matter, and in nerve-substance in particular, might be held to imply for every substance *one* kind of dissimilation and assimilation only, with the corollary that living matter can only undergo in *one* kind the changes described as ascending and descending alteration. But as early as 1874, I brought forward the opposite opinion. That the *A*- and *D*-process should always be of the same, or approximately the same, nature in living matter that subserves only one kind of function in the organism, is such an obvious conjecture that I will not venture here to insist on my own theory, according to which the conducted process of excitation is by no means exactly the same in two symmetrical motor nerves of the two halves of the body. Yet I must protest against the inadequate assumption that the chemical processes representing inherent vital activity are always qualitatively the same in the same nerve-fibre, or cell, or perfectly homogeneous in all nerve-fibres or cells. This theory has too long been paramount.

Johannes Müller's conception that different specific energies must be predicated for the different sensory nerves, can, in my opinion, be applied to the endless varieties of living matter in the plant and animal kingdoms. We must ascribe *specific* characteristics to the living matter of the germs of every species of plant and animal; and beyond this again, the living substance of each single germ has *individual* properties, from which proceed the intrinsic nature of its further development. It is the inherent property, the specific energy, of the living substance of the liver to secrete gall, as it is the specific energy of the living substance of the mucous gland to secrete mucin, &c. I have elsewhere expressed my general views on the question of specific energies.[1]

The activity of the gland, or the movement of the muscle-fibres bound up with the nerve, do not tell us *what* happens in the nerve-fibre, but only that *something* takes place in it. Nor do we learn much more from the nerve current, action current, and negative variation. In view of the manifold variety of the chemical pro-

[1] ("The Specific Energies of the Nervous System," Inaugural Lecture, 1882. "Lotos," v.)

cesses which may give rise to electrical currents, we must surely hesitate to conclude from the similarity of electromotive response in two nerve-fibres (especially in cases where excitation is followed by different central and peripheral reactions), that there must be similarity of internal process in dissimilar nerves—to exclude the possibility that certain nerves may transmit different kinds of internal alteration,—or even to assume that the same process takes place in all, with the possible exception of certain sensory nerves? With regard, finally, to the central nervous organs, how can we hope to explain the control (imperfect though it be) of movements in the lower limbs, notwithstanding the destruction of the majority of conducting paths in a localised area of the spinal cord, if we exclude the possibility that the impulse descending from the brain (after crossing the surviving bridge of the spinal cord, albeit with difficulty, as an unaccustomed tract), may return to its proper path on the other side, because the latter is constitutionally predisposed to the specific quality of the passing impulse.

Muscle, gland-cells, plant-cells, perhaps indeed every living substance, exhibit electrical manifestations under given conditions, which, in their mode of appearance, present a striking analogy with the electrical phenomena in nerve: must we conclude from this that the internal chemical processes that cause these phenomena are identical in the living matter of all these parts, or that when the same electrical manifestations are to be observed in one and the same substance, in two cases, the chemical processes which underlie them must necessarily be identical?

Du Bois-Reymond's investigations of animal electricity directed general attention, in correspondence with the prevailing scientific tendencies, to the physical tokens of vital activity, and many persons deluded themselves with the hope that the intrinsic nature of nerve-activity was disclosed in the electrical phenomena of the nerve. Even optical and morphological differences in nerve-fibre were wholly subordinated to apparent identity of electromotive properties, and uniformity of electrical reaction was held to justify deductions as to uniformity of chemical constitution in nerve-substance, or at any rate, uniformity of excitatory process. According to this view there could only be one process—at least in nerve-fibre as the conducting organ, and differences of time or intensity would alone indicate possible differences of function. An exception was at most admitted for sensory nerves, but here also there was a strong tendency to attribute the so-called specific energies to qualitative differences, not in the conducting paths, but in the centres. And if there was thus a disinclination to recognise

qualitative differences in the chemical structure of different fibres, or cells, of the nervous system, and in "excitations" passing along them, still less was there any notion that multiple qualities of "excitation" might exist in the same cell or fibre. Accordingly, my proposition, that various kinds of dissimilation and assimilation may occur in the same excitable substance, so that we must distinguish between various modes of *D*- and *A*-excitation, found no response, and was even contradicted, in the first instance. As much may indeed be said for the whole of the preceding theory. Its primary application to the physiology of vision was disputed by the majority of my colleagues, the "subversal of general nerve physiology" which they professed to find in it, being regarded as "non-proven in this direction."

Even the presumption, I might say the facts, of colour-antagonism were discredited.

In the course of the year, however, individual voices began to protest against the current theory of complete homogeneity of function in all excitation of nerve fibres, and all processes of nervous conduction. With the aid of my former pupil and fellow-worker, Biedermann, I succeeded in adding to the well-known facts of general nerve and muscle physiology, which (*infra*) can be adduced in support of the above theory—new data of such an order that the presence of antagonistic conditions became still more prominent in the electromotive and excitatory phenomena of nerve, or muscle, substance also. I, therefore, allow myself to hope that the preceding considerations may now meet with a more favourable reception than in my earlier attempts to bring them forward.

Nothing essentially new has presented itself in regard to the facts in the department of visual sensation, upon which I based my theory of light and colour sense; further consideration having only deepened my conviction that the principles I then advanced require no real alteration in order to derive from them all the main facts of visual sensation; while the few points still unexplained are nowhere in contradiction with it.

Nor has extended work in nerve and muscle physiology, as well as in general biology, failed to convince me that the views I formerly expressed more briefly as to function in excitable matter are a scarcely less legitimate deduction from the facts then known than from the more striking data now before us. A hypothesis proved is no longer a hypothesis. Thus it only remains to see whether the above propositions throw light on the facts; whether they give a working plan of their interconnection; and whether they are of predictive value.

PART II.

Modern physiology distinguishes between the state of rest and that of activity, or excitation, in excitable matter. In the terms of the theory proposed above, there could only be *one* proper state of rest for living matter, viz., that condition of autonomous equilibrium which we termed "apparent death"; because its simultaneous dissimilation and assimilation are so reduced that they may be reckoned at zero. But what the modern physiologist terms state of rest embraces not merely all the states of autonomous equilibrium, whatever the actual magnitude of assimilation and dissimilation, provided both are of equal intensity, but all those conditions also which were described above as characteristic of autonomous ascending alteration. It is true that the physiologist applies the specific name of "recovery," or restitution, to this kind of 'up' change; but the process itself is included in the general concept of rest, or non-activity, of living matter.

The different states of equilibrium between *D* and *A* may, of course, be regarded as states of comparative rest, since the living substance neither undergoes descending nor ascending alteration, but remains qualitatively and quantitatively the same. But that condition of equilibrium, which appears only when an external stimulus has acted constantly for a prolonged period, and to which the living substance has adapted itself, —denoted above the state of allonomous equilibrium—must be reckoned, in modern physiology, among the states of activity or excitation; at least, whenever the external stimulus is a *D*-stimulus. For it combines with the idea of general stimulation or excitation the notion of consumption, disintegration of living substances, and implies constant excitation, because an external stimulus is acting upon the substance.

The prevailing concept of excitation or activity really covers only those states or processes which we have designated as descending allonomous alteration. On the other hand, it is usual to associate the idea of excitation, or activity, with the idea of dissimilation, without reference to actual intensity of simultaneous assimilation; and many difficulties thus arise from the use of the ordinary terminology.

We have seen that if the assimilatory conditions of a nervous substance are disturbed, *e.g.*, by cutting off the blood supply, or by separating an organ from the rest of the organism, while its

D-conditions are not simultaneously affected to the same extent, autonomous descending alteration will make its appearance. But this may be indicated by symptoms which are also characteristic of allonomous descending alteration, whence we might erroneously infer that increase of dissimilation had taken place, instead of the contrary. If it may thus be assumed of any given nervous substance that it can, after stimulation, be "excited" by increase of dissimilation, as well as by decrease of assimilation, because there is descending alteration in both cases, we must distinguish between two fundamentally different kinds of stimuli as producing a 'down' change in nervous substance. In such a case it is obvious that the absolute increase of dissimilation at the excited point is not the sole factor in excitation; but that there must rather be such a disturbance of equilibrium between *D* and *A* that the former shall obtain a certain preponderance over the latter. The presumption is at least valid.

In all cases where "excitation" is not expressed by absolute increase of dissimilation, but by some symptom characteristic of descending alteration in general, the two alternatives as above must certainly be taken into consideration.

When artificial excitation of a nerve has provoked a demonstrable increase of dissimilation in the organ innervated, it is, in the first place, conceivable that the innervation acts as a *D*-stimulus on the living matter; in the second, however, that it acts as an *A*-stimulus, raising the living matter above par, and inducing augmented autonomous dissimilation. In the first case, its action on dissimilation would be direct; in the second case, indirect.

I must confine myself to these few remarks upon the different possible interpretations (as suggested by the foregoing discussion) of the same symptom of "excitation." The theoretical enumeration of every conceivable case would only be fatiguing, and what has been said suffices to show that the prevailing conception of many vital phenomena suffers from a certain onesidedness, which may easily give a warped direction to further investigation —— .

In conclusion, and to complete what was said in the first section, I will briefly point out how the essential facts of general muscle and nerve physiology can be brought under the same theoretical propositions. It was, indeed, from these facts, that the theory first developed itself. Not that it would be possible to deduce the above propositions solely from experiments on nerve and muscle fibres;—they derive from many different departments of biology, and, in particular, from the physiology of the senses, and from the general physiology of metabolism.

When, nearly twenty years ago, these propositions were formulated from the phenomena of life—physical and psychical—the prevailing conception of the facts of general nerve physiology was strictly physical. I had, therefore, to insist, especially as against du Bois-Reymond's almost exclusively physical account of function in the nerve fibre, that these functions were essentially chemical, and that the intrinsic chemical nature of vital processes must not be overlooked in favour of their physical symptoms. Du Bois-Reymond's theory of nerve and muscle currents is purely physical. It is true that he subsequently described the supposed electromotive molecules as a definitely orientated crowd, with pronounced chemical activities; but he explained the changes of electromotivity occurring under different conditions in nerve and muscle, not by alterations in chemical activity, but by changes in the position of the molecules. My own view is, in some respects, more nearly allied to that of Hermann; but he too, in consequence chiefly of his researches into electrotonus, and polarisation in nerve and muscle, has turned more and more to a physical conception of nervous activity.

For my own part, I have only detected in the electrical manifestations of nerve and muscle, physical symptoms from which no more can be inferred as to the qualitative aspect of the functions of living matter, than from their thermic manifestations. The galvanometer, or thermometer, only express the alterations and differences of function in different parts of a living continuum, as well as the time distribution and quantitative relations of the same, and reveal nothing as to its quality.

Hermann's weighty dictum of the iso-electricity of uninjured "resting" nerve or muscle, signifies to me that such a tissue develops no current that can be led off externally, *so long as its metabolism*, i.e., *internal chemical function, is equal at all parts. Every disturbance of this equilibrium sets up currents that can be led off.* Alteration of chemical function in any part of a living continuum may, however, be expressed, not merely in its becoming *negative*, but equally in its becoming *positive* to unaltered parts. Hence, if we are to characterise the point differing in chemical function from the rest of the substance, as (relatively) altered, we must, in my opinion, *distinguish between a (relatively) positive and a (relatively) negative alteration.* And it is not altered chemical composition, but altered chemical function, which may lead to altered composition, that characterises this change. This conception thus expressed may be worked out in detail, according to the propositions developed in Part I.

We must, accordingly, distinguish, in the living matter of nerve and muscle, between ascending alteration, descending alteration, and state of equilibrium. Both ascending and descending change may occur at a very different rate, according as the intensity of assimilation, referred to the unit of substance, exceeds the intensity of simultaneous dissimilation, or *vice versâ*. If all parts of a living continuum are in equilibrium, or alter at the same rapidity, ascending or descending, there will be no current to lead off. Every difference in rate or direction of alteration, however, produces a current that can be led off (so that we may include the state of equilibrium between *A* and *D* under this general proposition, as an ascending or descending alteration at zero velocity). *We may, accordingly, conceive of all the different rates of positive or negative alteration as forming a series, of such a character that the most rapid ascending alteration forms the upper*, i.e., *positive—the most rapid descending alteration the lower*, i.e., *negative—end of the series.*

If two parts of a living continuum, which differ in chemical function, are joined by an external circuit, there will, *cæteris paribus*, be a stronger current, in proportion as the P.D. of the two points connected in circuit is greater in the above series, and the positive current will always flow through the external circuit from the point nearest the positive end of the series to that nearest the negative end. *This is the universal law of all physiological currents (vitalen Eigenströme) in nerve and muscle.*

A sartorius muscle exposed with every possible precaution, *e.g.*, one that is no longer normally nourished, will presumably undergo descending alteration (however much retarded) because dissimilation preponderates over assimilation; it is slowly dying. If the 'down' change occurs in all parts with equal rapidity, or delay, no current will be detected, even by the most sensitive galvanometer. This ideal case is never, of course, fully realised. But with a moderately sensitive galvanometer there will actually be no sign of current in such a muscle, as was conclusively shown by du Bois-Reymond (parelectronomy), and by Hermann (isoelectricity of uninjured muscle). As soon, however, as a transverse section is made in the muscle, a more rapid down change sets in at the injured point of the muscle-substance; the part immediately adjacent to the cross-section mortifies. This dead part is no longer included in the living continuum, and must be regarded as an inessential tag. The accelerated descending alteration and death, however, proceed slowly in the muscle-fibre, as may sometimes be followed directly

with the microscope; and a more rapid down change invariably occurs at the cross-section than in the rest of the fibres. *The transverse section is, therefore, negative to the longitudinal surface of the muscle.*

So, too, with the longitudinal-transverse nerve current; and it should be remarked that the dying of the nerve as a whole is a slower process than the death of muscle; and that the assimilatory conditions of the excitable substance in excised medullated nerve are more favourable than in excised muscle.

The so-called *state of excitation in nerve or muscle* is equally characterised by descending alteration in the living matter. Each "excited" point becomes negative to each "unexcited" point, even where the whole living continuum is already undergoing a much slower descending alteration. Upon this depend the "*action-current*" (Hermann) and the "*negative variation*" (du Bois-Reymond).

"Action-currents" are, in my opinion, due to the same causes as the currents of the so-called resting nerve or muscle, in so far as both must be regarded as the external symptom of a different rate of 'down' change in the two leading-off points connected in circuit.

How far the single negative swing (*negative Einzelschwankung*) is analogous with the current-action has been sufficiently discussed by Hermann.

In my opinion, we must distinguish from the action-current in Hermann's sense (which is due to a 'down' change at one of the leading-off points) another kind of action-current, produced by an 'up' change at one lead-off, while the other is not necessarily affected in the downward direction.

Isolated muscle is quickly fatigued, and soon succumbs to "excitation," while excised medullated frog's nerve exhibits extraordinary resistance even under prolonged excitation (Wedenski, Maschek). This coincides with the more favourable conditions of autonomous assimilation in isolated medullated nerve, as indicated above. So, too, the negative variation of muscle diminishes with extraordinary rapidity on prolonged stimulation, unlike that of nerve; there may even be a *positive after variation* in the latter at the close of excitation. During excitation the longitudinal lead-off of the nerve is thrown into rhythmically recurring, rapid, descending alteration, and its value is depreciated. If it had been undergoing slow descending alteration before excitation, or was still in a state of equilibrium between D and A, it will, at close of the stimulation, be below par, and accordingly exhibits autonomous ascending

alteration, *i.e.*, becomes, in comparison with the continuous descending alteration at the transverse section, more positive than in the first instance. In excised muscle, owing to inadequate power of spontaneous recovery in the living matter, no such reaction is demonstrated after excitation; there is not even a reappearance of the original muscle-current, let alone any temporary increase of it.

The positive after-variation is most easily demonstrated on the olfactory nerve of the pike. This nerve, as Kühne discovered, exhibits marked electromotive force, since it practically consists of axis-cylinders, *i.e.*, of intrinsically excitable substance; while the accessory sheath of medullated nerves is wanting. Unfortunately, this nerve perishes more rapidly than, *e.g.*, the frog's sciatic. On leading-off the longitudinal-transverse current of a carefully prepared olfactorius, and at the same time making a rapid cut with sharp scissors near the other end of the nerve, this single stimulus will produce a negative variation, followed, as long as the nerve remains in good condition, by an unmistakable positive after-effect. I mention this experiment, with which I have long been familiar, because it does not involve introduction of any external electrical current.

If a nerve or muscle is brought longitudinally into the circuit of an electrical current, an allonomous 'up' change occurs at the point where current enters the uninjured living tissue, an allonomous 'down' change at the point of exit. This last causes the place of exit to become the starting-point of an excitation ("*closure excitation*"), *i.e.*, a down change spreading through the living continuum. If, *e.g.*, the living matter had previously been at par, and, therefore, in autonomous equilibrium between D and A, it rises above par at the point where the current enters. When the current ceases to flow, there is a corresponding autonomous down change at the point of entrance, which is the more rapid in proportion as the substance had risen above par during the previous up change. Thus, the point of entrance may become the starting-point of a second excitation ("*opening excitation*") spreading over the fibre. At the point of exit, on the contrary, there is an autonomous up change on breaking the current, provided this point has not been seriously injured by the previous action of the current, or, generally speaking, disturbed in its assimilatory conditions.

Since a rapid allonomous 'down' change occurs during the passage of current, at the point of exit, this point is negative to the rest of the fibre (in so far as the latter is not in transmitted "excitation"); while the point of entrance, in consequence of

localised allonomous 'up' change, gives the opposite reaction. This causes an internal current in the fibre, opposed in direction to the led-in "foreign" current. This internal current withers the foreign current. It has been termed a "polarisation current." But inasmuch as it is a *physiological heterodromous current*, an intrinsic vital manifestation, it must be rigorously distinguished from those polarisation currents which are not properly physiological, since they do not arise from the up or down changes in the living substance, at the points where current enters or leaves it: *i.e.*, heterodromous currents may also appear, with artificial excitation, in dead tissues, or parts that are no longer intrinsically excitable in the still living organ.

Given normal activity of living matter, an autonomous 'down' change, may, as we have seen, arise at the anode at break of the led-in, foreign current, since this point is now negative to the rest of the fibre, in so far as the latter is not undergoing progressive descending alteration; while the kathode in virtue of an autonomous 'up' change, may become positive to the rest of the fibre. In this way a physiological current is developed in the fibre, in the same direction as the previously opened foreign current. This current may be termed a *physiological homodromous*, in contradistinction to the above *physiological heterodromous* current. It appears the more certainly in proportion as the substance is more energetic; and the less the vital processes are affected by the foreign current, the more rapidly will the allonomous alterations induced by the latter (after-effect of excitation) disappear, and the opposite autonomous changes develop, when it is broken. The homodromous physiological current is more or less likely to be disturbed by complication with physical polarisation currents, of opposite direction to foreign current.

If an external current is led through the central portion of a medullated nerve, the points by which it enters and leaves the excitable matter spread far beyond the contacts of the physical electrodes. So far as these points of entrance and exit extend, there is correlatively with the distribution of the lines of current, a purely *physical* "an- and kat-electrotonus;" as may be demonstrated, *e.g.*, on a dry hollow stalk of grass without internodes, or on a bundle of the same stalks that have been lying for some time in distilled water, or weak alcohol, and are then moistened externally, and saturated internally, with salt solution.[1] From

[1] I demonstrated this experiment at the end of the sixties in Vienna. With regard to distribution of lines of current, due to the peculiar relations of conductivity in medullated nerve, I am, therefore, of the opinion to which Grünhagen finally subscribed.

this wide distribution in the excitable substance (axis-cylinder) of the nerve, of the collective points at which the external current enters and leaves it—*i.e.*, the *physiological* anode and kathode proper—those 'up' and 'down' changes develop respectively in the nerve, which are fundamental to physiological *electrotonus* (Pflüger). Both down and up change may, after closure of the foreign current, be transmitted along the nerve beyond the tracts altered in a kathodic (negative) or anodic (positive) sense by the direct action of the current, so that fugitive alterations may occur even in very remote parts of the fibre, as expressed in its electromotive reactions. On breaking the foreign current, an opposite change appears at the points of entry or exit, together with its correlative effects in the living substance, *i.e.*, an autonomous down or up change. The two points have interchanged their parts; the up change, characteristic of physiological anelectrotonus, now appears at the former point of exit, the down change significant of physiological kat-electrotonus, at the former point of entrance.

In non-medullated nerve, *e.g.*, olfactorius, and in muscle, where the excitable substance, unlike medullated nerve, has no imperfectly-conducting sheath, the characteristic diffusion of entrance and exit points is wanting. The electrical phenomena which depend upon this diffusion (due, in the first place, to relations of conductivity), together with the physiological *local* consequences of the same, are accordingly absent. On the other hand, the phenomena caused by *transmission* of the up or down changes induced at the anode or kathode points of the foreign current, are more or less plainly exhibited both in non-medullated nerve and in muscle fibre.

If a tract of nerve has been traversed for some time by an external current, and the current is then reversed, the excitable matter at the point of exit (*i.e.*, former point of entrance) will be absolutely, or relatively, above par, and thus has a greater disposition to down change; the current accordingly produces a more rapid descending alteration than would otherwise be the case (Volta's alternative).

Muscle fibre, as compared with nerve fibre, has the great advantage of expressing the excitation due to descending alteration by change of form of the part affected; while a foreign current can, moreover, enter and leave by the natural ends of the fibres. With the former, the allonomous change which occurs at closure at the point of exit is, in the first instance, transmitted along the fibre, but after the closure twitch has expired it

is confined to the vicinity of the kathode during the passage of the current (*persistent kathodic contraction*), and steadily decreases. Meantime, the autonomous up change continues at the point of entrance, and may raise the living matter considerably above par, given adequate strength and duration of current. At break, there will, therefore, be an autonomous down change, which, if sufficiently rapid, may produce an *opening twitch*, or *persistent opening contraction*, in the vicinity of the anode. Even when this autonomous down change is so weak that no visible alteration of form can be detected in the muscle, it may express itself in the physiological homodromous current (*supra*), which appears on connecting the anodic end with, *e.g.*, the centre of the muscle.

Autonomous ascending alteration cannot always be demonstrated at the kathode, on breaking the internal current, because the autonomous assimilation of the living matter in excised muscle is too slow and inadequate a process—as was pointed out above. Yet in favourable cases the autonomous upchange is exhibited in a physiological homodromous current, that makes its appearance at break, if the now kathodic end of the muscle is put in circuit with the centre.

The fact that muscle, like nerve-fibre, fails to react to transverse passage of current, obviously signifies that living matter is not identically the same living continuum in the transverse as in the longitudinal direction: as also appears from optical polarisation phenomena, and from the relations of elasticity. The failure in reaction is perhaps due to the fact that the antagonistic points at which the current leaves and enters are too closely approximated in the structural elements traversed at right angles by the current.

When a strong foreign current has been flowing longitudinally through an uninjured muscle for so long that the persistent kathodic contraction has already expired, the persistent anodic contraction (*supra*) will appear when the current is broken, and may extend over a large tract of the muscle, and last for a considerable period. If the current is then closed again, it acts as an *inhibitory stimulus* to the contracted muscle, which at once relaxes completely. The anodic stimulus of the foreign current, which tends to upward alteration in the substance, now works against the rapid autonomous down change that prevails after break at the point of entrance, and substitutes an up change. Owing, however, to the previous and exhaustive allonomous descending alteration, there will not necessarily be a renewed make contraction at the Kathode.

Just as the persistent opening contraction of a muscle may be inhibited by renewed closure of the current, so another contraction depending on autonomous down change may be inhibited by the action of an anodic current. If, just at the beginning of systole, a strong current is sent in through one brush-electrode, the point of which rests upon the frog's heart (exposed with uninterrupted circulation), while the other electrode forms contact with, *e.g.*, the skin of the throat, a more or less extended diastole of the heart-wall, starting from the point where the current enters, will make its appearance. The commencing autonomous down change is immediately converted, by the anodic action of the currrent, into an allonomous up change, and the relaxed part of the cardiac wall swells out freely in consequence of blood pressure. The contrary effect appears when current leaves the heart by the brush-electrode. If closure occurs at the beginning of the general diastole, a new systole will at once appear at the point of exit (*kathodic closure contraction.*)

If the current is left undisturbed for some time in this last direction, and is then opened during a general diastole, the wall of the heart near the brush-electrode will not take part in the ensuing systole, owing to the marked autonomous up change; it remains diastolically relaxed, and the systolic pressure of the blood causes the relaxed point to swell out considerably. This is the *kathodic opening inhibition*, which thus expresses itself in precisely the same way as the *anodic closing inhibition* above described, and cannot be viewed as a mere fatigue effect. If, on the contrary, current *enters* the wall of the heart for a prolonged period by the brush-electrode, a contraction appears immediately after it is broken, in the proximity of the Kathode. This contraction may even be more pronounced than the natural systolic contraction, as appears externally from the paler colouring of the heart-wall. This is the *anodic opening contraction* derived from autonomous descending alteration, the analogue of the *kathodic closure contraction* described above, which depends upon allonomous descending alteration.

The anodic opening contraction and kathodic opening relaxation are fundamentally analogous with the phenomena of successive contrast, as observed in other living substances, and are as little as these, to be referred to a mere fatigue effect.

Cardiac muscle is an example of the living substances which, in the state of equilibrium between D and A, vary to and fro in a regular alternation of down and up changes, so that during the period of ascending alteration the previous down change is com-

pletely neutralised. Such a "periodically active" substance does not become exhausted, however long or short the periods, provided its assimilatory conditions remain undisturbed, and its dissimilation be not unduly increased by other influences. Within certain limits, it is capable of adapting itself to altered *A*- and *D*-conditions, or *A*- and *D*-stimuli, which may effect alterations in the period, as well as extent, of the individual changes.

Periodic "activity," in which the single up and down changes do not neutralise each other, produces an alteration in the average value of living matter, which is usually denoted as fatigue when it is descending, and is of appreciable magnitude. Tetanising excitation of the nerve also involves a periodic alternation of up and down changes; and at close of excitation an autonomous up change (positive after-variation in tetanised nerve) may appear when the substance is below par.

These indications may suffice to show the applicability to the functions of nerve and muscle substance, of the general propositions laid down at the beginning. I have laid stress on points common to both substances, since a more exact enquiry into individual questions would have to take account of the discrepancies. I am, however, far from wanting to bring all living matter under the same law, in spite of my conviction that vital function is everywhere comparable in its fundamental principles. For the rest I can only refer to the "Beiträge zur allgemeinen Nerven und Muskel Physiologie," issued from the Physiological Institute at Prague, and published in Vienna, from the year 1879. These papers are the sequel to the considerations discussed in Part I., and are, generally speaking, the extension of the same points of view, since they furnish the reader with many more examples of what was there brought forward.

F. A. Welby.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

January 14.—Dr. G. H. Savage gave his inaugural address on "Heredity in the Neuroses."

March 4.—Dr. F. E. Batten read a paper on "The Muscle Spindle under Pathological Conditions," and illustrated his subject by means of lantern slides, photographs and sections under the microscope. Professor Sherrington and Professor Victor Horsley also made communications on Muscle Spindles, and illustrated their remarks by lantern slides and sections under the microscope. In the discussion which followed, Miss Forster, Professor Haycraft and Dr. Mott took part, and Dr. Batten replied.

April 22.—A clinical meeting was held, at which the following cases were shown :—

Dr. Ferrier.—Myoclonus epilepticus. A remarkable form of nervous disease occurring in several members of one family. Two brothers, aged 16 and 13, with periodical fits, and constant irregular muscular spasms affecting almost every muscle in the body. Both became affected at the age of 7 years, as did an elder brother, who died aged 17. The disease steadily progresses, causing an ataxic spastic gait; the elder boy is now unable to stand, and the younger is about the same or a little worse than the other was three years ago.

Dr. Donkin.—(1) Probably subacute poliomyelitis. Man, aged 45 years, with paralysis and atrophy of the whole left lower extremity, supervening on an accident to the foot.

(2) Tabes with a great preponderance of sensory disturbance. Man, aged 34 years, manifold disturbances of sensibility and some loss of co-ordination in upper and lower extremities, some affection of sphincters, absent knee-jerks, doubtful wasting of some muscles of his hands, history of syphilis.

(3) Pseudo-hypertrophic paralysis in an advanced stage. Boy, aged 11 years, with paresis and wasting of some muscles of the lower extremities dating from the age of 7 years. Knee-jerks absent, electrical reactions normal.

Dr. Gossage.—Progressive muscular atrophy commencing at the early age of 10½ years. The muscles of the left thumb were first affected, and during the next eighteen months the atrophy spread up the forearm and arm to the shoulder. The affection is limited to the one side, and has been stationary for the last 15 months.

Dr. Lunn, Dr. Beevor and Mr. Ballance.—Case of removal of cerebellar tumour. C. J., aged 49 years, admitted to St. Marylebone Infirmary, October 4, 1894. Symptoms (not relieved by antispecific remedies): Headache, purposeless vomiting, optic neuritis, weakness of right arm, right knee-jerk more brisk than left, vertigo, lateral nystagmus, tendency to fall to left, cerebellar gait. Operation, 1st stage, November 19, 1894; 2nd stage, November 26, 1894. Tumour removed was an encapsuled spindle-celled sarcoma, weighing 173 grains. It was situated at the anterior part of right lateral lobe of cerebellum. Patient recovered, but with the loss of the right eye.

Dr. James Taylor. — Syringomyelia with bulbar symptoms. Female, aged 29 years. Six months ago began to be troubled with difficulty of breathing, which has persisted. For some years had not been able to appreciate the heat of water or fire with her hands. Now marked analgesia and thermo-anæsthesia on hands and arms, slight wasting of 1st dorsal interosseus of left hand, wasting of tongue, paralysis of palate and of abductors of vocal cords.

Dr. Harris.—Man with syringomyelia. Two distal phalanges of right hand lost from whitlows, which date from 15 years ago. Analgesia and thermo-anæsthesia of arms and upper half of the body. According to the history there had been rapid wasting of left arm, with severe pain in it since a bad fall on the left shoulder four months ago, indicating a probable hæmorrhage into the cavity, if the patient's account is reliable.

BRAIN.

PART III., 1897.

Original Articles and Clinical Cases.

ON THE ENDOGENOUS OR INTRINSIC FIBRES IN THE LUMBO-SACRAL REGION OF THE CORD.

BY ALEXANDER BRUCE, M.A., M.D., F.R.C.S., ETC.

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For a number of years after it was ascertained that the posterior columns of the spinal cord were composed of two great tracts—an internal (column of Goll or postero-median column) and an external (column of Burdach or postero-external column)—it was generally supposed that these two tracts were found in the lumbar and sacral regions as well as in the cervical and dorsal portions of the cord, in which they were first described. Some text-books of anatomy and nervous diseases still make this statement, or if they do not do so directly, at least leave this opinion to be inferred. It has gradually, however, come to be recognised that this view is erroneous, and that in the lumbar and sacral regions it is not possible to separate a distinct postero-median column from a postero-external column in the same manner as can be done in the dorsal and cervical regions in cases of ascending degeneration. The differentiation of the two tracts does not become completed until the level of the eleventh dorsal segment is reached. Below this level the long, intermediate, and short ascending fibres (of Singer and

Münzer) are intermingled, since the long fibres which pass from the lower posterior root ganglia to the medulla have not yet all reached the position near the posterior median septum which they ultimately occupy when they enter the postero-median column. In the second place a considerable proportion of the fibres of the column are not continuous with the posterior roots, but are derived from nerve cells in the grey matter of the cord itself. These fibres have been termed by Marie *endogenous* fibres, and it is to these that this article is devoted.

These *endogenous* fibres form two very well-marked tracts, one lying in the anterior part of the posterior column in close apposition to the posterior cornu, commissure and septum; the second in immediate relationship to the posterior median septum, and in part, to the surface of the cord. The former of these has been termed by Marie (1) the *cornu-commissural tract*, to indicate that it was regarded as connecting different levels of the grey matter of the cord. The German title dorsales Hinterstrangsfeld, indicates its topographical relations. The second tract has been termed by Dr. Muir and myself (19) the *septo-marginal tract*, the name being designed to indicate its relationship to the posterior median septum, in the same manner as the sulco-marginal tract of Marie is related to the anterior median sulcus. Obersteiner has also termed this tract the dorso-median sacral bundle (dorso-medianes Sacral-Bündel) and Edinger has called it the medianes Hinterstrangsfeld.

In order that a set of fibres may be regarded as of endogenous origin, there must firstly be an absence of degeneration in it when the posterior roots (or exogenous fibres) are divided or destroyed; and (secondly) it should itself degenerate when that portion of the grey matter from which it arises is atrophied or necrosed. These two conditions hold good in the case of both of the tracts mentioned.

The *cornu-commissural tract* is spared in locomotor ataxia, [which is now by very many neurologists recognised as a disease of the posterior roots,] even in its advanced stages, and in injury to, or compression of the cauda equina. On the other hand, it is found to undergo degeneration in

conditions which lead to the atrophy, or degeneration of the cells in the posterior cornu. Thus Ehrlich and Brieger, and Singer and Münzer (4) after causing necrosis of the nerve cells of the lumbo-sacral region by compression of the abdominal aorta, and thus cutting off their blood supply, found a distinct but somewhat diffuse degeneration throughout the whole of the posterior columns, but most marked in the anterior part of these columns. In pellagra, which is regarded as a disease of the grey matter of the cord, Tuczek (10) and Marie (1) found it distinctly affected. Still more recently Schlesinger (11) has shown that it is frequently degenerated in syringo-myelia, a disease which tends to destroy the grey matter, especially that portion of it in proximity to the central canal. Lastly, Fajersztajn (9) has shown that it is composed of fibres of short course, the majority of which have a descending direction. On the other hand, Déjérine and Spiller (7) maintain that it contains a considerable number of ascending fibres derived from the posterior roots.

Sections derived from advanced cases of locomotor ataxia are particularly well suited for the study of this tract. When these are stained by the Weigert-Pal method it stands out with great distinctness against the degenerated fibres. It extends through the whole of the lumbo-sacral region of the cord, being traceable from the lowest dorsal segment to the extreme tip of the conus medullaris. It attains its greatest size at the level of the lower lumbar region, and diminishes somewhat rapidly above and more gradually below this level. Throughout its whole longitudinal extent it stands in close relation to the posterior commissure, and (except perhaps in the lowest sacral segment) to the anterior part of the inner margin of the posterior cornu. Its inner margin, below the third lumbar segment, lies in contact with the posterior median septum. Above this it becomes gradually displaced outwards by the ascending fibres of the posterior roots of the lower spinal nerves, as they pass upwards to enter the postero-median column. When this latter column is fully developed, *e.g.*, at the level of the eleventh dorsal segment, what remains of the cornu-commissural tract is displaced

from the septum completely, and also from that portion of the posterior commissure which lies immediately in front of the tip of the postero-median column. Posteriorly the tract has no definite margin. Its outer portion merges gradually into the part of the column behind it (the so-called middle root-zone of Flechsig), while in the neighbourhood of the septum, below the level of the first sacral segment, its inner portion becomes fused with the septo-marginal tract, in such a way that in sections derived from ataxia it cannot be determined where the one ends and the other begins.

A fuller idea of the topographical relations of the tract may be obtained by a study of figs. 1—9, which have been made from careful drawings of Weigert-Pal sections of an advanced case of ataxia. It will be seen from these that at the fifth sacral segment (fig. 9), the tract is in contact with the posterior commissure and the anterior part of the septum, but that it does not quite reach the cornu, being separated from it by a narrow area of degenerated fibres. Posteriorly it passes, without any line of demarcation (fig. 9), into the septo-marginal tract, appearing to form with it a continuous tract extending from the commissure to the periphery.

At the fourth sacral segment (fig. 8), the external margin has reached the cornu and extends along it backwards as far as the gelatinous substance of Rolando. The other relations are unaltered. The tract is not yet of any great extent, and the apex of the column is narrow and pointed.

In the upper sacral segments (figs. 7, 6, 5), the tract has considerably increased in size. The external margin extends further backwards along the posterior cornu, and the inner border passes along the septum to become fused with the septo-marginal tract.

At the fourth and fifth lumbar segments (figs. 4 and 3), at which levels, as already stated, the tract seems to have attained its maximum size, the inner margin does not extend so far along the septum, and is now distinctly differentiated from the septo-marginal tract. This latter is now limited to a small area at the posterior half of the septum. The separation of the two tracts seems to be effected

by the ascending (in this case degenerated) fibres of the posterior roots, which are becoming applied to the septum in their passage upwards to aid in the formation of the column of Goll. As additional fibres from the posterior roots become applied to these at higher levels (upper lumbar, fig. 8), the separation of the cornu-commissural and the septo-marginal tracts gradually increases (as in ataxia these root-fibres are degenerated the intervening space is paler than either of the tracts in question). The cornu-commissural tract is displaced outward, till at the twelfth and eleventh dorsal segments (fig. 1), it comes to form a narrow band bordering the posterior commissure and grey matter of the posterior cornu.

Above this level it is not easy to distinguish it as a distinct tract, but it apparently diminishes rapidly in size. The fibres which compose it can be seen entering it from the grey matter of the posterior cornu, and almost exclusively from that of the same side.

The *septo-marginal descending tract* also remains undegenerated in ataxia, and its position very closely corresponds to the area of fibres which Dr. Muir and I (19) found to degenerate after a crush of the cord at the level of the upper lumbar segments. In this case very little degeneration was found in the cornu-commissural tract, a proof that the greater part of the fibres of the latter arise below this level, while many of the septo-marginal tracts have a higher origin. This is in agreement so far with Hoche's observations as to the high origin of the tract. [The lesions in Hoche's cases were respectively below the level of the eighth cervical, and between the fourth and fifth dorsal segments (12).] It also enables us to indicate where the septo-marginal and cornu-commissural tracts meet each other. The fact that it has degenerated in a downward direction, renders it quite certain that it is not an upward continuation of posterior root fibres, and this has been corroborated by the observation of Pineles (23), that it remains unaffected in ataxia affecting the sacral segments. It stands out from the degenerated fibres in the lumbo-sacral portion of the cord even more distinctly than does the cornu-commissural tract.

At the level of the lowest sacral root (fig. 9), the undegenerated area in question is situated at the postero-internal angle of the posterior column. It forms a racquet-shaped area, or a triangle with the edges rounded off, two of its sides being applied to the posterior median septum, and to the periphery respectively, and the third forming a somewhat indefinite margin between the degenerated and undegenerated parts of the cord, which at this level are not sharply differentiated from each other. The anterior angle of the tract seems to merge into the undegenerated area lying in the anterior part of the column—the cornu-commissural tract. The remainder of the posterior columns is not so degenerated as at higher levels, so that the healthy areas do not stand out so clearly. In the next segment, the fourth sacral (fig. 8), the tract has greatly altered. It has become narrower, and much elongated antero-posteriorly. In the greater part of its extent it forms a very narrow band along the posterior median septum, which as it nears the periphery, suddenly widens out into a club-shaped head. This head is rendered more prominent by the apparent shrinkage of the degenerated areas of the column, which produces a retraction of this part of the periphery of the cord from which the rounded club-like head of the tract projects boldly, the depression thus formed in the outline of the cord being filled in by the connective tissue and vessels of the thickened membranes. It is seen in the illustration as a pale area on the outer side of the projecting head. At this level the undegenerated area is very distinctly marked off from the remainder of the column. In the third sacral segment (fig. 7), there is very little change in the appearance of the tract, what change there is being in the shape of the head. This instead of being rounded is almost pointed, like the head of a broad spear, and projects further beyond the remainder of the column than in the former section. The recession of the periphery is deeper and more angular, and is as before, filled up by the thickened membranes. In this, as in the previous sections, the anterior end of the tract becomes merged in the fibres of the undegenerated cornu-commissural tract.

At the level of the upper sacral segments (figs. 6 and 5), the tract has become distinctly larger, the band along the posterior median fissure being broader and the head also larger. The head still retains its pointed appearance, but does not project beyond the general line of the periphery of the cord: the angular depression at its outer side is still filled up by connective tissue. The tract continues to be sharply differentiated from the remainder of the column and to merge in the cornu-commissural tract anteriorly.

When the fifth lumbar segment is reached (fig. 4) there is again a great change in the appearance of the tract. It has greatly diminished in extent, now occupying only about a third of the length of the posterior median fissure instead of the greater part of the length; and, further, it no longer reaches either the cornu-commissural tract or the periphery of the cord, but forms a narrow tract, with a rounded posterior extremity and tapering to a point anteriorly. Roughly speaking, the shape resembles that of an Indian club. The broad posterior end bulges somewhat toward the posterior median septum, beyond the general surface, and a small interval is thus formed behind it, between the two posterior columns; this is filled up by connective tissue as in the previous sections. At this level the tract corresponds fairly closely to the "oval field" of Flechsig.

In the fourth lumbar segment (fig. 3) the tract is further diminished in size, and altered in position. It has passed backwards again, and forms an angular band outlining the postero-internal angle of the cord, one limb being placed along the periphery, the other along the posterior median septum for rather less than a fifth part of its extent. The tract is thus separated by a considerable interval from the cornu-commissural tract, which is well marked at this level.

In the segment above this, the third lumbar (fig. 2), the tract is still diminishing. It is no longer applied to the posterior median septum in any of its extent, but forms a very small, narrow band, of which one extremity touches the middle line, the other terminates gradually a short distance along the periphery. The tract has now become

scattered, with no very definite line of separation from the degenerated areas.

The final position taken up by the tract is seen at the level of the twelfth dorsal segment (fig. 1). Here the tract is represented by a small group of fibres, somewhat triangular in shape, placed at the periphery of the cord, about one-third of the distance from the middle line to the posterior roots. The fibres are few in number, but constitute a perfectly well defined tract seen in many successive sections. Lying anterior and external to it is an area containing more diffusely scattered healthy fibres.

The tract cannot be traced clearly higher than this level in cases of locomotor ataxia, but Hoche's case of degeneration after a lesion in the lowest cervical segment (14) has proved that it may originate as high as this. Above the eleventh dorsal segment, however, it appears to become diffused within the hinder part of the postero-external column, and is therefore not so easily followed as at lower levels. The evidence as regards its origin is thus still insufficient, and it can only be said at present that the tract originates in part in the higher segments of the cord, gains greatly in bulk about the level of the first sacral segment, and terminates in the lower sacral and the coccygeal region, by sending fibres forward along the posterior median septum into the grey matter near the central canal where they are lost amongst the other fibres in this part. The endogenous nature of many of its fibres is proved by the fact that in the case of ataxia described by the writer, there was a great accession to the number of its fibres at the first sacral segment, in spite of the almost complete degeneration of the posterior roots for some distance above and below this level.

It is very probable that at one part of its course, *i.e.*, in the lower lumbar segments, the tract corresponds to the "oval field" described by Flechsig, since at this level it occupies a small area close beside the posterior median septum and at some distance from the periphery, which when taken together with its fellow of the opposite side is oval in form. The discrepancies in the descriptions given

by the various authors as to the exact level at which this oval field appears are no doubt due in part to differences in the situation and nature of the lesion and in part to the different methods of staining employed. This also applies to some other slight differences in the descriptions.

The tract is undoubtedly mainly a descending one, but Déjérine and Spiller have concluded that it also contains some fibres which degenerate upwards after injury to the cauda equina. This is very probable, but there are, as yet, so few cases on record in which this tract has been accurately described, that on this and on many other points it is quite impossible as yet to form a definite conclusion.

As regards the cells from which the cornu-commissural and the septo-marginal tract are derived, it is not possible as yet to make a definite statement.

Cajal first showed that in the chick some cells of the posterior horn sent their axis-cylinder processes to the posterior columns, and Lenhossek has been able to demonstrate similar cells in the guinea-pig, etc., as well as in the human embryo. They are situated at the base of the horn and in the Rolandic substance. None of the cells of Clarke's column send their processes to the posterior column.

With regard to the function of these two tracts nothing definite can be stated further than that they are longitudinal and commissural in character. Their position strongly points to their being connected with the lower organic reflexes.

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Septo-marginal Tract.

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(13) Berdez. *Revue Médicale de la Suisse Romande*, May, 1892.
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(17) Souques and Marinesco. *Revue Médicale*, 1895.
(18) Dufour. *Arch. de Neurolog.*, 1896.
(19) Muir and Bruce. Brain, 1896.
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(21) Déjérine and Sottas. *Soc. de Biolog.*, 1895.
(22) Schlesinger. *Arbeit aus d. Inst. f. Anat. und Physiol. des Nervensystems*, Wiener Universität, 1895.
(23) Pineles. *Arb. aus d. Inst. f. Anat. u. Physiol. des Nervensystems au der Wiener Universität*, 1897.

DESCRIPTION OF PLATES.

The lettering is the same throughout.

c.c. = Cornu-commissural tract.
s.m. = Septo-marginal tract.

The figures are reproduced from drawings of the posterior columns made by Dr. Jessie M. MacGregor.

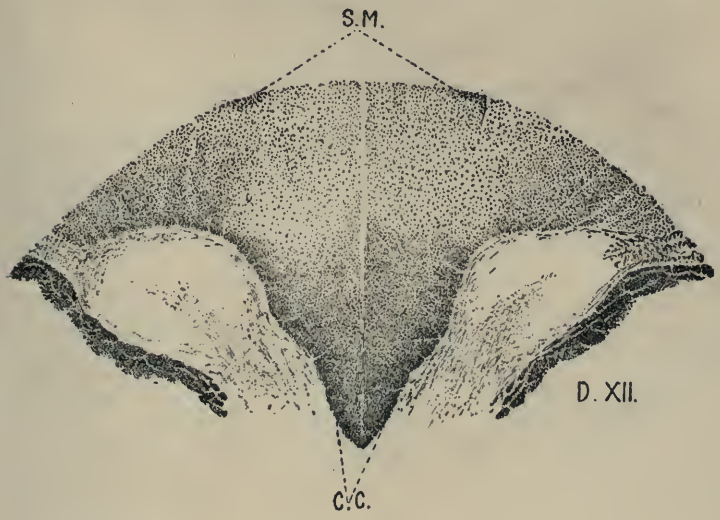


FIG. 1.

Level of twelfth dorsal segment.

FIG. 2.

Level of third lumbar segment.

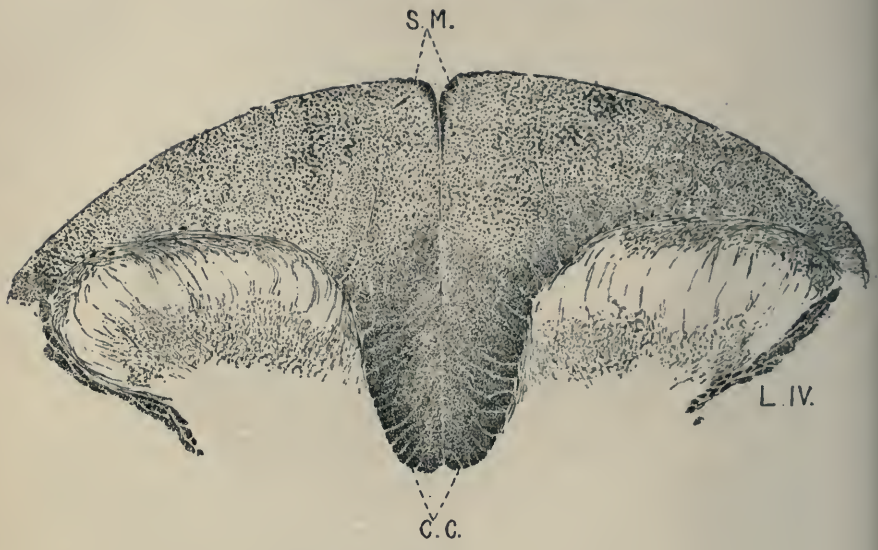


Fig. 3.
Level of fourth lumbar segment.

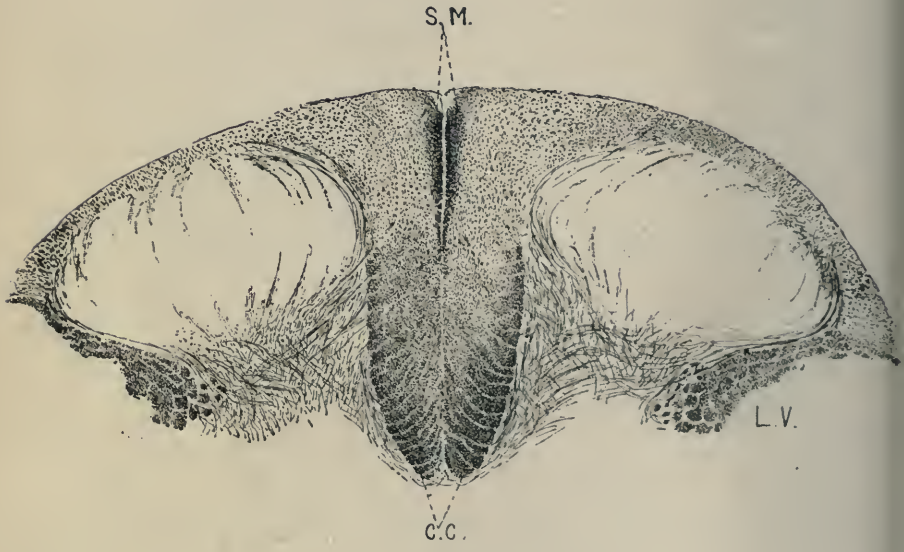


Fig. 4.
Level of fifth lumbar segment.

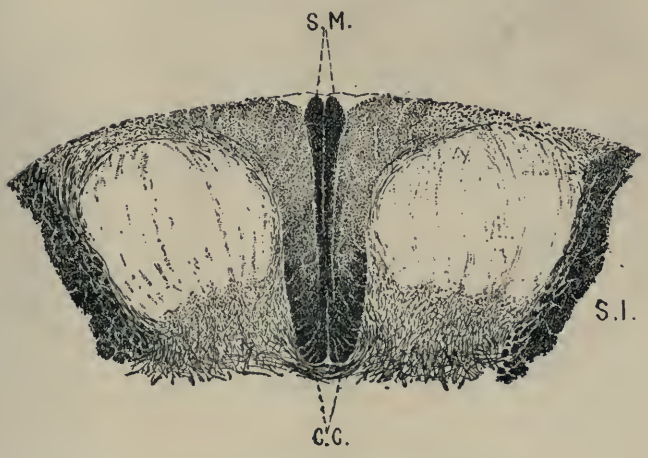


FIG. 5.
Level of first sacral segment.

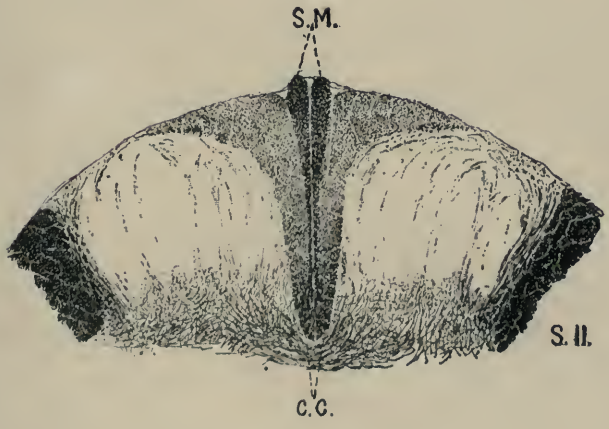


FIG. 6.
Level of second sacral segment.

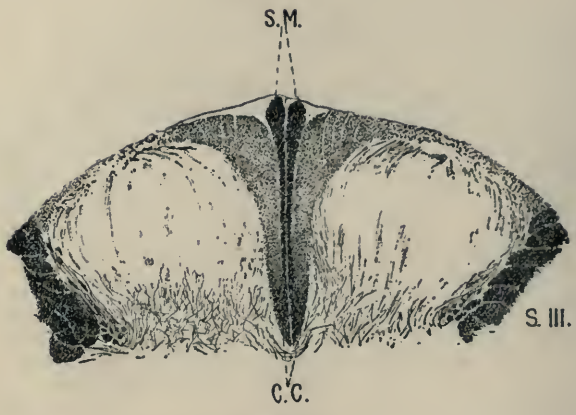


FIG. 7.

Level of third sacral segment.

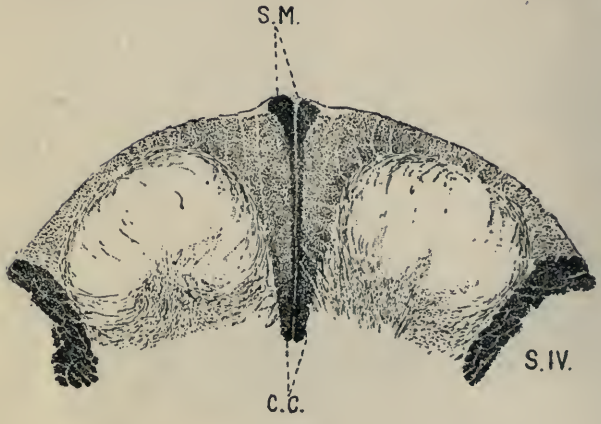


FIG. 8.

Level of fourth sacral segment.

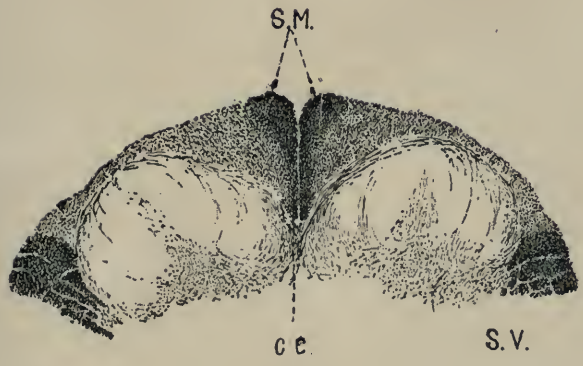


FIG. 9.

Level of fifth sacral segment.

THE MORBID ANATOMY OF A CASE OF HEREDITARY ATAXY.

(No. VI. of Dr. Sanger Brown's Series of Cases).

BY DR. ADOLF MEYER, WORCESTER, MASS.

WITH AN INTRODUCTION BY DR. SANGER BROWN.

By way of introduction to Dr. Adolf Meyer's Anatomical Report, and with the purpose of rendering the same more easily understood by those who have not read, or who do not distinctly remember, my clinical report of the series published in 1892, in Brain, and in the *North American Practitioner*, I now reproduce my original diagram, showing how the disease was transmitted, and the ages at which the respective subjects of it were attacked, with other data calculated to give a fairly good general idea of the nature of the symptoms, and a more particular clinical report of the case which furnished the material for Dr. Meyer's report.

For the purpose of giving a general idea of the symptoms in my series, perhaps I cannot do better than to quote from my original paper :—

"Taking these cases alone for a text, and assuming them to be cases of hereditary ataxy, the following diagnostic criteria might be fairly deduced :—

"Hereditary ataxy is a disease which may be traced through several—at least four—generations, increasing in extent and intensity as it descends, tending to occur earlier in life, and advance more rapidly. It usually attacks several members of the same family. It occurs most frequently between the ages of 16 and 35, but it may begin as early as 11 and as late as 45. It shows no marked preference for sex, but it descends through females four times as frequently

as through males. The influence of an exciting cause can rarely be demonstrated, but in some instances a fall or injury has appeared to determine the onset; and any cause like child-bearing or lactation, which very much depresses the vital forces, may produce a rapid advance of all the symptoms. There is always considerable incoördination of all the voluntary muscles, and a sluggishness of the movements, which they produce when the disease is well established. This is usually noticed first in the muscles of the legs, but in a few months or years extends to the arms, face, eyes, head, and organs of speech. Sometimes it occurs first in the upper extremities, and sometimes in the organs of speech.

"The ataxy is often extreme, and the gait devious, the patient deviating several feet on either side of the intended line of progression, before he loses the power of walking. The ataxy is not markedly increased by closure of the eyes. The sense of posture is perfect.

"Some weakness of the muscles of the legs, without atrophy, is frequently an advanced symptom, and occasionally there is permanent spastic contraction of the legs. In developed cases there are usually choreiform movements of the head, and often of the arms, accompanying all voluntary movements. These irregular movements occur in the hands, legs, or head whenever it is attempted to maintain either of these parts in a fixed position by a voluntary muscular effort.

"There is usually some degree of static ptosis, with overaction of the levator on looking upward. In rare cases there may be temporary diplopia in the early stages, due to weakness of the external rectus. There is no nystagmus of any kind.

"Atrophy of the optic nerve is a constant and early symptom, and usually progresses slowly with the other symptoms. Rarely it begins earlier in one eye than the other.

"The response of the iris to light and accommodation is sluggish, and diminishes with the advance of optic nerve atrophy. When this latter is complete, as may happen in

advanced cases, there may be complete internal and external ophthalmoplegia.

"There is always marked disturbance of the articulation, probably due to incoördination of the muscles concerned, for weakness cannot be demonstrated. In some cases there is a troublesome tendency to strangulation in swallowing liquids, due to their getting into the larynx, but otherwise swallowing is in no way difficult.

"Occasionally the sphincters are slightly, but positively, affected, this symptom only appearing in those cases where spontaneous pains in the legs co-existed, having some of the characteristics of those occurring in locomotor ataxy. Excepting the spontaneous pains already mentioned, there is no disturbance of sensibility. There are no vaso-motor or trophic symptoms, but there is a marked tendency to emaciation; there is no hypertrophy or valvular lesion of the heart.

"The knee-jerk is always exaggerated, and there is frequently ankle-clonus, and the cutaneous reflexes are also always exaggerated, but to a less degree. The exaggeration of the reflexes is an early symptom, and they often decline considerably when the disease is far advanced.

"There is never paralytic club-foot, nor any other deformity, excepting rarely permanent spastic contractions of the legs in advanced cases. In none of these cases have the patients ever suffered from rheumatism, so far as I can learn.

"I wish to repeat that the above summary of symptomatology of hereditary ataxy is only intended to apply to this particular series of cases; and I have only presented it in this way so that it might be the more easily compared with other series."

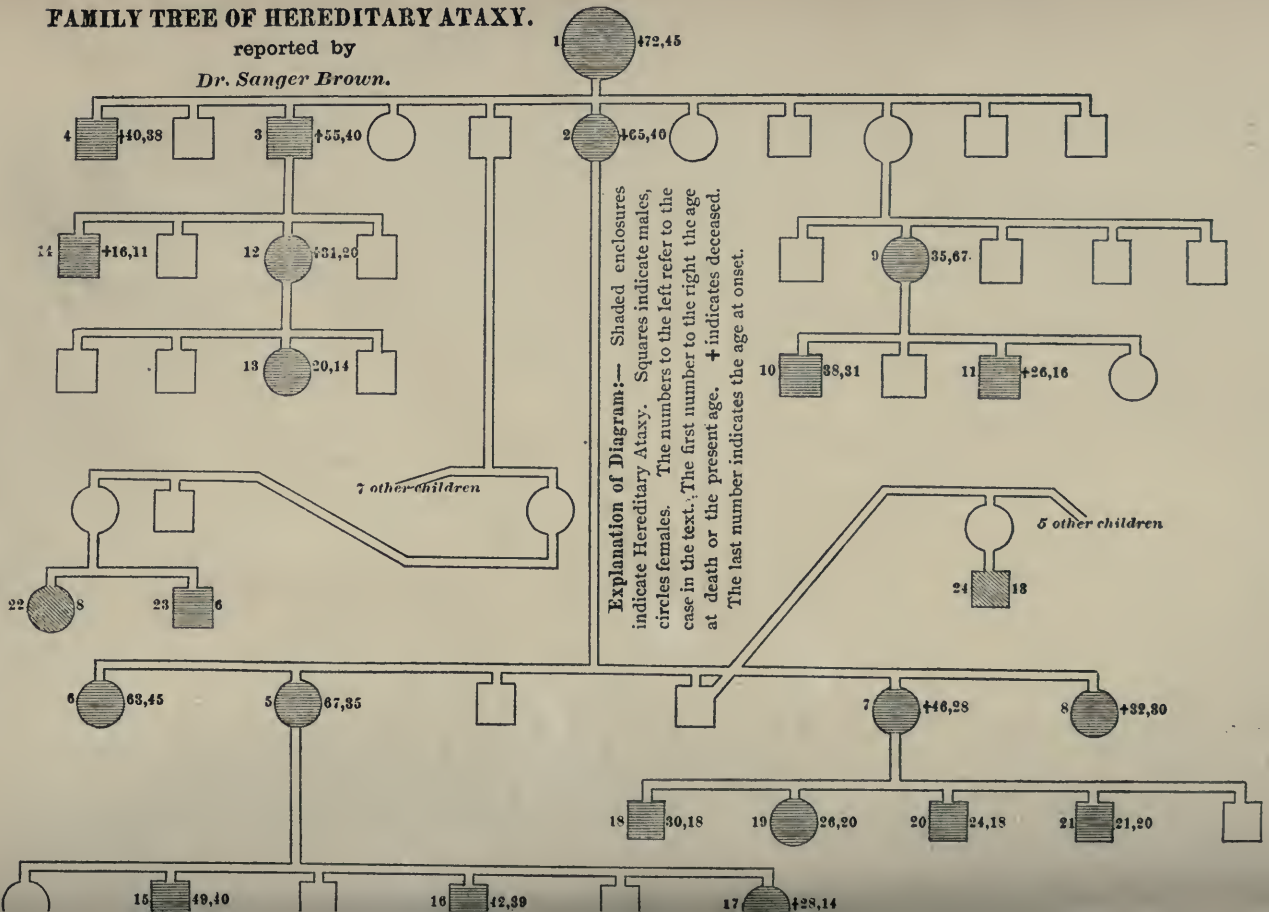
I shall again quote from my original paper the description contained of case vi., upon which Dr. Meyer's report is based.

"*Case VI.*—Female, age 63. First symptoms at 45, consisting of ataxy of legs. The course of the disease has been slow. She can still walk on an even surface without assistance, but has to go very slowly and sways about considerably, usually steady-

FAMILY TREE OF HEREDITARY ATAXY.

reported by

Dr. Sanger Brown.



Explanation of Diagram:— Shaded enclosures indicate Hereditary Ataxy. Squares indicate males, circles females. The numbers to the left refer to the case in the text. The first number to the right the age at death or the present age. † indicates deceased. The last number indicates the age at onset.

ing herself by holding on to furniture. The hands are certainly involved, but not greatly, as she can still do needlework and pour tea. Vision is considerably affected, but she can read coarse print in dim light. Cannot read in a bright light. Speech has become very slow, but is quite distinct. General health fairly good. Other symptoms negative. History given by a relative, himself affected, who has seen her almost daily for years."

This person died at the age of 67, of tubercular diarrhœa, from which she suffered for several months. About four months prior to her death, the neck of the femur was fractured by a fall, after which she was unable to leave her bed. Dr. R. L. James had been her medical attendant for over a year prior to her death, and noted very carefully the symptoms pertaining to her nervous disease, which he was particularly well qualified to do, inasmuch as he had in the past four years been medical attendant to, and seen almost daily, socially or professionally, two nephews and a niece of the patient who suffered from the same disease. He states that the knee-jerks were greatly exaggerated, but that there were no sensory symptoms of any kind; that there had been marked failure of vision two years at least before death, so that the patient was unable to read, but could see sufficiently well to play cards. She had lost the power of walking alone for at least two years before breaking her thigh, and for the last year she had been unable to do needlework or pour tea, though she could still manipulate cards fairly well. The facial expression for the last eight or ten years of life was highly characteristic, that is to say, the partial ptosis and relaxation of the facial muscles, being suggestive of somnolence when in repose, but when in animated conversation there was over-action of some of the muscles of expression and under-action of others, giving the face a very peculiar appearance. There was never any evidence of mental disorder.

The autopsy was performed under great difficulties, and the brain reached me in a rather lacerated condition, in Müller's fluid. Unfortunately there are no data on hand on the autopsy, the fresh appearance of the tissues, the weight of the brain, and especially the cerebellum. The

latter was centrally softened, as far as could be ascertained, owing to the slow action of Müller's fluid in the very warm weather. The spinal cord was well preserved. Of the brain, the medulla oblongata, cerebellar cortex, and the optic nerves and tracts were taken for examination. In absence of macroscopic description we proceed at once to the study of the sections.

Spinal Cord.—Sections from the lumbar, lower and upper thoracic and cervical cord were made. For the study of the corpora amylacea a short fixation in Müller's fluid, and subsequent alcohol hardening proved satisfactory for a stain with Bohmer's hæmatoxylin. The other specimens were completely hardened in Müller's fluid, and, with or without a total stain in a 1 per cent. carminate of sodium solution, dehydrated and embedded in the usual manner. The stain for the medullated fibres, was Wolter's modification of Weigert's method, with or without counter-stain by 0·5 per cent. acid fuchsine.

The cross-section of the cord is not thinner than might be expected in a woman of 67. The diameters are as follows:—

	Transverse.	Antero-posterior.
Cervical enlargement ...	10·8 mm.	8·5 mm.
Mid-dorsal region	7·0	7·0
Lumbar enlargement ...	9·5	8·0

The specimens stained with Böhmer's hæmatoxylin after a short fixation in Müller's fluid, show a remarkable number of corpora amylacea throughout the spinal cord, the bodies are distributed very much as Redlich represents them in his drawing.[1]

They are most numerous in the dorsal half of the cord, especially in the entrance zone of the posterior roots, where they are arranged in strings of several layers; further along the posterior septum, in the subpial neuroglia layer, along blood vessel septa, and all through the posterior columns. In the lateral columns they keep near the surface; they are

[1] E. Redlich, Die Amyloidkörperchen des Nervensystems. *Jahrbücher f. Psychiatrie.*, vol. x., 1892, p. 23.

relatively rare in the anterior columns and anterior horns and practically absent in the rest of the gray matter and in the ground bundles. In one posterior root zone over two hundred can be counted readily. They vary in size, and are easily distinguished from the much smaller nuclei. As Redlich, Koller and others have shown, they occur frequently in old people, but I have not been able to obtain such an excessive number in any of my cords, nor could any one show me similar specimens. We must say though that in the ordinary staining methods the granules do not present themselves as clearly as in a specimen which was not very long in Muller's fluid. The material of comparison is therefore not quite fair.

The neuroglia and the blood vessels were examined with a stain of Ehrlich's hæmatoxylin and subsequent counterstain with picric-acid fuchsine solution. This stain gives a very satisfactory bright colour to the mesoblastic connective tissue, while the neuroglia proper remains a duller red.

The pia is somewhat thickened, from 0·1—0·15 mm. over the lateral columns, but only about 0·025—0·04 over the dorsal columns, and 0·04—0·05 over the anterior. The blood vessels are normal in number, and there is very little if any hyaline degeneration of the muscularis of the larger ones.

The neuroglia is somewhat increased in the whole extent of the subpial neuroglia layer. The latter measures about 50μ over the dorsal columns, about 25μ over the ventral half, and over the dorsal part of the lateral columns and the posterior root zone it varies between 40 and 100μ. Fromann[1] says the diameter of the neuroglia margin varies between 0·01 and 0·06 mm., according to Goll from 0·02 to 0·03 mm. Kölliker[2] states that the layer varies from 0·022 to 0·045 mm. There is no doubt, therefore, that the increase is fairly well marked in our case. In the region of the pyramidal decussation this is quite plain, since the thickening is limited to the extent of the cerebellar tract.

[1] Fromann, Untersuchungen über die normale und pathologische Anatomie des Rückenmarkes. I. Theil, 1864, p. 28, quoted by Weigert, Beiträge zur Kenntniss der normalen menschlichen Neuroglia, Frankfurt, 1895, p. 146.

[2] Kölliker, Handbuch der Gewebelehre des Menschen, 6. Aufl. Vol. II. 1896, p. 151.

The neuroglia of the rest of the cord will be spoken of in the analysis of the various tracts.

Lumbar Cord.—The roots are quite normal. *The posterior columns* have a normal proportion of thick and thin fibres. It is hardly possible to detect anything abnormal; perhaps the neuroglia septa are slightly broad in the central portions of the posterior columns. But it is impossible to make out anything but a faint similarity with Flechsig's[1] or Marinesco's[2] figures since the roots, and especially the lateral part of the middle root zone are quite normal; only the mesial part of Flechsig's middle root zone may have a slight increase of neuroglia.

The amount of fibres and collaterals is quite normal in the gray matter; the motor cells, though pigmented, show mostly a well defined nucleus near the centre of the cell body.

The antero-lateral columns are densest close to the gray matter (ground bundles). The region of the pyramidal tracts shows very few thick fibres, but no striking increase of neuroglia.

In the *Thoracic* region nothing deserves special notice besides the condition of Clark's columns. The number of their cells is decidedly small. The cells that remain are partly normal, partly show a change which I noticed often in general paralysis and elsewhere, and which Marinesco[3] has lately described in cases of locomotor ataxia.

The normal cells, very few in number, have the nucleus round, and in the centre or near it. The granulations, of course, are not visible, since the hardening with Müller's fluid rarely preserves them. The larger number of the cells are more vesicular; the nucleus is pressed to the periphery and is crescent shaped. There are further a few cells which are quite shrunken and have no differentiation. There can

[1] Flechsig, Ist die Tabes dorsalis eine "System-Erkrankung." *Neurolog. Cbl.* Vol. IX. 1890, p. 73, fig. 6.

[2] Marinesco, "Lésions de cordons postérieurs d'origine exogéne." Part V. of "Babes' Atlas," plate VII.

[3] Sur une particularité de structure des cellules de la colonne de Clarke, et sur l'état de ces cellules dans le tabès simple et dans le tabès associé à l paralysie générale, par G. Marinesco. *Revue Neurologique*, 4e Année, Nov. 15, 1896, p. 633.

be no doubt that these findings stand in some connection with the condition of the cerebellar tract mentioned below.

The posterior and antero-lateral columns show no deep lesion. Their condition stands about midway between that found in the cervical and the lumbar regions with regard to the increase of neuroglia and the proportion of thick to thin fibres.

The Cervical Cord.—Here we find a number of very pronounced alterations.

The *posterior columns* show a field of degeneration along the middle of the septum; the superficial part of Goll's column is also affected, though less markedly, and, on either side, the transition area between Burdach's and Goll's tracts

is somewhat rarefied. In the normal cord most of the fibres in these regions are of moderate thickness, forming plain rings with the Weigert stain; thin fibres, with almost no lumen, are rare. In our sections many of the thick fibres are gone, and also the thin fibres are scarcer. The neuroglia is correspondingly increased, the blood vessels are a little wider and surrounded by thick neuroglia masses. The changes are somewhat more diffuse and less marked than in tabes.

The *lateral columns* of the *normal* cord show a layer of thick fibres plainly limited all along the surface of the pyramidal tract, but less distinctly outlined in the region of Gowers' antero-lateral tract. The distinction is formed by the fact that the fibres of the direct cerebellar tract have very few collaterals, while these are abundant in the pyramidal area and also in the ground-bundles and a little less in the antero-lateral columns. The collaterals show with the Weigert stain under a low power (Reichert or Leitz 3) as points without lumen; under a higher power (Reichert 7) they show a small lumen, except the smaller ones, which are point shaped.

In our case, we notice at once the diminution in size of the direct cerebellar tracts. Only relatively few large fibres are left, and they are separated by neuroglia. In the normal cord, this area, so free from neuroglia and collaterals, is about 0·4—0·5 mm. broad; in our sections, the atrophic area measures 0·15 mm. at the most on the side where the proliferation of neuroglia is less marked; on the other side it is of normal breadth (0·4—0·5 mm.), but containing much interstitial neuroglia, fewer fibres, and broader blood vessels. Farther ventrally beyond about the middle of the lateral columns the area loses its demarcation rather suddenly, and all we can see is a general deficiency of thick fibres along the surface. On the one side the bundle is rarefied *en bloc* for 2·2 mm. from the posterior root and superficially only with a little less demarcation for further 2·4 mm. (fig. 1).

The fibres which remain are very large, averaging a diameter of 10·15. Throughout the rest of the anterior and lateral columns there are only few fibres of this size in our

specimens, while they are fairly numerous in the normal throughout the pyramidal tracts and the antero-lateral tract area. The direct pyramidal tracts show a slight increase of neuroglia, the lateral pyramids lesser. It is hardly possible to make out Gowers' tract; all we can say is that the ground-bundles are denser and richer in collaterals than the more superficial layers.

The gray matter shows no evident alteration.

Transition into the Medulla.—All we have to note is the extension of the changes described; hardly any change in the pyramids, slight degeneration in the posterior columns, and especially degeneration of the direct cerebellar tract on both sides, not complete, but leaving a triangular area with scanty large fibres, broad neuroglia septa, and especially a well-limited thickening of the marginal neuroglia layer. The field is decidedly smaller than normal (it is easy to see it in the normal carmine specimens and also in Weigert sections). The thick fibres of the antero-lateral tract (ventral cerebellar) are also rare, but not kept apart by neuroglia.

Medulla Oblongata.—The medulla had been torn off from the pons at the autopsy and was not satisfactory for a careful examination of the topography. All we can say from the sections is that the area of the direct cerebellar tract continues as described and then disappears in the restiform body, where at best a slight rarefaction of fibres can be made out. The ventral cerebellar tract has few large fibres and is somewhat pale. The olives have a normal amount of fibres and cells. The pyramids are slightly less dense than normal.

Cerebellum.—Only pieces of the cortex of the hemispheres are examined. The folia are hardly atrophic as a whole. Nothing can be made out but, perhaps, a slight reduction of then umber of Purkinje cells in a few parts. This also occurs in normal specimens, although perhaps less than here. Other folia have a very large number of perfect Purkinje cells.

The *optic nerves* are free from degeneration, and also from neuroglia-overgrowth.

In the monograph of Londe,[1] the autopsies of Fraser,

[1] Londe, "Maladies familiales du système nerveux. Hérédo-ataxie cérébelleuse." Paris, 1895.—*See* BRAIN, 1895, p. 421.

Nonne and Menzel are discussed. Our case seems to be the first addition since Marie established the clinical picture of cerebellar heredo-ataxia. On account of the defective condition in which the brain reached the pathologist, several points cannot be as clearly decided as would be desirable; but the following statements are well founded.

1. There is no circumscribed cerebellar lesion, nor does the cortex show a marked decrease of the number of the Purkinje cells.

2. Parts of the spinal cord and medulla which are known to have relations with the cerebellum were found affected.

3. The spinal cord as a whole shows increase of the superficial neuroglia and a remarkably large number of corpora amylacea, similar to what is seen in very old people, in paralysis agitans, &c.

It would not be fair to build on this one observation a dogmatic theory of the disease of the family. We may, however, safely say this:

The separation of a *type cérébellaire* from the general picture of hereditary ataxia is clinically justified, but anatomically to less extent than Marie seemed to expect. In his case Nonne saw a simple hypoplasia of cerebellum and spinal cord and a peculiar preponderance of thin fibres in the anterior roots. Fraser found atrophy of the cerebellum, but no lesion of the cord (his case dates from a time when the Weigert hæmatoxylin stain did not exist). Menzel, however, had in his case even more varied lesions than we found: atrophy of the cerebellum with notable disappearance of Purkinje cells and changes of the dentate nucleus, changes in the spinal ganglia and posterior roots; degeneration of Goll's and Burdach's tracts, the pyramidal tracts and cells of Clarke's column; changes in the nuclei of the lateral and posterior columns, of several motor nuclei of the bulb, of the gray matter in the pons, the olives, corpus Luysii, and black substance of Sömmering.

The safest explanation is, that in our case we met with a diffuse deterioration of the afferent cerebellar system, with special involvement of the direct cerebellar tracts. Moreover, there is an affection of the peripheral sensory elements

in their course within the neuraxis, but apparently only of those collaterals which connect the lower level with the higher (cerebral and cerebellar) apparatus. We could not otherwise understand the persistence of an exaggerated knee-jerk in our case, and the absence of degeneration of the dorsal columns in the lumbar region; the reflex collaterals are intact; only the cervical cord shows plainly a degeneration of Goll's column. In a similar way we have found in general paralytics a degeneration of the crossed pyramidal tracts of the lower segments of the cord, where the crus cerebi and the oblongata did not manifest a degeneration of the pyramid. Whether the great number of amyloid bodies should be interpreted as an excessively senile phenomenon cannot be decided, since the patient died rather old, and we have not enough control specimens; it is, however, fair to admit, that even for a woman of 67, the number of these bodies and the general increase of neuroglia was at least indicative of marked retrogressive changes. The cerebellum manifests a slight decrease of Purkinje cells. The optic nerves are intact. There is no marked degeneration of the pyramids, but a slight diminution of the thick fibres.

My general attitude in explaining diffuse nervous affections has been put forth in a paper on Landry's paralysis, published with Dr. Diller, in the *American Journal of Medical Sciences*, April, 1895; this view is already taken for heredo-ataxia both by Nonne[1] and by Edinger.[2] The latter says: "I am inclined to accept Londe's opinion, according to which one would do better to keep the two diseases together under the name of hereditary ataxia; the one form Friedreich's disease proper, should be diagnosticated where the disorder begins with spinal symptoms, while the name cerebellar heredo-ataxia should be reserved to the cases which start with cerebellar symptoms. Londe shows beautifully how Marie's type gradually associates itself with spinal symptoms, and Friedreich's with cerebellar ones. Moreover, cases are on record where both were attacked simultaneously. Schultze arrived apparently at similar conclusions. On the ground of the 'Ersatz

theorie,' I am of opinion that according to the varying localisation of the congenital weakness function will create various pictures."

That the character of the lesion is not identical in appearance with the typical gliosis of Friedreich's disease as described by Déjérine, must have its reasons in the difference in the age of onset, and the extent of the pathological process.

[1] Ueber einen in congenitaler beziehungsweise acquirirter Coordinationsstörung sich kennzeichnenden Symptomen-complex." *Arch. f. Pysch.*, vol. xxvii., p. 497.

[2] (Art. "Friedreichsche Krankheit." "Real-Encylop. d. ges. Heilkunde," 3 Aufl., 1895, vol. vii., p. 117).

"SURVIVAL MOVEMENTS OF HUMAN INFANCY."

Being the substance of an address given before the Manchester Branch of the British Association for Child Study, March 2, 1897.

BY ALFRED A. MUMFORD, M.D.

When we watch closely the limb-movements of human infancy and early childhood, we cannot but be struck with their peculiar purposeless character, and notice how widely they differ not only in form but even in rhythm from the voluntary movements of later date.

These early limb-movements as we see them immediately after birth, are presumably similar to those occurring in utero, though from the nature of the surroundings they necessarily present a greater range.

They are generally known as "Spontaneous" or "Involuntary" movements, sometimes as "Stretching movements." They steadily diminish in number and in importance till the end of the fourth or sixth month of independent life, by which time many have entirely disappeared, while others survive but in a modified form. In fact, on careful examination, a definite selective process is found to occur among them, by which some become utilized for special purposes, serving mainly for the expression of the various feelings and emotions. These are therefore permanently preserved. Others gradually diminish and tend to disappear, being replaced by the gradually evolving voluntary movements, which appear in successive stages or batches of increasing complexity throughout the early years of childhood. For the early spontaneous movements the writer suggests the term "Survival Movements," and besides describing their nature, proposes to attempt to give

some explanation of their origin. The full description of the definite stages in which the *voluntary* movements appear, and how they arise from and supplant these early survival movements must be left to another paper.

A careful study of these early infantile movements shows that though they are aimless as far as the individual infant performing them is concerned, they are not necessarily meaningless as regards the development of the race of which the individual infant is but an off-shoot. It is quite possible, indeed we might say probable, that they are vestiges of functions of the limbs which were of prime importance to the members of the race at another and early period of its growth, but began to lose their prime importance, and therefore their full development, when the forelimb gradually acquired other and higher functions. They remain, I believe, as true survival movements from earlier stages of existence, and thus serve to show the order in which the evolution of the human forelimb took place; just as many other bodily structures remain, such as the pineal gland and the vermiform appendix, which denote the existence of functions that are not now in activity.

Preyer,[1] who seems to have made the most thorough investigations into early infantile movements of all classes, divides them into four groups:—

(i.) Impulsive or spontaneous movements:—Comprising the stretching and yawning movements, noticeable as the infant wakes from sleep, also limb movements in the bath, protrusion of the lips in pouting. These are supposed to be due to spontaneous discharges of energy in the motor cells of the nervous system, and not to be due to the action of any external stimulus. As there be no evidence of a *mind* at this stage, they receive the name of "spontaneous" or "impulsive." It is in this class that most of the survival movements occur.

(ii.) Reflex movements:—Occurring as a result of some external or internal impression, such as stimulus of touch, sight, hearing, or visceral change. They appear at, or very

[1] Preyer, "The Mind of the Child": English Translation, "The Senses and the Will." Appleton, New York, p. 195, et. seq.

soon after, birth, and include the movements of breathing, swallowing, reflex movements of the eye in response to light, spreading of the toes in response to stroking or warming the soles of the feet.

(iii.) Instinctive movements:—These also require the stimulus of sense-impression to arouse them, but according to Preyer, differ from the purely reflex in being always preceded and accompanied by a distinct emotion. They are, therefore, accompanied by changes on the psychical plane. They all have a definite aim. He cites as instances:—pecking movements, by which a chick breaks the shell or seizes a fly, running or walking, biting, chewing, and so on; in fact any purposive movement which the individual does not voluntarily acquire for himself, but seems to inherit from his ancestors. Some of them appear at birth; others, such as walking, only at a later stage. We speak of teaching a child to walk as if walking were a purely voluntary and intelligent act, learned by imitation, it is true; but in fact, as we see below, we really here draw out by touch stimulation an instinctive movement. The rhythmical alternate movements of the feet before walking have been noted by Baldwin during the ninth month, *i.e.*, before the child can really balance itself on its feet. By holding a young baby on the palm of my hand, stomach downwards, and supporting the head with the other hand, and allowing the feet and hands to touch the counterpane of the bed, I have elicited alternate movements both in the feet and hands as early as the third day after birth (see fig. 4).

(iv.) Voluntary and Intelligent movements:—These are not inherited; each individual acquires them for himself. They are preceded by, or caused by, an "idea." They therefore not only have a definite purpose, but are consciously and intentionally performed. In the process of building up and elaborating of these intelligent movements, some of the most interesting problems in Mental Philosophy are involved. They include the origin of the will and of the intellect. Preyer, Baldwin,[1] and others have shown

[1] Baldwin, "Mental Development in the Child and in the Race," p. 81.

how important a part imitation and suggestion play in their growth. How many nerve processes occur in them is well shown by Preyer where he says: "In order to imitate, one must first perceive through the senses; secondly, have an idea of what is perceived; thirdly, execute a movement corresponding to the idea." He gives as instances a child making faces, such as pouting at a stranger or a nurse; holding out the arms to show he wants to be lifted up; speaking definite words. He considers walking to be simply an instinctive movement, not an intellectual or ideational one, because a child does not see others walking and then determines to imitate them, but because the parent or other instructor holds the child up by the arms or body, and makes its feet touch the floor, and thus sets in action the instinctive reflex of this mode of progression.

Such as classification of early infantile movements, however helpful to the psychologist, is unsatisfactory to the biologist. By bringing in the terms "consciousness," "intention" and "idea," even when we translate them into terms of their physiological correlatives of higher and lower cerebral and spinal changes, we get no nearer understanding their origin; while the true meaning of the intermediate stages through which the perfected forms of movement have passed, and by which alone they have been rendered possible, is entirely lost. In harmony with the method of enquiry that evolution has introduced, we need to study these movements in their gradual growth to perfection, not only in their ultimate forms.

Evolution of the Human Limbs.

Leaving then, for a while, the study of infantile movements along the lines of their mental accompaniment, *e.g.*, whether they are conscious, volitional, or reflex, let us study them along the lines of their possible evolution. The structure of the limbs in which the movements occur will be our first guide. A study of the limbs of animals shows that their structure varies according to their habits and instincts, and so directly or indirectly with the food the

animal eats or the means by which it is acquired.[1] In this way limbs have been modified according to their use for *locomotion*, in water, land or air; *prehension*, tree and rock climbing; *destruction of prey*, carnivorous animals; or *manipulation*.

In the following paper I have confined myself mainly to the movements of the upper limbs, though movements of head and neck and legs present equally interesting problems.

The Structural Plan of the Human Hand.

In studying the general plan and structure of the human hand and comparing it with that of other animals we notice that while it is most highly developed in function, it is also in some respects the least modified in the general plan of its skeleton. In shape and bones it is more like the primitive amphibian paddle[2] than is the limb of any other mammal, even including the monkeys, and this fact alone debars us from looking upon the other existing mammals for traces of direct human ancestry.

It is also fully established that the primitive form of organic life, including that of man, was aquatic, and the hand must have existed as a paddle, at the time that gillslits in the neck were in functional activity. Von Bardeleben[3] shows the human hand possesses some very interesting traces of such formation. Finally, the method of development of the hand seems to indicate that the palm is originally the more important, and the independence of the fingers is acquired at a later stage of existence.

The more tempting and more varied land dietary, with its greater stored-up energy, must have had some share in alluring our ancestors from the sea and marshes, and in turn have helped in the transition from cold to warm-blooded

[1] For a full discussion of this question as it affects man, see Munro: "Prehistoric Problems," chapter ii., "On the Influence of the Erect Position," being the Presidential Address in Anthropology at the British Association at Nottingham, 1893.

[2] Romanes, "Darwin, and after Darwin," vol. i., pp. 54, 183.

[3] "Mammalian Hand and Foot," *Proc. Zoolog. Soc.*, London, April, 1894.

life. No doubt, too, the greater stored-up energy in the food helped in the capacity for further variation in structure and growth, and the higher land animals have been divided by Professor Owen into three classes of vegetable feeders: (1) root eaters, (2) plant eaters, (3) fruit eaters; and two classes of carnivorous animals: (1) insect eaters, and (2) flesh eaters. The habit of obtaining and devouring each different kind of food has resulted in the formation and fixation of a specially adapted limb for each purpose.

Darwin says:—"Our progenitors, no doubt, were arboreal in their habits and frequented some warm forest-clad land.[1] . . At a still earlier period the progenitors of man must have been aquatic in their habits, for morphology plainly tells us that our lungs consist of a modified swim-bladder which once served as a float. The clefts on the neck in the embryo show where the branchiæ once existed. . . . We apparently still retain traces of our primordial birthplace, a shore washed by the tides."

Analysis of Early Infantile Movements.

Let us now turn to the subject with which we are immediately concerned,—the study of the early movements of the human infant—especially those of the hand and arm. We shall see how readily the spontaneous movements group themselves in terms of their value in the racial struggle for existence and survival of the fittest. Limb movements to be of value must be appropriate; (i.) to simple progression, either aquatic, terrestrial, or aerial; (ii.) to prehension, or arboreal existence; (iii.) to manipulation, including the destruction and breaking up of food, or the search for it by digital investigation into nooks and crannies; and finally, to the making and using of tools or weapons.

Limb movements having been once acquired by the race, may persist in the individual in their fully developed form, or may remain only as partially developed rudiments or vestiges.

[1] "Descent of Man," pp. 160, 161.

"Survival Movements" of Locomotion in marshy or aquatic surroundings.

FIG. 1.

Showing primary position of hands and digits. Second month of intra-uterine life. Also position of foot. From His.

FIG. 4.

Drawn from life, eighth day. Showing position of hands and digits, and toes when child suspended his own weight. Left thumb folded forward, but not in use.

If we watch carefully the position of the limbs of an infant either soon after birth or almost any time during the first three or four months, we find them sometimes in the primitive development position, viz.:—folded across the chest, thumb towards the head and with the palm towards the thorax; but much more often the palm is away from the chest-wall, and is directed anteriorly by means of extreme pronation, the dorsum of the hand often lying on or near the shoulder, sometimes an inch or two outside. As the child wakes up the elbows begin to open out and the palm is pushed outwards in a way that would be useful in locomotion, especially in a fluid or semi-fluid medium. In fact, it

FIG. 5.

Drawn from life, eighth day, to show position in which the child was held to draw out reflex movements of progression. Child supported body weight by legs, which showed marked alternate movements. Arms also showed alternate movements, though to a less extent.

is the movement of a paddle. Slow, rhythmical movements of flexion and extension of the fingers occur, which instead of possessing the quick, incisive character of voluntary movements partake of the sluggish rhythm so familiar to the visitor to the tanks of an aquarium. They often occur in a series of three at a time during a quarter of a minute, then follows a pause during which there is apparently an accumulation of energy in the nerve cells. Then another series of spontaneous discharges takes place, to be in its turn followed by another pause. It was this peculiar sluggish rhythm, so unlike that of any other limb movements that first suggested to me that they might be survival movements of aquatic or amphibian life.

The resemblance becomes even greater when we further examine these movements. As the fingers become fully extended, a rotatory movement of the wrist—hyper-pronation—occurs, by which the ulnar border and little finger knuckle are lifted upwards as in raising a cup to the mouth, though the elbow-joint becomes extended instead of being flexed, the hand is thus moved backwards and outwards as a paddle would be used in swimming. The whole of this movement persists throughout life associated with the act of yawning, which act is thus found to be one of the most fundamental limb movements in the human being. It is also frequently met with in the blind where it appropriately serves the sense of touch.

It may be urged that such a movement or series of movements is not necessarily suggestive of aquatic progression, but may be the remains of a four-footed land progression. That this is not the whole case is proved, I think, by the fact that the movements in the two limbs are not always alternate but often synchronous, though alternative movements are clearly noticed when the child is held face downwards and the palms and soles touch something rough. These movements, moreover, do not take place in an antero-posterior plane, but in an oblique plane half-way between antero-posterior and transverse. Other vestigial movements which are prehensile and truly terrestrial occur in an antero-posterior plane. The movement in the oblique plane can be well studied in water lizards, where the use of the eversion of the ulnar border of the manus is clearly manifest. Lastly, the rhythm is the rhythm of cold-blooded aquatic or amphibian progression, and though perhaps suitable for a purely graminivorous or root-eating animal, is not suitable for one that has to pursue some of its food with any degree of rapidity.

In the discussion of the psychological nature of these early spontaneous movements, Bain[1] showed that they did not depend for their immediate causation upon any reflex stimulation from the outside, nor upon any internal emotion,

[1] "Senses and Intellect," p. 67.

but rather upon an abundance and exuberance of nervo-muscular energy; he suggested that in the greater or less endowment of such spontaneous energy, and in the greater or less sensitiveness to external impression, we have already in infancy some key to the fundamental difference in temperament exhibited by the man of action and the man of feeling.

Other impulsive movements, such as "rolling of the eyes," "twisting of the neck," &c., occur. Perhaps the eversion in some cases of talipes valgus is an instance of the loss of a (racially) late acquired arching and inversion of the foot in infancy, which Huxley considers a vestige of arboreal life. If the above explanation of these early movements, as vestiges of a previous stage is untrue, I do not know how else to explain them. They serve no definite purpose, unless we admit the teleological one, that they exist for the purpose of exercising certain groups of muscles and are thus pleasing to the child, though then we leave unexplained the fact that other series of movements equally simple are not thus exercised at this stage, such as movements of supination. It can hardly be that they are meaningless as well as purposeless. It seems more natural to look upon them as physiological or psychological vestiges whose fuller study will open up some hitherto closed chapters of man's history, and while they excite our curiosity, yet also kindle our wonder at the distance man has travelled in his upward journey.

Reflex and Instinctive Movements.

Let us now consider the movements which Preyer embraces under the terms "Reflex" and "Instinctive." These are, according to him, started by the stimulation of some sense organ from without, and do not arise from any spontaneous discharge of nerve cells from within the organism. They differ from the previous ones also in having a definite aim, while they resemble them in depending for their first manifestation upon an inherited nerve organisation or nexus. The difference between Reflex and

Instinctive movements according to Preyer is that reflex ones are not preceded by consciousness, while the latter are preceded by consciousness in the form of an emotion, though this does not amount to a definite idea. Such a division is not very satisfactory and only applies somewhat loosely, and accordingly the two are in this paper considered together.

The earliest reflex movements that occur naturally after birth are apparently those of respiration. These seem to be set in motion by the stimulus of external cold acting on the skin and mucous membrane of the nose—sneezing. The sense of touch seems the most active and most early in

FIG. 3.

Drawn from life, on eighth day, to show several positions of hand at rest, with eversion of ulnar border and little finger; the last is a position useless for pure terrestrial locomotion. It is an aquatic "displacement position," though adaptable to arboreal life.

calling out reflex and instinctive movements. Then smell and taste, finally hearing and sight. With reference to the movements of the limbs, the sense of touch seems to be the sole avenue by which reflex movements are called into action. As before stated reflex movements of locomotion of both fore and hind limbs can be very early called into action by allowing the palms and the soles to be brought into contact with a rough blanket. These seem to be special applications of the stretching movements, and can only be studied when the child is wide awake. The stretching

movements are best noticed during gradual emergence from sleep.

Survival Movements of Prehension.

On touching or tickling the palm of the hand or sole of the foot, reflex action causes the fingers or toes to contract and to seize the object. If the right moment is chosen and especially if the rest of the body is not securely held, the clutching sometimes becomes so strong that the child can be lifted entirely from its bath or from the ground without any other hold. The grip seems even stronger on the ulnar side than on the radial border. How does such a movement fit in with our Developmental classification of the functions of

FIG. 2.

Further figures to show position of hand at rest in a child two months old. Power of supination very slight.

a limb into locomotion of varying kinds, viz. :—aquatic and terrestrial; prehension (arboreal locomotion), manipulation (movements involving supination and independence of the thumb, capacity for destruction and division of food, &c.)? They at once fall into the category of prehensile movements, and Dr. Louis Robinson in an article on "Darwinism in the Nursery," in the *Nineteenth Century* for November, 1891,[1] drew some very interesting parallels, and showed that by watching some sixty babies he found out that from the very beginning of their separate existence they possessed sufficient

[1] See also Dr. Robinson's paper on "Infantile Atavism," B.M.J., Dec. 5, 1891, p. 1226.

grip and muscular power to sustain their own weight on a horizontal bar or finger, and even delighted in the process. I have myself frequently tried this experiment, but have had only varying success. I believe, however, that he is right in ascribing such early movements to inherited prehensile power, and that further investigation along these lines will be fruitful of other illustrations of the law of recapitulation.

In November, 1894, the *Nineteenth Century* published an article by S. S. Buckman on "Babies and Monkeys," in which further comparisons were instituted, from which the following extract is taken:—

"In the method of using its hands the baby shows to the full its descent from arboreal ancestors. When it wishes to take hold of anything, like a glass or a flower pot, it does not, like an adult, put the hand round it, or even put a thumb inside to use as a lever. On the contrary, it places all the fingers inside, makes no use of the thumb, and clasps the rim of the flower pot between the fingers and the palm of the hand. This is exactly the action which would be acquired from arboreal ancestors, in going from bough to bough they would take their hands, palm first, and would strike from above downwards, grasping the bough with the fingers. Such is the action of an infant picking up a cup. So little use have some monkeys made of the thumb, that abortion has resulted, and in the most arboreal species of monkeys known, the fingers have grown together, because the whole hand was used merely as a grasping hook."

Seizing or Clutching Movements, with a Psychical Accompaniment of Emotion or Idea.

Preyer discusses the Seizing movements under the class of Instinctive Movements, and says that the first noticed grasping at objects with manifest desire to have them was seen by Sigismund in a boy of nineteen weeks, and by himself in a girl in eighteenth week, and in a boy in seventeenth week. The factors in such an action are evidently very much more complicated than in the mere clutching of a stick. In the observations on my own child I paid more

attention to the method of grasping, and the date of the use of the thumb, and I extract the following from my notebook :—

Development of Independent Action of Thumb and power of opposing it to rest of the hand.

Eleventh week.—Baby distinctly uses his hands as a grasping organ to pull the feeding bottle towards him when very hungry, mainly by pressing ulnar border of palm on it and drawing towards him.

Twelfth week.—Does not use his thumb properly for grasping; when he tries to bring the bottle towards him he tucks the thumb out of the way. For the past fortnight, however, he has been beginning to grasp with his fingers, and will pull his mother's hair or dress if his fingers come in contact (see fig. 6, and refer to footnote).[1]

Fourteenth week.—Now moves his hand to press the teat into his mouth, and grasps things with greater vigour. No power of opposing the thumb to the rest of the hand, though it is noticed that the thumb does not get in the way so much as before. The thumb is also occasionally kept outside the fist.

Sixteenth week.—Has been using the thumb more and more since last note now, and never shuts it inside his fist, but always outside. He grasps objects intentionally now, but only for a few seconds. He does not hold out his hand to reach a thing but lets his finger travel over them as they lie on his lap; only after feeling them does he grip them.

Nineteenth week.—Played with a toy for the first time.

Twenty-fifth week.—Spontaneous purposeless movements of fingers now ceased. Movements of putting up and down and of flexion and extension of forearm.

Twenty-eighth week.—Grasping movements much more perfect, though he still does not pick up things between the tip of his thumb and finger. He occasionally grasps a thing

[1] On a Possible Obsolete Function of the Axillary and Pubic Hair Tufts, *Journal of Anatomy and Phys.*, 1892, p. 254.

between the bent last joint of thumb and the fingers. In stretching out for a thing he often grips it between second and third finger, this especially if the fingers so slip round

FIG. 6.

Drawn from life, four months after birth, to show prehensile use of fingers, before the movements of the thumb are fully called out. The ball of the thumb in centre figure is used as a support. Inter-digital clutching movements of right hand figure illustrates a method of grasping objects of support which would be called into play in Dr. L. Robinson's theory of the use of axillary and pubic hair tufts (see reference in paper).

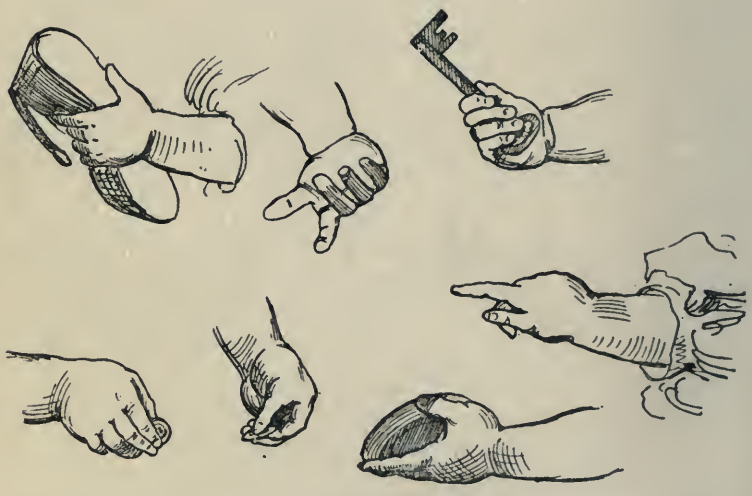


FIG. 7.

Eleven months. To show full development of powers of the thumb, of supination, and also the searching movements of index finger—digital investigation.

the object. In grasping small flat objects, such as a domino on the table, he grips between the tips of the fingers and the base of the palm, often bringing the ball of the thumb to

steady from the side. He now tries to pull things to pieces, such as a piece of paper (first appearance of purposive supination). The fingers are becoming more and more independent and have considerable power. Toes very rarely used for grasping, though they have not entirely lost that power. The thumb has been growing in importance and in variety of action very rapidly during the last week.

The reason I have quoted these notes in such full detail is to show how deeply rooted these inherited movements are, and by how slow a process those that do not survive disappear.

It is often six months or more before the more elaborate movements of manipulation possessed by the human being are rendered possible by the development of the capabilities of the thumb, and the power of purposive supination.

Survival Movements of Digital Investigation.

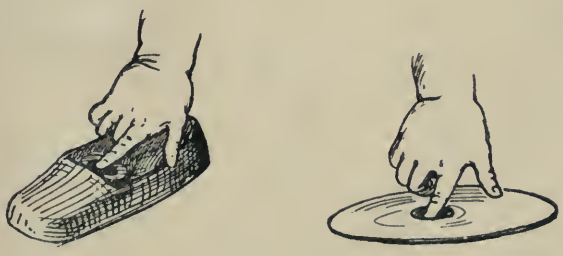


FIG. 8.

Drawn from a child twenty months old. To show full development of searching movements of index finger, a key in a shoe, and the hole in the head of a stethoscope.

Another survival movement presents itself prominently before our notice, namely, the way in which a child points and pokes at any object with its index finger. My wife and I noticed this digital exploration vaguely manifest about the seventh week, and it still persisted vigorously at the sixteenth month, though in a highly special and voluntary form. It is

the method by which not only the form, but the resistance, the consistency, and texture of a thing are determined. The movement may have originated during arboreal life for burrowing into nooks and crannies, and to have owed its survival to the great knowledge it gives about food through the sense of touch. It has also very conveniently adapted itself to gesture language in pointing. A still more ultimate analysis may show it to be a specialised form of the stretching movements of aquatic life. In watching its early appearance, my own child at first indiscriminately used the second, third, or even fourth digit occasionally for this purpose, but finally rejected them all in favour of the index digit.

Lastly, I have frequently watched a child stroking the floor, the table, or other surface, and asked myself whether this originated as a movement of progression. In the movement of crawling the palms are flat downwards, fingers directly forwards. In this stroking movement the hands are pushed outwards away from the middle line, not inwards towards the centre, though when voluntary movements are performed there may be a to-and-fro movement. This may or may not be a survival from movement of aquatic locomotion, but its very early appearance suggests it to belong to the inherited movements and not to those voluntarily acquired.

In conclusion, I should like to urge the importance of further study of these early infantile movements, and to plead for their inclusion in any complete study of the development of the nervous system. Child Study is very young amongst us, but such a line of enquiry as I have suggested offers fruitful reward. Whatever the rightful explanation, there is certainly a gradual selection and a gradual supersedence of involuntary movements by voluntary ones, and these appear in a definite order, each successive batch being more complicated than its predecessor. The independent use of the thumb and the power of supination seem altogether voluntary and are of late appearance, and deserve separate consideration under the group of "manipulation." Medicine teaches us how these late arrivals are early departures, not only does the

power of supination and of opposing the thumb become early lost in various poisonings; but even the gait of the infant acquiring the art of walking reminds us irresistibly of that of the ataxic drunkard, whose alcohol has made him temporarily lose the complete co-ordination necessary for normal progression. While, however, a study of the processes of degradation and decay is apt to leave a feeling of depression and even despair, the study of the opening powers of the infantile mind fills us rather with joy and hope.

HEMIANOPIA, WITH ESPECIAL REFERENCE TO ITS TRANSIENT VARIETIES.

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THE following paper, with the exception of a few alterations, some of which were rendered necessary by the subsequent section of the brain in one of the cases described, was presented in June, 1897, as my thesis for the degree of M.D. of Cambridge. The paper was originally intended to deal with several extraordinary cases of transient hemianopia, associated with unilateral convulsions, which I have had the good fortune to meet with, the disturbance of vision lasting often twenty-four hours or more. Such cases, so far as I am aware, have never hitherto been described, and, in my opinion, they go far to prove a theory of cortical representation of the macula, and that the macular region in the retina is really innervated on the same plan as the rest of the retina. Their close analogy with epilepsy on the one hand, and with migraine on the other, the association of hallucinations in the blind field with hemianopia, and other points of interest, have led me to extend the original title of my paper. I shall, therefore, deal with my subject under the following headings:

(1) Bi-temporal hemianopia.
(2) Bi-nasal hemianopia.
(3) Hemianopia in hysteria.
(4) Homonymous hemianopia.
(5) Quadrantic hemianopia.
(6) Colour hemianopia.

(7) Hemianopic hallucinations.
(8) Hemianopia commencing with blindness.
(9) Central incomplete hemianopia.
(10) Double hemianopia.
(11) Cortical representation of the macula.
(12) Transient hemianopia.

I have to thank most of my late chiefs at the National Hospital for the Paralysed and Epileptic, Queen Square, Dr. Beevor, Dr. Bastian, Dr. Hughlings Jackson, Dr. Buzzard, Sir William Gowers, Dr. Ferrier, and Dr. James Taylor, for permission to make use of cases under their care in the hospital during my term of nearly two years as house physician. To Mr. Gunn also I am much indebted for his kind criticism and help, and to Dr. Luff for permission to refer to a case seen amongst his out-patients at St. Mary's Hospital.

Hemianopia, Monoyer's variation of the original word hemiopia, so as to indicate the symptom of half-blindness rather than half-vision, is, I think, preferable to the more clumsy term suggested by Hirschberg, hemianopsia. The term has generally been used to denote complete blindness in one or other half of the visual field, and it may be homonymous, in which either both the left or both the right halves of the two retinæ are anæsthetic, causing blindness of the opposite visual fields, or it may be bi-temporal, bi-nasal, or altitudinal. These latter forms will be only incidentally considered in this paper, altitudinal hemianopia, in which the dividing line is horizontal, occurring only in peripheral lesions of the optic path, as in neuro-retinitis, or possibly from disease of the chiasma, though, as suggested by Wilbrand (17), it is possible to imagine a loss of the lower quadrant on the one side and the upper quadrant on the other side, due to lesions in each cuneus. Indeed, such a case has now been recorded by Weymann (16), in a man who had a sudden fit, with loss of consciousness and hemiplegia. His vision was much affected, and perimeter charts showed the case to be one of double quadrantic hemianopia, the lower right and upper left quadrants in each eye being blind.

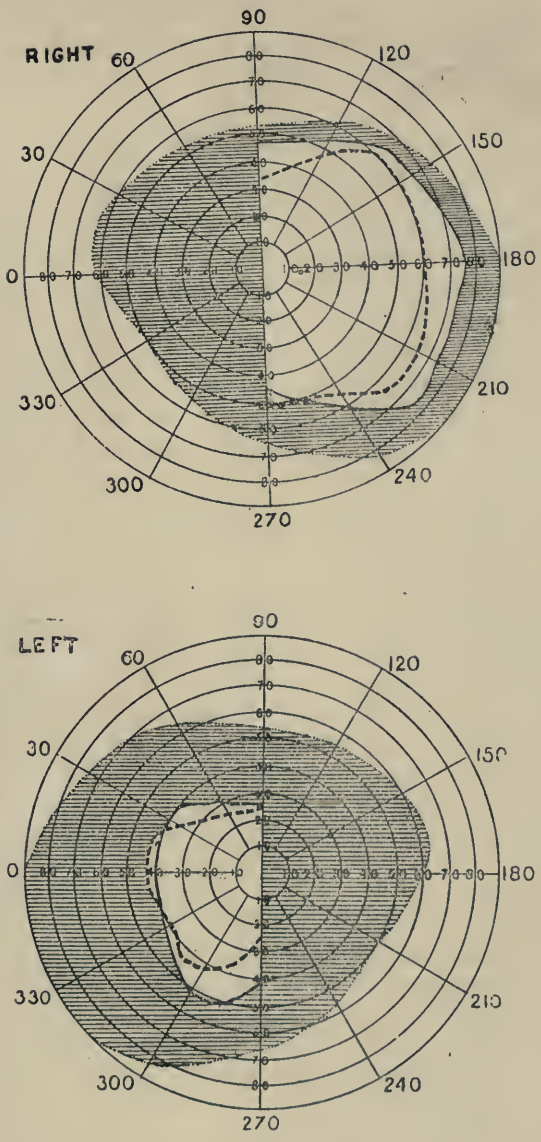
Bi-temporal Hemianopia.

The bi-temporal form can only be caused by a lesion at the chiasma, generally by a growth pressing down on its centre, as in tumours of the pituitary body, in acromegaly, or a fractured base. With both eyes open, the visual loss in these cases amounts to a bilateral peripheral contraction of the field down to 60°. Würdemann and Barnes (1) record an interesting case of bi-temporal hemianopia with the hemianopic pupil symptom, probably due to hæmorrhage in the region of the chiasma. The visual loss proceeded to nearly complete blindness, and then cleared up, a second attack later on causing right homonymous hemianopia.

Bi-nasal Hemianopia.

The bi-nasal form is the rarest, possibly because of the difficulty in examining for it, and the extremely slight visual loss when both eyes are open. It has been observed in tabes, and in one or two obscure cases probably due to a symmetrical neuritis of the outer parts of the chiasma, and it is possible to imagine symmetrical growths on both sides of the chiasma giving rise to it, as in Knapp's (5) case, quoted by Starr. Hamilton (2) has recently described and given perimeter charts of a case of bi-nasal hemianopia occurring in a man aged 56, who suffered from aortic regurgitation, and had two seizures followed by right hemiplegia. There was double optic atrophy, and considerable diminution of visual acuity, with partial colour loss in the remaining temporal fields, white and blue alone remaining. The bi-nasal hemianopia was, however, absolute, and was ascribed to embolism damaging the vascular supply to the tracts, both being symmetrically affected. No autopsy was obtained. The description of the case is, however, meagre, and it may possibly have been an instance of hemiplegia occurring in a case of tabes.

Strange to say, this form of hemianopia has also been recorded twice as occurring in hysteria, one case by Dr. D. B. Lees (3), in which bi-nasal hemianopia was found in a boy aged 11, and which had disappeared by the next day.



----- Blue field.

Fig. 1.

Bi-nasal hemianopia, after Hamilton.

Another instance is figured by J. H. Lloyd (4), from a case of Mitchell and de Schweinitz, in which also the remaining half fields are much contracted.

Hemianopia in Hysteria.

Homonymous hemianopia, or blindness to one side, may rarely occur in hysteria. Briquet (7), as long ago as 1859, says, "In some hysterics either lateral half of the retina may be insensitive, giving rise to hemianopia," but he gives no method of examination. Svynos (12), Galezowski (9), Rosenthal (13), Westphal, and Bonnefoy (6), also give cases, Svynos mentioning that hysterical hemianopia is more frequent on the left side. Dercum (8) also gives the charts of a case of left hemianopia in hysteria with contraction of the remaining half fields. According to Galezowski and Daguenet (11), hysterical amblyopia sometimes assumes the form of hemianopia, or of a transitory central scotoma, or even of complete amaurosis of shorter or longer duration. This hysterical amaurosis is rare and affects sometimes one eye, sometimes both. Galezowski (10), quotes a case of a young woman who, being frightened by her sister being attacked with cholera, fainted, and on her recovery from the faint was quite blind. After some days her vision returned on one side, taking the form of right homonymous hemianopia. The blind field then also began to recover, her field of vision enlarging daily, and soon her cure was complete.

I have met with one similar case.

M. P., a married woman, aged 29, was admitted into the Queen Square Hospital, under Dr. Buzzard's care, in September, 1895. She had had numerous fits, probably hysterical, and when admitted she was quite unable to stand, or even to move the legs at all. There was general analgesia of the whole body, and tactile anæsthesia of the feet and legs up to the knees. There was no unilateral loss of the special senses, but on taking charts of the visual fields with the perimeter there was found to be left hemianopia, with contraction of the remaining half fields. Her vision was R. V. $= \frac{6}{36}$, L. V. $= \frac{6}{24}$, though on putting the trial frame before her eyes and a plain glass instead of a lens, her

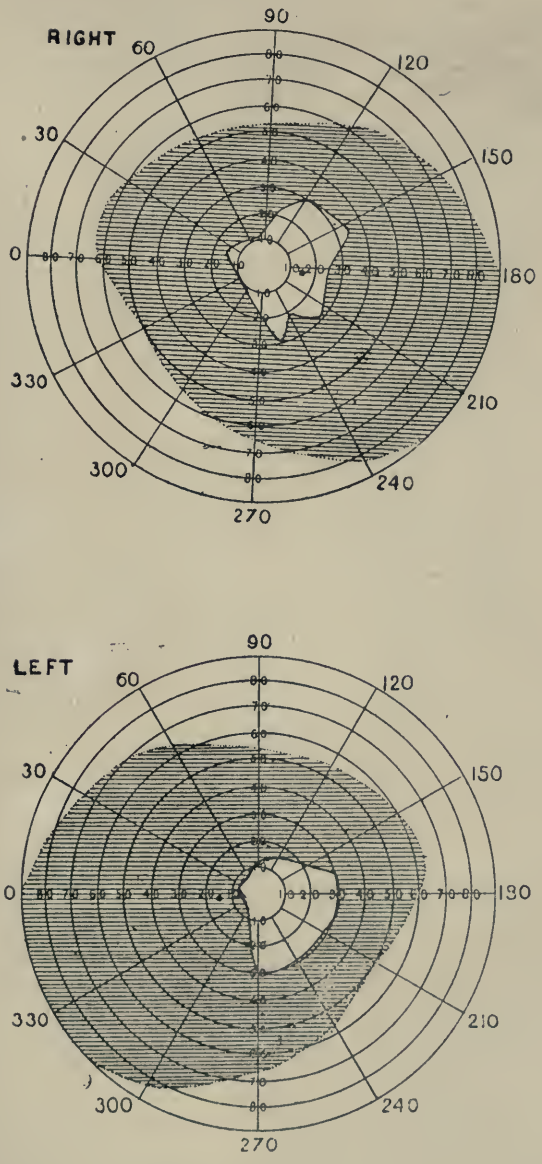


FIG. 2.

M. P.—Case of hysteria, with left hemianopia, and contracted fields.

vision at once improved to R. V. $= \frac{6}{12}$, L. V. $= \frac{6}{9}$, nearly. A week later I suggested to her that she should not be able to see towards the right side, and the next morning when she woke up she was totally blind, remaining so all day. Her pupils reacted perfectly to light, but she was much upset at her loss of sight, and a match suddenly lit and brought quickly near the eyes produced no reflex starting or movement of the lids. In the evening the passage of galvanic shocks through her eyes and temples, combined with suggestion, restored her vision partially, and by the next day it was much improved. She steadily got well under treatment by the wire brush, the visual fields gradually enlarging, and acuity of vision improving, until her discharge a month later, when her visual fields were quite normal again, and vision $\frac{6}{5}$ in each eye.

Homonymous Hemianopia.

Homonymous hemianopia, the ordinary form, is generally due to a lesion in the optic path anywhere between the chiasma and the occipital cortex, either in the optic tract, the external geniculate body, pulvinar, the optic radiations in the posterior part of the internal capsule, or in the cortex of the cuneus.

A case has been reported by Eskridge (44), of right hemianopia, complete up to the mid-line, which was caused by a gun-shot wound, the *post-mortem* showing complete destruction of the left cuneus alone, as clean as though done for a physiological experiment. Henschen would limit the half-vision centre in the cuneus to the grey matter of the middle third of the lips of the calcarine fissure, on the internal surface of the posterior lobe. In the large majority of cases hemianopia is due to cortical softening, but various tests have been devised to distinguish between cases due to damage to the tract, or internal capsule, or to destruction of the cortex. The best known is the hemianopic pupil of Wernicke, the pupil contracting when light is thrown obliquely through it on the sound side of the retina, but remaining motionless when the light is thrown from the other side on the anæsthetic half of the retina. This symptom, if present in homonymous hemianopia, proves the lesion to affect the optic tract between the chiasma and

the ant. corpora quadrigemina where the fibres concerned in the pupillary reflex leave the tract to reach the nucleus of the third nerve. This symptom occurs in hemianopia due to tumours of the base or crus pressing on the tract, in the bi-temporal hemianopia due to lesions of the chiasma, acromegaly, &c. Homonymous hemianopia may also occur in tabes, as I have seen in two cases with optic atrophy, and in disseminated sclerosis also with optic atrophy, probably due to a patch of sclerosis affecting the optic tract.

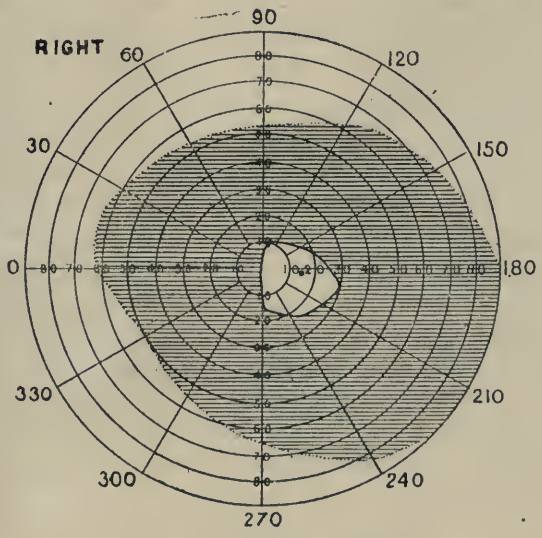


FIG. 3.

W. L.—Case of tabes, with optic atrophy. Left hemianopia, and contraction of remaining half field. The hemianopia had been noticed by the patient for at least eight months, but when the charts were taken the left eye had become blind.

If the pupil reacts equally well when the light is thrown from either side, the hemianopia is either functional, or due to a lesion in the optic radiations or cuneus.

Visual sensations are divided into the three perceptions of light, form, and colour, and various subdivisions of the occipital cortex have been allotted to each, Wilbrand placing the colour centre in front of the apical region, and Sir William Gowers accepts his theory. Mackay (18), however,

proves that there is always some diminution of the form sense in so-called pure cases of hemiachromatopia, and according to Swanzy (45) "It is now generally believed that relative hemianopia (*e.g.*, colour hemianopia alone) is the result of a lesion of less intensity than that which causes absolute hemianopia, ... and that the colour sense is more easily affected by disease than the form or light senses, and that, too, irrespective of the position of the lesion in the visual path." Again, the light sense only may be retained, perception for both form and colour being lost.

Hemianopia, if occurring alone, is probably cortical in origin; but, according to Swanzy the chief diagnostic symptom of a cortical lesion is "vision nulle," that the patient, though he may be aware of the loss of half his visual field, is as unconscious of any scotoma as a healthy person is of his blind spot. Lesions below the cortex, as in the optic radiations, are said not to give rise to vision nulle, that the patient then has a sensation of darkness in the blind half field. I have examined for this symptom several times without any satisfactory result, and in one case to be presently related, in which the lesion was in the posterior part of the internal capsule, the patient was aware of no defect of vision whatever, and could with difficulty be persuaded that a considerable portion of his visual field was lost.

Quadrantic Hemianopia.

Quadrantic hemianopia is said to be generally due to a cortical lesion, the lower part of the cuneus corresponding to the lower quadrant of the field in each eye, as in a case published by Hun (15) in 1887. Wilbrand mentions the great preponderance in the statistics of cases in which the left lower quadrant is affected.

Sir W. Gowers (14) also figures a case of quadrantic hemianopia in which the diagnosis was a lesion near the hinder part of the thalamus. The following case is a good illustration of the fact that quadrantic hemiachromatopia may be due to a lesion in the posterior part of the internal capsule.

R. F., a man, aged 43, in August, 1896, was suddenly seized with left hemiplegia without loss of consciousness, with the sensation as though he had been struck on the back of the neck. The hemiplegia was not complete at first, but became nearly so later, with great rigidity of the arm and leg, and pain on movement. When admitted into the Queen Square Hospital in March, 1897, under Dr. Bastian's care he had almost complete left hemiplegia with great rigidity, partial left hemianæsthesia,

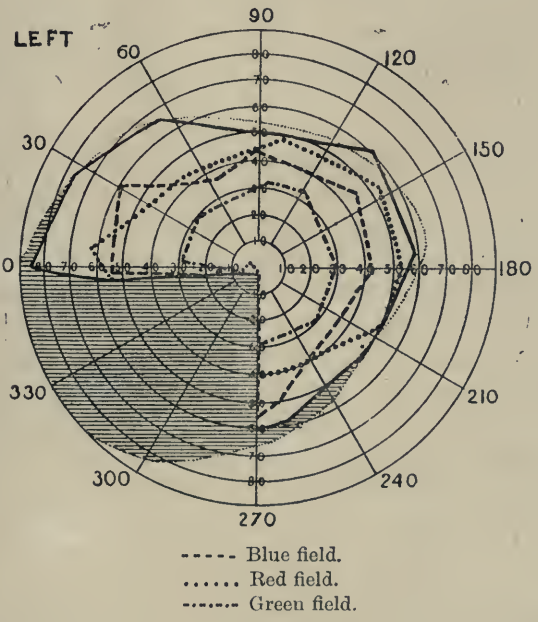


FIG. 4.

R. F.—Case of left quadrantic hemianopia, complete for colour, but partial only for form sense. The field of the right eye could not be charted by the perimeter, owing to the patient becoming exhausted by sitting up. Careful testing, however, showed it to correspond accurately with that of the left eye.

and anæsthesia dolorosa most marked on the left arm. He had evidently some affection of his left field of vision on rough testing, and on being carefully tested with the perimeter he was found to have complete loss of all colour perception, even of large area, in the left lower quadrant for each eye; small objects also, as the head of a pin, or a disc of white paper 1 cm. in diameter, were

also invisible, and he could not distinguish between one and two fingers held up in the same area, though he could readily do so in each of the remaining quadrants. A large object, as a hand moving, or a sheet of notepaper, he could see readily enough in the affected area, and his light perception was also good. His discs and fundi were normal, and he was not aware of any defect of vision before being tested. His heart, rapid from the first, became very irregular a month after admission, and a loud systolic murmur appeared. He became much weaker and delirious, and died suddenly on April 14. At the autopsy, eighteen hours after death, the cuneus was found quite normal on each side, but there was a patch of softening in the right internal capsule. The heart and valves were normal.

Hemianopic Hallucinations.

Hallucinations in the blind field are said by Swanzy (19) to be possible in lesions of the optic radiations, but not in hemianopia due to cortical damage. The reverse I think rather is more common, and the only case with autopsy I can find which supports his contention is one published by De Schweinitz (27), of a man, aged 29, who had visual hallucinations of chairs, tables, and other furniture, to his left, followed soon after by convulsions and left hemianopia. He had repeated seizures, and developed delusions of grandeur, and was sent to an asylum. At the autopsy made by Dercum, gummatous infiltration of the base of the brain, pressing on the right optic tract was found but, no mention of the occipital lobe is made. Henschen (24) records four cases, two with autopsy. In one case of right hemiplegia and hemianopia with hallucinations of seeing a boy near the foot of the bed on the blind side, the autopsy showed softening in the occipital lobe and internal capsule. In the other there was left hemianopia without hemiplegia, and hallucinations of seeing persons and faces in the blind field, and the autopsy showed softening in the right cuneus and lingual lobe, and in the right thalamus. Mackay (18) records the case of a farm labourer, aged 37, who suffered from partial right hemianopia following a blow on the back of the head, with recurring attacks of erythropia, and of

visual hallucinations, lasting a few minutes, of folk and horses moving in a reddish atmosphere, limited to the blind field and never crossing the mid-line. He had complete right hemiachromatopia with some impairment of the form sense, the dividing line passing vertically through the fixation point in each eye. He died three years after the injury, being seized with right hemiplegia and aphasia, but no autopsy was made. Mantle (25) describes the case of an artist, who had pure motor aphasia, followed eleven weeks later by word deafness, and word and mind blindness. He became gradually deaf, with dimness of sight, the deafness and blindness becoming complete in about twenty-four hours, though the pupils still reacted to light. The deafness and blindness continued for ten days, when his vision gradually cleared up into right hemianopia after a fortnight. He had two relapses of loss of sight, the second one lasting four weeks, but clearing up again into right hemianopia. He also had visual hallucinations in the blind field of a man standing at the back of his head, holding two lighted candles. Seguin (28) in 1886, was the first to record a case of this kind, and others have been recorded since by Bidon (20) in 1891, Putzel in 1888 (26a) and by Wilbrand (17) in 1890. In Wilbrand's case there were hallucinations of heads, furniture, cats, &c., in the blind field, and at the autopsy there was found hæmorrhagic softening of the medullary substance in the third occipital convolution, the superficial cortex being yellowish and discoloured. Dr. W. S. Colman (21) has recorded the case of a woman, aged 45, suffering from left hemiplegia, hemianæsthesia, and hemianopia, due probably to a lesion in the posterior part of the right internal capsule. For three weeks after the onset she saw numerous faces of men, women, and children, always in the blind field only. The faces were unfamiliar and not unpleasant, and she quite recognised that they were unreal. The hemianopia persisted, although the hallucinations passed off. Peterson (26) thinks the pathological basis in these cases must be irritation of the cortex of the occipital lobe, and he describes hallucinations occurring only in the right half fields in a case of chronic delusional insanity

without hemianopia, and mentions a case of Dr. J. Van Duyn's, of Syracuse, U.S.A., of a woman, aged 45, who developed left hemianopia, and one week later had constant hallucinations of seeing animals and children moving in the blind fields, this continuing for four weeks, and then suddenly ceasing, though the hemianopia persisted.

In Putzel's case, a man, aged 54, developed right hemianopia, followed ten days later by hallucinations in the blind field of men, flies, insects, &c. At first he recognised their unreality, but after a few days he became convinced they were real. The spectra became more frequent, and he would then hunt for them in cupboards and corners. He developed uræmia and died six weeks from the onset of the hemianopia. At the autopsy a spot of softening was found in the left cuneus, extending across the calcarine fissure, and forwards to the parieto-occipital fissure. The kidneys were extremely cirrhotic.

Hack Tuke (22), mentions hemianopic hallucinations as occurring "in the sane as well as in those subject to epilepsy, migraine, anæmia, and delusional insanity. When coincident with hemianopia, such hallucinations probably originate in a cortical irritation or malnutrition limited to the occipital lobes." Dr. Colman, in describing some hemianopic hallucinations, mentions that they consist usually of faces, the most easily remembered individual characteristics, or else of objects, such as rats, beetles, &c., which from early infancy are usually associated with sensations of alarm. He agrees in the refutation of Brewster's theory of hallucinations, that there is an actual excitation and chemical change produced in the peripheral organ, such as the retina, as would have been produced had an image of the real object fallen on it. He goes on to say that it is due evidently to a condition of the perceptive centres, and draws attention to the fact that opacities of the vitreous may at first cause muscae volitantes, which are recognised to be part of the individual, though later, as the mental condition deteriorates, they may take the form of faces or animals. These are for a time recognised as existing only in the patient's own sensations, without any external warrant,

but later on he fails to recognise that they have anything to do with himself, and refers them to the world outside him.

Binet has supported the theory of peripheral origin of hallucinations, and has shown that in hypnotic hallucinations the vision may be doubled by means of a prism before one eye, but this point is disputed by Hack Tuke. The truth would appear to lie midway between the two assertions, and we may regard hallucinations and illusions as two extremes of the same phenomenon, and that there is no hard and fast line separating the one from the other. On this basis, we can understand how that in a patient, hypnotised to believe that a spot on a piece of paper is a photograph, the prism test may produce doubling of the images. This test, then, depends on the strength of the external stimulus, and when the vision is more subjective in origin, and persists after closure of the eyes (approaching Esquirol's definition of an hallucination as differing from an illusion in that, in the latter an external object forms the basis of an erroneous perception) then lateral pressure on the eyeball would cease to cause doubling of the image.

J. Christian (43), in his article on hallucinations, adopts Ritti's theory in placing the seat of hallucinations in the optic thalami, and he lays down in his fourth law that there is always the reminiscence of something previously seen, that the sensory centres create nothing, invent nothing. This axiom will, I think, hold good if not pressed too far, that is to say, the main outlines of figures appearing in hallucinations must have been perceived before, though the faces may be unfamiliar, the figures differently grouped, clothed, &c. Talma, the actor, had the power of voluntarily producing the hallucination of divesting his audience of clothes, hair, flesh, &c., and of seeing them as skeletons filling the theatre, and applauding. That he must have seen skeletons at different times of his life may easily be believed, but that he could have seen so many at one time, and grouped in such a way, is impossible.

Assuming then that visual hallucinations occurring in the blind field are not peripheral, that is, retinal in origin, the question arises as to the seat of their production.

Peterson considers them due to irritation of the occipital cortex. In de Schweinitz's case, however, already mentioned, the lesion was in the optic tract, and occasionally hemiopic hallucinations have been met with without hemianopia, as in delusional insanity. It is, in my opinion, impossible to conceive that such highly elaborated sensations as have been described in cases of hemianopic hallucinations, such as visions of persons, animals, furniture, and the like, can possibly be elaborated in the half vision centre in the cuneus. This centre, as a sensory centre, no doubt corresponds to Hughlings Jackson's "middle level" of the motor representation of the limbs and trunk, the so-called "motor area" in the Rolandic cortex. Low-elaborated visual sensations, such as red and green lights, or the fortification spectra of migraine, are probably originated by a paroxysmal discharging lesion in the cuneus, but complex visual phenomena are probably no more originated there than are complex purposive movements originated in the "motor" limb centres. A higher visual centre, then, must have a large share in their production, and to this centre, which possibly is the angular gyrus, we must look for the ultimate seat of elaboration of such sensations, though such action may be set up reflexly by peripheral irritation of the optic path. For this purpose we may consider the cuneus to be part of the periphery, and in this way we are enabled to understand how complex visual hallucinations may be due to disease anywhere in the optic path, either to vitreous opacities, retinal disease, lesions of the optic tract, optic radiations, or cuneus. The peripheral disease merely determines the localization of the spectra, which in disease of one eye are referred only to the visual field of that eye, whereas in disease of the optic tract, optic radiations, or cuneus, they are referred to the corresponding half fields.

An instance somewhat parallel to migraine, of discharge in a sensory centre, is seen in some cases of trigeminal neuralgia, rightly called epileptiform neuralgia, in which the discharge occurs, no doubt, in a centre higher than the Gasserian ganglion, probably in the sensory nucleus of the fifth nerve, but which may be set up reflexly by irritation of a peripheral portion of the nerve.

I have myself met with five cases of hemianopia in which there were or had been visual hallucinations in the blind field.

In a case I published last year (23), of localized convulsions on one side, the patient on several occasions soon after the onset of his hemiplegia and L. hemianopia, which occurred six months before his admission into the Queen Square Hospital, had hallucinations in which he saw Buffalo Bill's camp, the men and horses &c., and fancied he heard the guns firing and the shouting at the exhibition at which he had worked ten years previously. These hallucinations occurred at night on going to bed, but if he put out the light and turned over on his right side they disappeared, an evidence of the influence of peripheral stimulation by the light. Later on he had frequent attacks of seeing red and green lights straight in front of him ; in his case there was complete loss of the form and colour sense, but the light sense was fair, even out to the periphery. The autopsy showed cortical softening both of the cuneus and of the angular gyrus on the right side, but the internal capsule was normal.

The following case is an instance of partial hemianopia in which a peripheral zone of the left lower quadrant remained intact. This was in all probability due to softening of the right cuneus, and the patient also suffered from frequent attacks of seeing red and green lights, always to the left, in the blind field.

J. B., a woman, aged 36, with a history of probable syphilis, suddenly lost her sight ten years ago, the day after a severe flooding. At first she could see scarcely anything, but in a month's time when she went to a hospital this had cleared up into left hemianopia, which has persisted ever since. She had no hemiplegia at that time, but two years ago she had a slight attack of right hemiplegia ; and again in September, 1896, had an attack of left hemiplegia without loss of consciousness, but no further affection of vision. When admitted into the Queen Square Hospital under Dr. Beevor's care five days later, she had complete left hemianopia, total paralysis of the left leg and upper arm, but could move the left thumb, fingers, and wrist slightly. There was marked early rigidity of the left arm and leg, with slight left hemianæsthesia, but no affection of hearing, smell, or taste. The heart was enlarged, with a systolic murmur at the

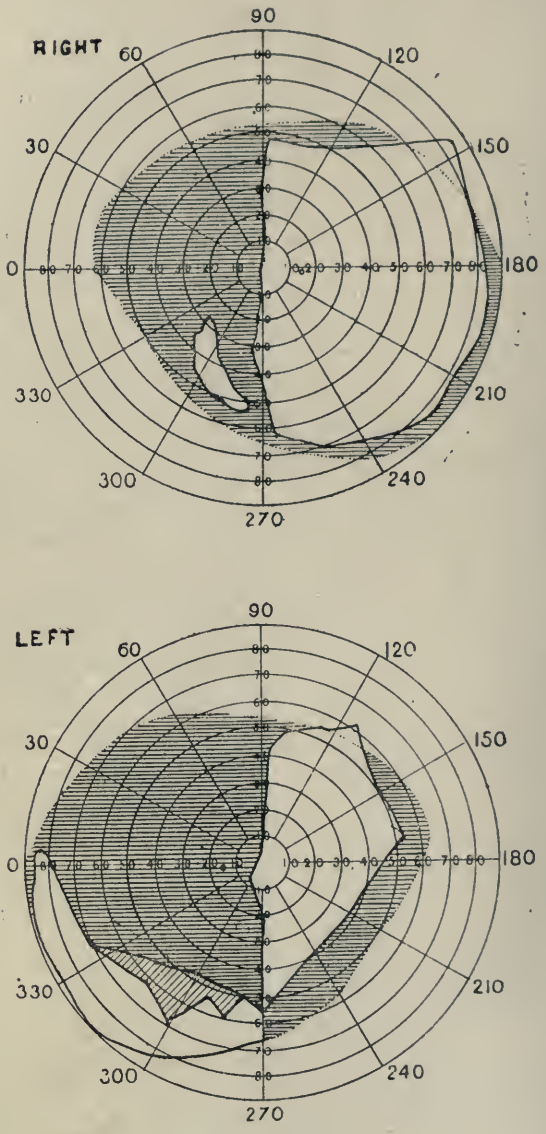


FIG. 5.

J. B.—Left hemianopia, complete in upper quadrants, but with peripheral strip of normal vision in lower quadrant, best marked in the left eye.

apex: artery walls thick, and albumen in urine $\frac{1}{12}$. The right arm and leg were normal, but her articulation was very thick and difficult to understand, and remained so for the six months she was under observation, this being indeed her only pseudo-bulbar symptom, as she could protrude her tongue and swallow quite well. Thickness of articulation is, I believe, an important symptom in double hemiplegia, and is often well-marked when there are no other bulbar symptoms proper.

By the end of October she had regained some vision in the periphery of the left half fields, and charts taken early in November showed that this return of vision was limited to the periphery of the left lower quadrant in each eye, a strip of about 20° in width from the extreme periphery inwards on the left side having regained normal vision, though the whole of the left upper quadrant and the central portion of the lower quadrant remained completely blind in each eye. In this peripheral strip in the left eye, she could distinguish between one and two fingers held up, and was able to recognise all colours, even in the extreme periphery, if of sufficient area, coloured cards about three inches square being generally used at a distance of about fourteen inches. A small corresponding patch in the lower nasal field of the right eye had also regained the light sense, but form and colour were not recognised in it. She remained under observation for five months longer, but the visual fields remained the same. During this time she had three fits, with loss of consciousness and left-sided convulsions, followed by temporary increase in thickness of articulation, but no alteration in vision. In one of these fits she dislocated her lower jaw. On several occasions she complained of feeling unwell, with headache, and of seeing red and green lights to her left. These were limited to the left or blind half field, never crossing the mid-line, and they appeared to be moving in both directions, from the periphery towards the centre and also in the reverse direction. They also seemed visible over the area of the peripheral strip of vision in the left lower quadrant.

The persistence of a strip of peripheral vision in hemianopia is rare. Delépine (30) records a somewhat similar case with autopsy, in a man who had sudden onset of right hemianopia with slight right hemiplegia. When his visual fields were charted, eight months later, there was found complete right hemianopia with the fixation point included

in the seeing half, but with a strip of peripheral vision, about 10° in width, situated chiefly in the lower right quadrant of the field, and only in the right eye, the nasal field of the left eye being completely blind. At the autopsy, three months later, there was found softening of the left cuneus, with the exception of its postero-superior margin. This case, then, is not in accordance with that previously recorded by Hun (15) in which loss of the lower quadrant was found to be due to softening of the lower part of the cuneus only. They would, however, correspond if Henschen's view be accepted, that the upper edge of the calcarine fissure represents the upper quadrants of the retinæ, and therefore the lower quadrants of the field, the lower edge of the calcarine fissure representing the lower quadrants of the retina. In Delépine's case there was no peripheral vision in the nasal half of the left eye, and my case resembles it in so far that when first tested she seemed also to have no patch of peripheral vision in the nasal field of the right eye corresponding to the peripheral strip in the left eye. Careful repeated testing was necessary before it was discovered, and even then the visual acuity amounted only to seeing an object move, and no colour could be perceived in it, though colours were easily recognised in the periphery by the left eye. Delépine gives a reference (33) to another similar case.

Another case of hemianopia with hallucinations in the blind field I have had the opportunity of examining is that of a man, N. B., aged 51, who had an attack in June, 1896, in which he suddenly lost power of speech, using wrong words, and forgetting the names of things. At the same time the fingers of his right hand became numb and useless. This all passed off in two minutes. He then began to suffer from left-sided headaches, and during the next six weeks he had five or six similar attacks. During one of these subsequent attacks of temporary aphasia and weakness of his right side he suddenly noticed while reading that his sight was confused and that the print seemed to run together. After that he noticed he could not see so well to the right, and he used to bump up against things on his right side, and had to be careful whilst crossing the road. He also has had visual hallu-

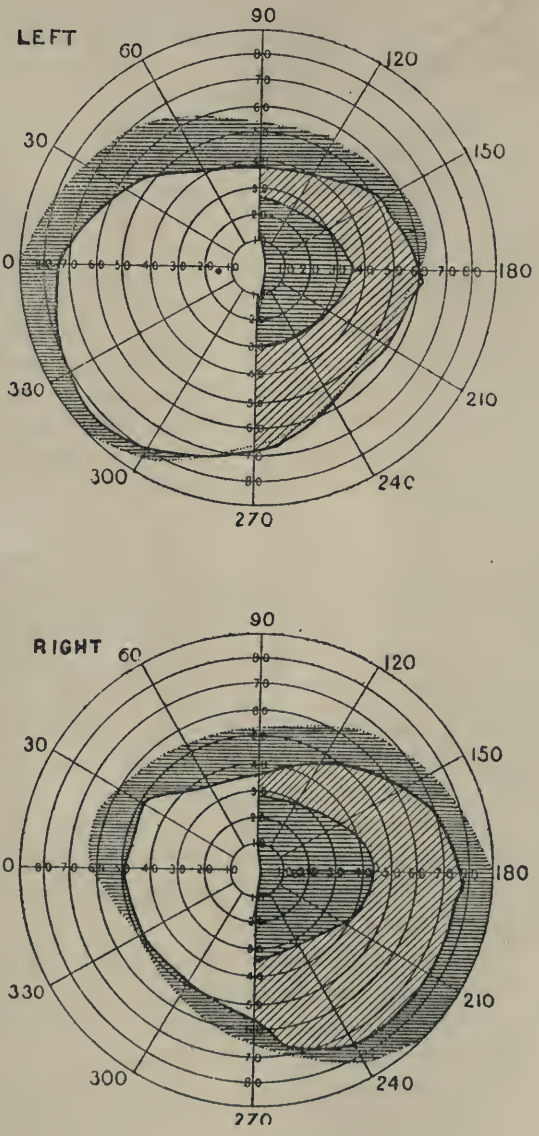


Fig. 6.

N. B.—Case of right central incomplete hemianopia. Fields for white spot 1 cm. in diameter. The area of indistinct vision is indicated by lighter shading.

cinations of animals and faces moving about to his right. His vision has remained the same ever since, and when admitted into the Queen Square Hospital under Dr. Hughlings Jackson, in February, 1897, perimeter charts taken showed incomplete right hemianopia, in which the central portions of the half fields were completely blind over an area extending about 30° above and below the fixation point, and 38° laterally in the left eye and 45° laterally in the right eye. To the right of this blind area he could recognise the movement of even small objects, though his form sense was much impaired, and he could not distinguish between one and two fingers; he could, however, recognise colours even of small area. His condition while under observation has remained the same, there being slight weakness of the right arm and leg. When last seen in May he had had no further fit and was in good health, but the hemianopia remained exactly as before. There was no history of syphilis, and an accurate diagnosis seemed impossible, though cortical softening seemed probable.

I have met with two other cases of visual hallucinations on the hemianopic side, which will be related presently in full, one in a woman aged 51, who had partial left hemiplegia and a history of recurring convulsions. Her visual fields were normal, except once after a fit when I found absolute left hemianopia lasting from four to six hours. Later on, after another left-sided fit without hemianopia, she developed visual hallucinations in which she saw people in wheel-chairs, coloured brightly in green and gold, passing her always from her left side as far as the mid-line. In another case, a man, aged 54, suffering from partial right hemianopia and word blindness, had recurring convulsions. Some of these attacks appeared to be truly epileptic, but two have been typically Jacksonian, without loss of consciousness, and with clonic convulsions of the right arm and leg lasting for two hours. He frequently sees the faces of his children, his pet dog putting up his paws on to the bed, or his cat, sometimes school children running along, some being dressed in red cloaks as they used to pass his window at home, and similar objects in his blind field to his right. These hallucinations last only a very short time, but are frequent, and irrespective of the fits, and are suffi-

ciently distinct for him unconsciously to step to one side to avoid them, though he at once recognises their unreality. The case is an obscure one, but the lesion is probably cortical, possibly in the neighbourhood of the angular gyrus, and damaging the underlying optic radiations on the left side.

Hemianopia commencing with Blindness.

Hemianopia may commence with complete blindness, which, after a varying interval, clears up, leaving normal vision in the half fields of one side. This transient amaurosis must, I think, be due to inhibition of the remaining half vision centre, and is perhaps more liable to occur when the lesion is sudden damage to one cuneus. Dr. Allen Sturge (29) describes a most instructive case of absolute blindness coming on suddenly in association with right hemiplegia, hemi-anæsthesia, and loss of taste, smell, and hearing on the right side. On the next day some vision had returned in the left eye. Two days later the right eye was still quite blind, and there was complete loss of the right field in the left eye, but the left half field was quite good. The left half field in the right eye rapidly recovered vision, leaving right hemianopia, which persisted. This case is generally misquoted as one of hysterical hemianopia, because it was related during a discussion on hysterical hemi-anæsthesia at the British Medical Association meeting at Cambridge, in 1880.

Gowers (14) mentions a case in which complete right hemianopia cleared up to quadrantic hemianopia. In this case a partial lesion may have temporarily damaged the whole half vision centre, or possibly damage to the optic radiations may have cleared up partially, leaving the fibres of one sector destroyed.

The disturbance of vision does not often amount to absolute amaurosis, but frequently there is very great disturbance of vision at the commencement, the patient stating that he could see scarcely anything, and the vision gradually improves, leaving hemianopia on one side.

The case described by Mantle (25) has been already related (see p. 14) of a man in whom right hemianopia was ushered in by complete blindness lasting twenty-four hours, during which the pupils re-acted perfectly to light.

Central Incomplete Hemianopia.

According to Gowers (14b), "when a complete hemianopia lessens, it may do so from the medial region towards the periphery so as to leave a symmetrical peripheral loss; or else from above or below, so as to leave a quadrantic defect. It rarely clears from the periphery, so as to leave symmetrical scotomata having one extremity in the central region."

Lang and Fitzgerald (31) have recorded such a case associated in its onset with hemiplegia and paralysis of the upward and downward movement of the eyes and lids, due probably to a lesion in the basal ganglia on one side. The ocular paralysis disappeared as the hemianopia cleared up from the periphery a week later, leaving central scotomata close up to the fixation point. They also give references (32) to several other cases. Wilbrand (17) also records one. Delépine's and Hun's cases have already been quoted, and the two cases I have met with—a woman, E. B., and a man, N. B., have already been related.

Double Hemianopia.

Double hemianopia also almost constantly commences with complete blindness, and according to Gowers it necessarily causes permanent amaurosis. Numerous cases are, however, now recorded in which complete blindness has suddenly occurred, or in which hemianopia on one side has been followed later by sudden complete blindness, and afterwards a small central area of vision returns, generally with considerably diminished acuity. The fields in these cases resemble the pin-point fields in hysteria, but a characteristic loss of power of orientation, and of finding their way about, is mentioned by Foerster (35), Groenouw (35b), and Vorster (35c), though it was not present in

Schweigger's case (35d), and this would distinguish them from the functional cases which seem in no way the worse for their restricted fields. In the latter case no doubt the half vision centres do perceive, and so visual impressions may produce reflex effects, though higher psychical influences inhibit the sense perceptions. A case of double hemianopia with autopsy is recorded by Schmidt-Rimpler (35a).

Cortical Representation of the Macula.

Such cases of double hemianopia, with return or persistence of central vision, are a very strong argument in favour of Foerster's theory of a cortical representation of the macula. A case recorded by Wilbrand (17), forms a converse to them : a man suddenly finding his vision confused, and on examination was found to have symmetrical scotomata touching the fixation point on the right side of each field, and equal in size to twice the diameter of the optic disc. A somewhat similar case I have myself met with has already been related. (See case of N. B., fig. 6.)

There are two opposing theories with regard to the representation of this region in the cortex. (1) That the whole of each macula is represented in each half vision centre, and that there is a special decussation of the macular fibres at the chiasma. This theory necessitates that the dividing line between the seeing and the blind fields in all cases of hemianopia, whether due to cortical or to tract lesions, should invariably pass round the fixation point, which is then included in the seeing half. Cases in which the line appears to pass close to or even to go through the fixation point are accounted for by individual variations in the nervous supply of the macula. Gowers strongly upholds this theory, and is of opinion that in all cases, if carefully and accurately tested, the dividing line would be found to pass around the fixation point.

Allen Starr (5) says "In no case of hemianopia is direct vision at the fixation point affected." Foerster's theory, quoted by Swanzy (19), supposes that the nervous supply of the macula is invariably arranged on the same plan as the

rest of the retina, that each half of it is innervated from the corresponding half vision centre in the cuneus. Further, that there is a special area in each half vision centre in which the corresponding halves of each macula are represented, and which is more richly supplied with blood vessels, or at all events offers more resistance to vascular softening than the rest of the visual centre. According to this theory, in a total destruction of one half vision centre, as by a hæmorrhage, or in destruction of one optic tract, the dividing line between the seeing and blind fields would pass through the fixation point, but that in a lesion such as softening from embolism or thrombosis, this special region, owing to numerous anastomoses in its blood supply, escapes, and the dividing line would then pass around the fixation point, leaving it in the seeing half. This theory, therefore, will account for both varieties in the position of the dividing line in hemianopia, and, since both varieties are well supported by numerous careful observations, it is worthy of greater credence. Wilbrand (17) states that in 77 cases of lateral hemianopia the dividing line was central in 29, but of 32 cases of bi-temporal hemianopia in only 9 was the dividing line central. Cases of double hemianopia with persistence of a small central area of vision strongly support the latter theory that in each half vision centre the area representing the macula is less liable to suffer than the rest of the cortical centre. Complete proof that the macula is innervated on the same plan as the rest of the retina, and that there is not a special decussation of the macular fibres, is in my opinion afforded by the study of cases of transient hemianopia. In nine instances of this, occurring in three patients, I have examined this particular point as to the position of the dividing line with especial care, not trusting to the perimeter alone, which is not sufficiently accurate for such a delicate observation, but using a method as follows which was shown to me by Sir William Gowers. The patient is made to sit facing the observer with one eye shut, at a distance of about two feet. The physician then closes his own eye opposite to that shut by the patient, who is then told to gaze steadily into the observer's open eye opposite,

who in turn watches the patient's pupil. The line of fixation is thus the same in each case and the slightest wavering of the patient's eye is at once noticed and allowed for by the observer. Then, using his own visual field as the normal, and holding the test object, such as a small piece of white paper on the end of a black pen, half way between himself and the patient's eye, the latter's visual field can be approximately charted. It is, however, for determining the exact position of the dividing line in hemianopia that I find this method specially useful. A very small test object must be used, such as a piece of white paper 1 mm. in diameter, fixed on the end of a black pen and held close to the observer's face so as to be in the same focus for the patient as the observer's eye that he is looking at. If it is then brought slowly inwards towards the fixation line, it can be exactly determined by the observer, after repeated trials, whether or not the object is seen by the patient before it reaches the line of fixation. In nine instances of transient hemianopia, each lasting several hours, in which I have examined this point during the stage of absolute loss of half vision, I have found the dividing line to pass accurately through the line of fixation, no object, not even the flame of a match being perceived until it crosses this line. In these cases, since the vision clears up to normal in about twenty-four hours, more or less in different cases, the loss of function of the half vision centre must be due to exhaustion, and not to any gross lesion, and therefore the centre for the macula may suffer equally with the rest of the cortical visual centre. In all these cases, too, vision clears up from the centre to the periphery, central vision being first established, and the field gradually widening, an additional evidence that the cortical centre for central vision has greater recuperative power than that for peripheral vision.

On the other hand, in no case of persistent hemianopia that I have as yet examined by this method has the dividing line passed through the fixation point, though in some cases it passes extremely close to it, and I think it highly probable that the fixation point would be found included in the seeing half in all cases of persistent hemianopia, as Sir W. Gowers

has suggested, if tested by this method. This apparent discrepancy between cases of transient and persistent hemianopia may, I think, be accounted for in one of two ways: (1) in cases of persistent hemianopia the cortical centre for the macula may have either escaped destruction, or have regained some of its functions; or, (2) the patient may have developed by education a new fixation point in his retina close to the original fovea centralis. This is, I think, most likely the case in those cases in which the dividing line passes very close to the fixation point. On the other hand, in cases of complete transient hemianopia there is no time for possible education of a new fixation point, and so the dividing line in them invariably passes through it.

Transient Hemianopia.

I now come to the main object of this paper, the discussion of *Transient Hemianopia*. This is a not uncommon phenomenon of migraine, and according to Gowers it sometimes occurs, like other manifestations of that disease, as an isolated symptom, apart from headache. Gowers (39) also, as long ago as 1877, mentions its occurrence in acute cerebral lesions with hemiplegia, during the stage of conjugate deviation of the eyes, as shown by the absence of any palpebral reflex to a finger darted at the eyes from the hemiplegic side. Swanzy (19), under similar circumstances, has seen it last three weeks, and in the following case I noted the presence of partial hemianopia four weeks after the attack of hemiplegia, though in another week it had disappeared.

E. W., aged 63, in September 1896, had a sudden attack of left hemiplegia, with partial loss of consciousness, and some interference with speech. On her admission to the Queen Square Hospital four weeks later, under Dr. Jackson's care, there was almost complete left hemiplegia, marked left hemianæsthesia, and partial left hemianopia, but no affection of taste, hearing, or smell. There was absence of any palpebral reflex to a finger darted at the eyes from the left side, though a brisk one was obtained from the other side. She could not count fingers further out than 30° on

the left side, and when reading the test types she would miss the left hand letter or the left half of a word. Vision was R.V. $= \frac{6}{18}$, L.V. $= \frac{6}{24}$. In another week her left visual field had quite recovered, though otherwise she remained much the same until her discharge.

Hemianopia, when due to an organic lesion, is rarely recovered from, and the following case is worth recording on account of the great improvement in the left visual fields which had been completely blind:

G. D., a man, aged 64, had an apoplectic attack on Nov. 3, 1896, with convulsion of the left arm and weakness of the left side, without loss of consciousness. At the same time he lost the feeling on the left side, and also the sight in the "left eye." There was no onset of complete blindness, and he could recognise people next day. When admitted into the Queen Square Hospital under Dr. Bastian, on Dec. 29, he had marked left hemiplegia, with rigidity and pain on movement. He could just move the left shoulder but not the elbow or hand. He could move his leg about a little, but could not stand at all, falling over at once to his left. There was definite slight left hemianæsthesia, and complete left hemianopia for all forms nearly up to the mid-line, but not including the fixation point. Vision was $\frac{6}{24}$ in each eye. Refraction, 2 D hypermetropia in each eye. No other affection of the special senses. His colour fields could not be tested owing to his being congenitally colour-blind. There were marked gouty changes in his left wrist joint and right foot, with visible chalk stones, and tophi in his right pinna. The arteries were much thickened, and there was a trace of albumen in the urine. The most probable diagnosis seemed to be a hæmorrhage in the posterior part of the internal capsule on the right side. After he had been in hospital three weeks, it was found that the hemianopia was not complete. He still did not flinch from an object darted at his eyes from the left, but he could tell when a large object as a hand was moving at 30° from the centre, though a light was not perceived until it had reached an angle of 45°, and he could not count fingers at all to the left of the mid-line. Two months after admission his left visual field had improved further, and he could tell when a large object was moving at 80° to his left, and could see fingers moving at 45° and count them at 30°, but in reading he still often missed the first two or three words of a new line. After three months, on April 1, he could see a large object move quite

at the periphery on his left side, and he could see a small disc of white paper and count fingers at 45°.

In Migraine the onset of hemianopia is generally sudden. One instance of this will suffice:

N. H., a woman, about 35 years of age, has been liable to sick headaches since girlhood. When aged 23 she one day suddenly noticed that she could see only the half of anything she was looking at. Becoming alarmed, she ran to a looking-glass and then noticed she could see only the reflection of the right side of her face, as far as the middle of her forehead and nose. This condition lasted about five minutes, and was followed soon by severe headache and by vomiting several hours after.

The pathology of this visual affection is disputed. Galezowski, Du Bois Raymond, Latham, and others ascribing it to vaso-motor spasm, as evidenced by the change of colour of the face. Liveing puts forward the theory of a "nerve-storm," and Gowers (14c) says, "The hypothesis that the derangement is primarily one of nerve cells of the brain enables us better to understand the relation to other neuroses, and especially that to epilepsy, which is occasionally so distinct. . . . These relations, however, make it intelligible that the two should occur in the same subject, and that intermediate forms of nerve disturbance should sometimes be met with."

Dr. Hughlings Jackson (38) speaks of migraine as a sensory epilepsy, the discharging lesion being in the posterior lobes, the ocular disorder and unilateral disorder of sensation as parts of the paroxysm, the headache and vomiting post-paroxysmal. He thinks these sensory epilepsies bear the same relation to hemianæsthesia with hemianopia from disease of the optic thalamus as unilaterally beginning convulsions do to the ordinary kind of hemiplegia from destruction of the corpus striatum.

Sir Samuel Wilks (41), on the other hand, sees no resemblance between migraine and epilepsy. He says they never pass one into the other, they do not occur in the same families or in the same class of persons, and each is relieved by different drugs.

Dr. Auld (36) considers migraine to be a functional

neurosis, and that the existence of a cerebral lesion is not established on satisfactory grounds.

Savage (40) considers that "migraine is undoubtedly a neurosis, occurring in families in which other forms of nervous instability are met with, and it is noteworthy that in some of these cases insanity develops, and that almost invariably with the onset of insanity the tendency to the recurrence of the headache ceases."

H. C. Wood (42), in a clinical lecture on "Epileptoid Migraine" at the University of Pennsylvania, showed a patient who suffered from typical epileptic attacks, with loss of consciousness and tongue biting, always preceded by a visual aura on his right side resembling the well-known fortification spectrum of migraine, which then faded, leaving a spot of darkness which increased until the whole field was obscured. Later, this patient suffered from minor attacks without loss of consciousness, preceded by a similar aura lasting twenty to forty minutes, and followed by violent temporal headache, nausea, and depression, in fact attacks indistinguishable from migraine. In spite of this, Dr. Wood has seen only two other cases in which there was any reason for suspecting the co-existence of epilepsy and migraine.

J. W. Gill (37) mentions two cases of recurring attacks of homonymous hemianopia, one occurring in a miner aged 20, who, after returning home, suddenly noticed he could see only the left half of any object. "At the same time an indescribable anxiety seized him, quite out of proportion to his concern at losing half his field of vision, and at the same time he felt a fulness at the epigastrium, as though he had wind on the stomach." After lasting a few minutes, a vibratory movement appeared in the blind field, gradually increasing, and then fading, until in about fifteen minutes his sight was again normal. Scarcely any headache followed the attack, and he has had similar attacks, at intervals varying from a few days to a few months, for the last forty-five years, though not so intense of late years, and varying in intensity and in duration from a few minutes to an hour. There was no history of epilepsy or of any nervous disease in the family.

Another case had similar attacks at an interval of four years, and then recurring at intervals of a few days. Such cases are indeed strongly suggestive of a close relationship between epilepsy and migraine. Gowers (48) has "met with cases in which epilepsy succeeded migraine, and the epileptic fits seemed, as it were, to grow out of the attacks of migraine, being preceded by such sensory symptoms as had occurred before the attacks of headache. . . . In rare cases of epilepsy, again, a visual aura may consist of fortification-spectra with colours, and even, as I have known, with hemianopia. In one such case the visual disturbance lasted ten minutes, occurring sometimes alone, sometimes with transient loss of consciousness, sometimes with a convulsive attack." He goes on to say (49), "In most cases of epilepsy with a visual aura, this is brief, lasting only a few seconds, while the visual disturbance in migraine lasts twenty minutes to half an hour."

During the last eighteen months I have had the opportunity of studying four patients, who have had between them eleven attacks of transient hemianopia, each attack lasting for several hours, and generally accompanying unilateral convulsions, but in one case definitely preceding a typical epileptic fit. These cases form, I think, a further link in the relationship of epilepsy to migraine.

J. R., a butcher, while walking to his work one morning in September 1895, his eyesight suddenly failed, and when he reached the shop he could see scarcely anything. He became dizzy, was put on a sofa, but rolled off, losing consciousness for five hours. He thinks he had no convulsion, and on recovering consciousness his sight had returned. Since then his sight has failed similarly on several occasions, and he has had numbness and tingling in both arms and in the left leg during the attacks, but no twitching. A history of syphilis acquired twenty-five years ago was obtained.

On admission into the Queen Square Hospital in March, 1897, under Dr. Beevor's care, he seemed fairly well, no weakness in any limb nor any anæsthesia, gait and coordination normal. Vision $\frac{6}{12}$ in each eye, visual fields normal, ocular movements, pupils and discs normal. Knee jerks normal and equal.

On March 25, on getting up at 5.30 a.m. he felt dizzy, as

though he should be sick, and his sight seemed blurred. He went out for a walk about 11 a.m. with the other patients, and thinks his sight was gradually getting worse up to this time. While out he noticed numbness, first in the left hand and then in the right, spreading thence to the feet, with a tingling sensation running up from the left knee to the hip. He was observed not to notice objects or people on his left, and to bump up against them. When I saw him on his return at 12.15 p.m. he was complaining of feeling unwell, with pain across the forehead, and dimness of sight. On testing his fields of vision at once there was found to be complete left hemianopia for light, form, and colour, and with most careful testing, by the method above described, neither a small piece of white paper nor the flame of a match was seen by either eye on the left side until the fixation line was reached. Charts were at once taken with McHardy's perimeter, with the same result, showing scarcely any contraction of the remaining half-fields for white, though the colour fields were markedly constricted.

His pupils were normal, with no hemianopic pupil reaction, and they reacted well to light and accommodation.

The ocular movements were normal, except for slowness in turning the eyes to the left.

Special senses. No affection of smell or taste, but he could not hear a watch on the left side on contact, though on the right side he could hear it at four inches. Vision was as before, $\frac{6}{12}$ in each eye. No word blindness. Slight weakness of the left arm also was noticed, and anæsthesia to light touches of the left hand and fingers. There was no weakness of the leg, and his speech and articulation were normal. Intelligence and memory perfect. Knee jerks normal; no increase of either wrist jerk.

He was put to bed, and given an injection of 3 minims of liq. Strych. with a dose of half an ounce of brandy and a teaspoonful of sal volatile. Three hours later, at 3.30 p.m. he had a typical epileptic fit, with convulsion of both arms and of the face, and total loss of consciousness, lasting for five minutes, though afterwards he knew nothing of it. At 6 p.m. two-and-a-half hours after the fit, the left hemianopia was found still complete for all forms up to the fixation point, as before. The numbness, anæsthesia and weakness of the left hand had passed off, and he could then hear a watch two inches distant from his left ear. He still had pain across the forehead and back of head, worse on the right side, with some tenderness behind the right ear and on the right side of the occiput.

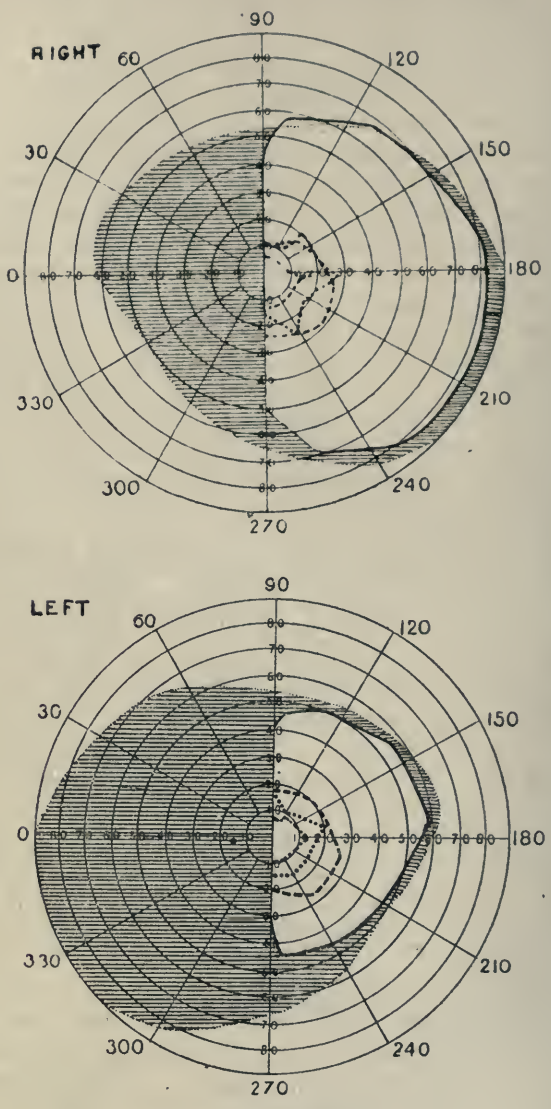


FIG. 7.

J. R.—Case of transient hemianopia, with constriction of remaining colour fields. The dividing line cuts the fixation point.—*March 25th*, 1897.

Next morning at 9 a.m. the left hemianopia was clearing up. He could then see a large object, as a hand, moving at 80° on the left from the line of fixation, though he could not count fingers further out than 25°, nor recognise any colour until it nearly reached the fixation line on the left side, though his colour fields on the right side were again normal. A small white disc, 1 cm. in diameter, such as is generally used with the perimeter, he could see at 30° to his left. In the evening his visual fields had again become perfectly normal. The hemianopia was therefore absolute for at least twelve hours, and about the same time elapsed before it completely cleared up. Again, on April 5, at 7 a.m. his vision became impaired, and when seen at 12 noon, there was incomplete left hemianopia. He could see a hand moving at 80° to his left, and a small white disc at 45°, though he could not see the red head of a pin, nor recognise its red colour, until near the fixation line. The object, however, became bright and clear before the fixation line was reached, an evidence of the escape on this occasion of the centre for the macula in the cortex, or else of its earlier recovery. At 4.30 p.m. his left field was still much impaired, and he could not distinguish between one and two fingers held up further out than 20° to his left, though he himself, and most people I have tested, can ordinarily in a good light, distinguish between one and two fingers held up at 80° from the fixation line. Next morning his visual fields were again normal, no fit having followed the visual disturbance on this occasion. He was then ordered by Dr. Beevor 30 grains of pot. brom. every night, since when he has had no further attack.

He now says that the attacks he has had of dimness of sight during the last eighteen months have been of precisely similar character, though less severe, and occurring about once a month. Their resemblance to the hemianopia occurring in migraine, being followed by headache, is remarkable, though they persist much longer, and the definite association with epilepsy in this case points strongly to the supposition that in this transient hemianopia we have evidence of a primary sensory discharge in or near one half vision centre, generally proceeding no further, but occasionally overflowing and giving rise to an epileptic fit with loss of consciousness; the hemianopia thus appearing as an aura, though lasting very much longer than any previously de-

scribed aura in epilepsy, and persisting for hours after the fit.

This long duration of the visual loss, especially in view of the result of the *post-mortem* in the case next to be described, is strongly suggestive of an organic lesion, hæmorrhage or softening, in the neighbourhood of the right cuneus, though the entire recovery of the visual field would preclude actual damage to the cuneus itself or the optic radiations.

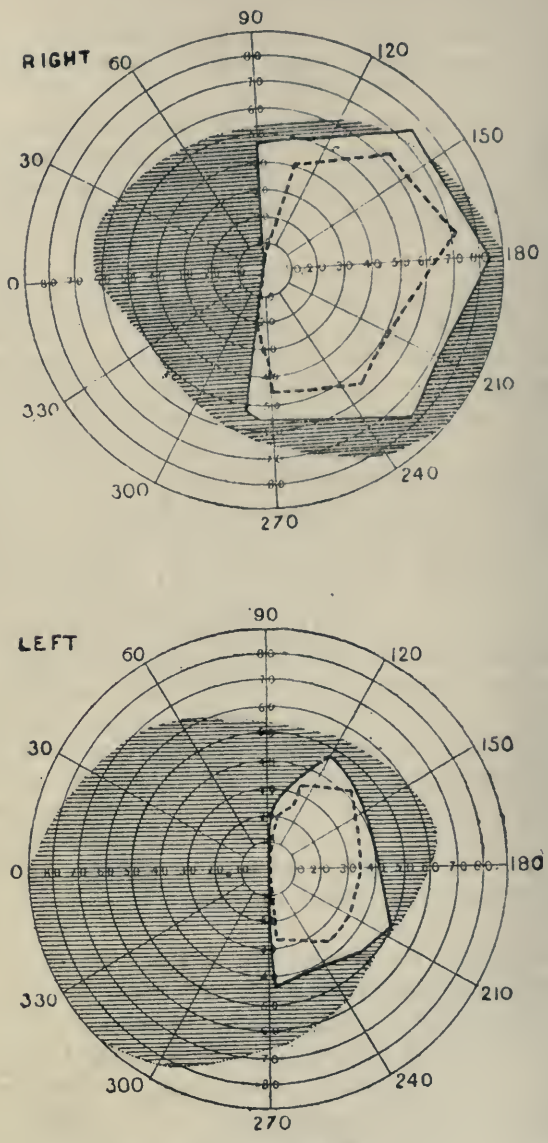
A somewhat similar case, though of greater value since its completion by an autopsy, is that of a woman A. B., aged 53, who was admitted into the Queen Square Hospital in August, 1896, under the care of Dr. Hughlings Jackson. On her admission she presented the typical appearance of myxœdema, though eleven years previously, in 1885, she had been under Mr. Lawson's care at Moorfields for exophthalmic goitre, and an old photograph taken at that time shows the characteristic goitre and proptosis. These symptoms had persisted for about a year, when one night, ten years ago, she had a sort of fit, described as a "throbbing of the muscles," with partial loss of consciousness. Since that time she has had a similar fit about every three months, and for the last few months every month. Six months ago she had two severe fits with struggling. The history excluded the possibility of venereal disease. During the last two years her facial expression had been getting heavy, and slowness of speech had been noticed during the last twelve months. Three months ago, after a fit, she was delirious, and had aural and visual hallucinations. When seen in August, 1896, she appeared to be a typical case of myxœdema, with heavy expressionless features, malar flush, dry, scaly skin, hair coarse and dry, speech slow and drawling, with very impaired memory. Her weight was 9 st. 2 lb., though formerly it had been 14 st. The temperature was generally subnormal. No thyroid could be felt in the neck. She was weighed daily for a week, and then treatment was commenced by giving her thyroid tabloids by the mouth, at first one of Burroughs and Welcome's tabloids every other night, the dose being increased early in September to two tabloids daily. Her weight fell 3 lbs. at the end of the first week of treatment, and continued to fall until December 3, when she had lost 2 st. in weight. In the middle of December the dose of thyroid was reduced to half a tabloid every other night, which it was found was all she could bear, as when taking more she lost all appetite.

Briefly, she improved steadily under the treatment, and the symptoms of myxœdema gradually disappeared, and she became brighter and more cheerful, though her memory was always bad for recent events. When discharged on March 28th, 1897, she weighed 8 st. 4 lbs., being 12 lbs. less than on admission.

Between October, 1896 and January, 1897, she had five fits, all of them left sided, and very similar in character, so that a description of the first will apply fairly closely to the succeeding attacks.

On October 20, at 1 p.m., she was suddenly seized with left-sided convulsions, her head and eyes being turned to the left, with twitching of the left hand and foot. Consciousness was only partially lost, and she could turn her eyes to the right when loudly told to do so. The twitchings of the left hand and foot lasted for half an hour, and were followed by complete paralysis of the left arm, and weakness of the left leg. There was no hemianæsthesia nor any affection of taste, smell, or hearing, but there was complete left hemianopia, for light as well as for form and colour, the dividing line between the seeing and the blind halves, after the most careful testing, being found to pass exactly through the fixation point. The paralysis of the left hand was complete for five hours, but was being recovered from during the same evening. Next morning at 11.30 the left hemianopia was still complete, and charts were taken with McHardy's perimeter. She was otherwise fairly well, except for some headache. Her vision was $\frac{6}{24}$ in each eye, much the same as before the attack. Towards that evening the hemianopia began to pass off, vision improving from the centre towards the periphery. Two days after the attack there was scarcely any contraction of the left field remaining, and on the third day her visual fields were again perfectly normal.

As in the case previously related, there was absolute left hemianopia lasting for several hours, in her case for twenty-eight hours, and then vision gradually cleared up again to normal. In the case of the man, J. R., the visual disturbance was found a considerable time before the onset of the fit, and amounted to a definite aura, but though in the case of the woman A. B. the hemianopia was not found until attention was drawn to her case by the convulsion, yet in view of the much longer time that the hemianopia lasted as compared with the paralysis of the left arm and leg, it seems



----- Blue field.

FIG. 8.

A B.—Case of left transient hemianopia. No contraction of remaining colour fields. The dividing line cuts the fixation point.—*October 21st*, 1896.

reasonable to suppose that the primary discharge in her case also commenced in or near the half vision centre on the right side and spread thence to the Rolandic area.

She had similar fits on four other occasions while in hospital, on November 19, November 28, December 22, and on January 20. Both she and her friends also were quite sure that all the fits she has ever had have been of the same character. In each there was slight twitching of the left arm and leg, followed by temporary left-sided weakness and complete left hemianopia, which lasted for about twenty-four hours, and then cleared up again in two days to normal vision. In each instance the dividing line between the seeing and the blind halves was found by careful testing to cut the fixation point, and this observation was confirmed by Mr. Marcus Gunn, who saw her during the hemianopic stage on December 22. On two occasions there was also noticed after the fits temporary weakness of the lower part of the face on the right side, with partial aphasia and word blindness. She could never write anything, not even her own name, during all the time she was in hospital, though formerly she could write well. She was right-handed.

The charts of her visual fields, taken during the stages of hemianopia, showed scarcely any contraction of the remaining half fields, nor were the colour fields at all contracted as in the case of J. R. After the fit on January 20, Dr. Taylor ordered her 30 grs. of bromide of potassium every night, and she never had another fit up to the time of her death on April 20. She was discharged on March 28, and continued taking the bromide every night, and half a thyroid tabloid every other night while she was at home. She soon contracted influenza and pneumonia, and died three weeks after leaving hospital.

Autopsy.—April 23, sixty-seven hours after death. An examination of the head only was allowed. The brain, though soft in places, appeared perfectly normal. There was no thickening of the membranes, and the pia mater stripped readily. Close examination of the cuneus and of the motor convolutions on each side revealed nothing abnormal, nor was any softening seen on section of either cuneus. The brain was then hardened in Müller's fluid, and on dissection two months later, a small cyst was found in the lower part of the right quadrate lobule. The cyst

was the size of a very small marble, about a quarter of an inch in diameter, and was evidently the result of an old hæmorrhage, containing granular yellowish pigment. It was placed just in front of the parieto-occipital fissure, in the white matter at the depth of about one third of an inch from the surface, on the internal aspect of the quadrate lobule. Microscopical sections of the surrounding brain substance showed no organised cyst wall, but sections stained by Marchi's method showed numerous degenerated white fibres. The nerve cells appeared quite normal, and no other lesion could be found in the brain.

The origin of her attacks is thus explained. In all probability her first fit which occurred ten years ago, when her symptoms of Graves' disease were well marked, was due to the occurrence of the hæmorrhage, and the recurrence at varying intervals since, of left-sided convulsion were due to instability of surrounding grey matter, set up by the irritation of the softened and necrosed white matter beneath.

The situation of the lesion, in such close proximity to the right cuneus and optic radiations, will account for the absolute left hemianopia which was found always accompanying the fits, and which was, no doubt, one of the initial occurrences, probably preceding the motor convulsion, and due to early loss of function of the half-vision centre.

Comparing then her case with that of the man previously related, we have an illustration of two different kinds of fit, each started by a "discharge of nerve force," whether originating in the nerve cells themselves or in the interfibrillary network beyond them, in or near the half vision centre, or right cuneus. In the man's case, a general convulsion with early total loss of consciousness, that is to say, an ordinary epileptic fit resulted, while in the other the discharge was more localised to one side of the brain, affecting the motor convolutions chiefly, and giving rise to unilateral convulsion with only partial loss of consciousness, or a Jacksonian fit. It is well known that aphasia, especially word deafness, may occur as a temporary symptom in migraine, and though the occurrence of a convulsion is said to exclude migraine, yet I think the attacks in this second

case bear some similarity both to migraine and to ordinary epilepsy. It is, perhaps, a significant fact that a daughter of this patient, aged 18, suffers from typical migraine with right sided visual spectra, though, so far as I am aware, without hemianopia.

Another case which closely resembles the last is that of a woman, E. B., aged 40, who was admitted into the Queen Square Hospital, under Dr. Bastian, in March, 1896. She had a history of headache, and of numbness of the left hand for five years, and during the last eighteen months she had three left-sided convulsive attacks. There was no history of syphilis. On admission she had slight left hemiplegia, chiefly affecting the arm, with marked wasting of the intrinsic muscles of the left hand. Her temperament was nervous and excitable. On April 1, at 7.30 a.m. she had a fit, preceded for half-an-hour by numbness in the left fingers, excitability and continual talking, with a flushed face and widely open eyes. Her left hand, left arm and leg then began to shake, and she felt a sensation of twitching in the left side of the face, though none was observed. The twitching persisted longest in the leg, and was followed, one and-a-half hours from the commencement of the attack, by a general convulsion with loss of consciousness, cyanosis, and turning of the eyes to the right. When I saw her at 11 a.m. she could move her left shoulder and elbow, but not the wrist or fingers, and there was distinct loss of sense of position in the left fingers, wrist and elbow. There was partial left hemianæsthesia to touch and pin prick, scarcely more marked on the hand than elsewhere. There was also complete left hemianopia, reaching close up to the mid line, though, this being the first case of the kind I had seen, I did not specially test the relation of the dividing line to the fixation point, beyond taking perimeter charts. There was no affection of either smell or taste, but her hearing in the left ear was much diminished, being $\frac{1}{40}$, with diminished bone conduction. Her pupils were equal and normal, and vision was R. V. $= \frac{6}{60}$ J6, L. V. $= \frac{6}{12}$ J1 nearly. There was no weakness of the left leg, but the left knee jerk was much increased, with left ankle clonus. Four hours later she had recovered a good deal of power in the left fingers and wrist, the left hemianæsthesia had nearly disappeared, and the sense of position in the fingers and hand had improved. Her hearing had then improved to $\frac{6}{40}$ on the left side, and the left hemianopia was also clearing up. When a black pen was moved inwards from the periphery she could tell

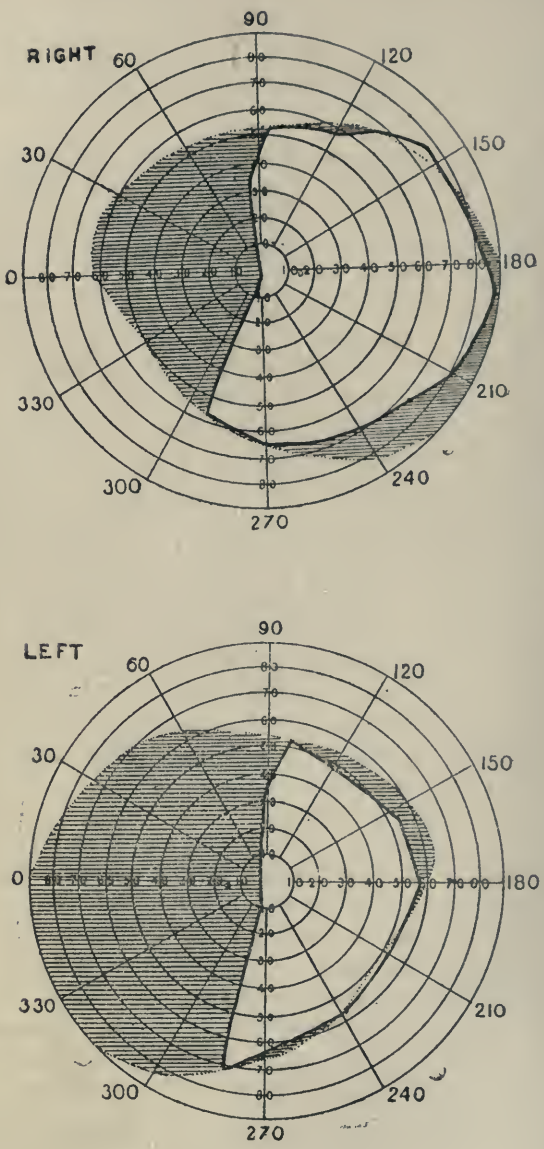


FIG. 9.

E. B.—Case of transient hemianopia.

something was moving at 60° from the centre, though she could not recognise the object until it was within 30°, and she could not distinguish between one and two fingers held up at 45°. By the next morning her visual fields had entirely recovered. Her hearing was $\frac{40}{40}$ each side, and there was no weakness or hemianæsthesia left.

On April 29, after complaining of headache the previous night, she had an attack at 7 a.m., commencing by numbness in the two ulnar fingers of the left hand, spreading up the arm to the shoulder which then commenced to shake, followed by flexion of the forearm and twitching of the fingers of the left hand for a few minutes. There was no movement of the head or eyes, no general convulsion, nor any loss of consciousness. An hour later she had a left sided visual spectrum, commencing as a bright point on her left side. This rapidly enlarged to a ring, the size of the palm of a hand seen at a distance of three feet, being coloured brilliantly in green and gold, and pulsating synchronously with her heart beat, which she could feel as a throbbing sensation all over her body. This was followed by visual hallucinations of people in wheel chairs, wheeling them from left to right as far as the mid line, all brilliantly coloured also in green and gold. On this occasion there was no hemianopia or contraction of the field, no hemianæsthesia or affection of any special sense, except hearing, which was again diminished temporarily on the left side to $\frac{6}{40}$. The visual spectra lasted for about half-an-hour, and did not recur again whilst she was in the hospital, though she says she has seen occasionally coloured curtains appear to come down in front of her, always to the left.

In this case the left hemianopia found after the first fit persisted for a much shorter time than in any of my other cases, being complete for only four or five hours, and then clearing up gradually *pari passu* with the disappearance of the left sided deafness, weakness, and hemianæsthesia. This fact, in view of the definite symptoms pointing to a lesion of the motor tract on the right side, probably in the cortical centre for the arm, make it probable that the primary focus of discharge occurred in the arm or hand centre and spread thence to the half vision centre on the same side, paralyzing its functions, and producing left hemianopia in the same way as the temporary weakness of the left arm and hemianæsthesia followed the left sided convulsion.

That this was the real sequence of events is made more probable by the nature of the second attack, nearly a month later, in which numbness of the left hand and arm was followed by convulsion of the left arm, and, after another hour's interval, by a left sided visual spectrum and hallucinations. Her description of this spectrum closely resembles that so frequently found in migraine, though the sequence of definite hallucinations of moving figures in the left field, all brilliantly coloured in green and gold, is, I think, unique. This visual spectrum and the hallucinations, occurring without hemianopia, would probably indicate that the higher centre in the angular gyrus was also affected by the progress of the epileptiform discharge, rather than the half-vision centre only. This case again forms a curious link in the resemblance and relationship of migraine, Jacksonian fits, and ordinary epilepsy. Jacksonian convulsions, however, like ordinary epileptic fits, are but rarely accompanied by hemianopia, and in several other cases of hemiplegia accompanied by recurring unilateral convulsions, sometimes so severe as to produce total loss of consciousness, and also in post-hemiplegic epilepsy, I have never found any affection of the fields of vision, even immediately after the fits. It is probable, therefore, that the half vision centre is not liable to become paralyzed by even a severe discharge taking place in neighbouring centres unless (1) it is already slightly damaged, or (2) if hypersensitive and prone to spontaneous discharge as in migraine.

The temporary unilateral deafness which followed both attacks in this case, and also in the case of J. R. previously related, may be due to similar spread of nerve discharge temporarily paralyzing the auditory centres.

In two other cases I have seen there has been permanent partial hemianopia, due probably to cortical lesions, in each case associated with recurring convulsions and temporary complete blindness of the half fields on one side.

W. B., aged 54, a gas engineer, was admitted into the Queen Square Hospital under Dr. Beevor on December 30, 1896, suffering from partial right hemianopia, word blindness and agraphia. During the last eight months he has had a fit every

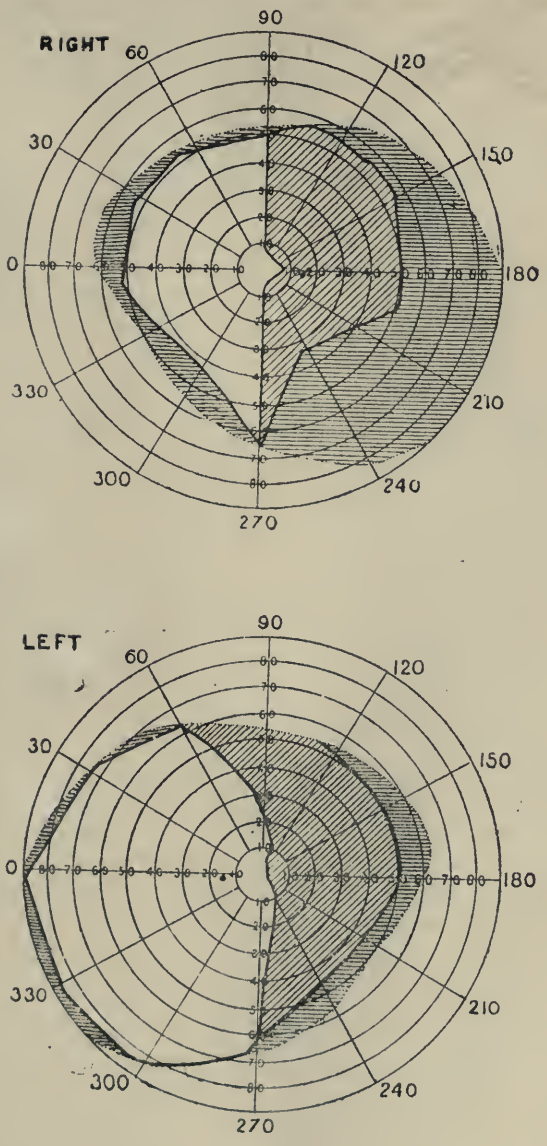


FIG. 10

W. B.—Case of permanent partial right hemianopia. The area of indistinct vision is indicated by lighter shading. Fields for white spot 1 cm. in diameter.—*January 3rd*, 1897.

month, the last occurring a fortnight before his admission. He says he loses consciousness early in the fit and his friends say he is "drawn" on the right side during the attack, and afterwards his right arm and leg are weak for a time. He knows when the fits are coming on by a sensation of an electric feeling rising from his feet and spreading all over him. He has had frequent visual hallucinations of faces he knows, especially of his children, animals, &c., always on his right side, sometimes before the commencement of a fit. He denies even exposure to syphilis. On admission his heart was very irregular in action, though there was no murmur to be heard, and he has never had rheumatism. There was no weakness of either arm or leg, the knee-jerks were slightly increased and equal, and his gait natural. He was quite sensible, though his manner was generally a little odd and exaggerated, possibly owing to his difficulty in expressing himself. He had great difficulty in reading, though he understood all that was said to him, and he could pick out any object asked for at once, though he had some difficulty in naming them, but he could write scarcely anything. The eye movements, pupils and discs were normal, and his vision R. V. $= \frac{6}{18}$, L. V. $= \frac{6}{9}$. There was incomplete right hemianopia, the inner part of each right field appearing blurred, and an object not seeming distinct until near the fixation point. The outer part of each right field beyond 50° was completely blind. Colours were recognised on the left side from 20°—30° from the centre.

His vision improved during the first three weeks, so that he could tell when a hand or a large object moved even quite at the periphery on the right side, and he could count fingers at 45°. On January 24, he had two severe epileptic fits, with half-an-hour's interval between them. For some hours previously he had felt dizzy and unwell and was more incoherent than usual. At 3.30 p.m. the fit commenced, and Dr. Stewart's account is that it began suddenly with turning of the head and eyes to the right; he then became completely rigid, and the right side of his face twitched, followed by general convulsion. There was early and complete loss of consciousness, with absence of the corneal reflex, and dilated insensitive pupils. Slight double ankle clonus was present. The fit lasted two minutes, and half-an-hour later he had another precisely similar fit. When I saw him the next morning, there was complete right hemianopia up to the middle line, except for a bright light which was perceived nearly out to the periphery. He could not see any object or a

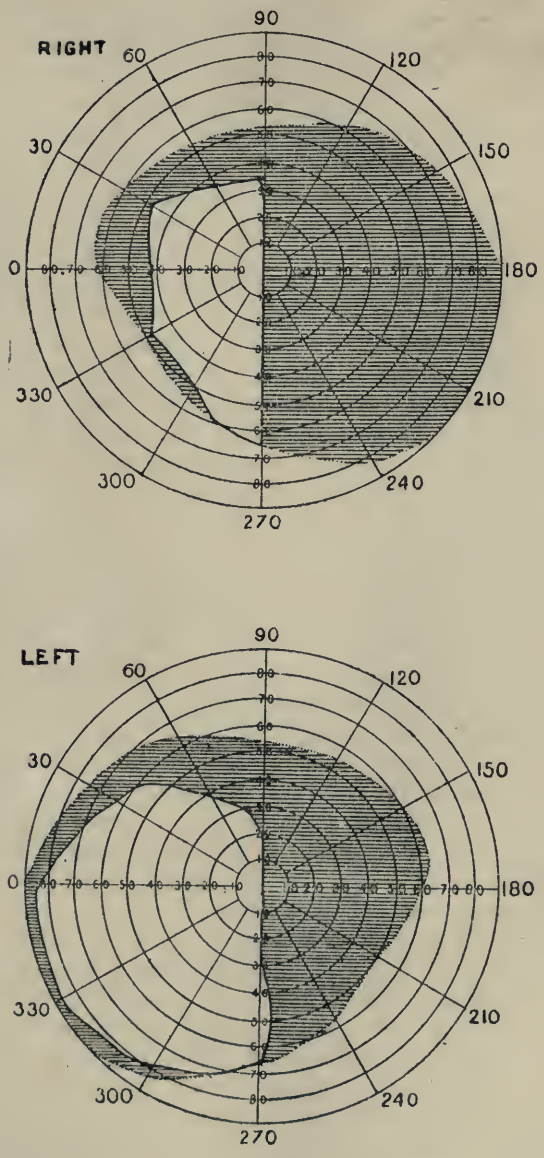


FIG. 11.

W. B.—Case of transient right hemianopia, the dividing line cutting the fixation point.—*January 25th*, 1897.

piece of white paper move until it exactly crossed the line of fixation, which observation Dr. Beevor confirmed at two o'clock in the afternoon. In addition, he was completely word blind, and could not name nor pick out objects, except coins, though he understood nearly all that was said. Late in the afternoon the hemianopia began to clear up slightly, vision returning first at the centre. The next day he could see a large object move at 30° from the centre on the right side, but he could not see a small piece of white paper until near the mid line. The visual field gradually enlarged again, and on the fifth day after the attack he could see a hand move at the periphery on the right side, and could count fingers at 45°, and could recognise colours at 30°. On February 20, he had an attack of localized convulsions on the right side, commencing in the thumb and finger, and spreading to the elbow and shoulder. There was no turning of the head or eyes, nor any loss of consciousness. The convulsions lasted for two hours, and, on testing him while they continued, there was found complete right hemianopia, this time for light as well as for form and colour, the dividing line cutting the fixation point. This remained complete until the next day, when it began to clear up gradually as before. The convulsions were followed by some weakness of the right hand and arm, which had disappeared on the following day, considerably before the recovery of the visual field. Just a month later, on March 23, he had another similar attack of localized convulsions on the right side without loss of consciousness. In the afternoon he complained of feeling unwell, and went back to bed. I saw him at once, and noticed slight twitchings of the right thumb and fingers. On testing the visual field immediately, there was found again absolute right hemianopia, the dividing line in each eye cutting the fixation point, as before. His right hand and arm were very weak, and he could not grip at all, though the leg was as strong as usual. He was almost entirely aphasic, and could not name anything, nor read a word, though he could ejaculate a few sentences; and he was also partially word deaf. He recognised, however, the difference in value between coins. The twitchings of the hand became more violent, and after continuing for nearly two hours were stopped by a dose of 25 grs. of chloral hydrate and 20 grs. of bromide of potassium. On the next day at noon he was still completely hemianopic on the right side, but the aphasia and word blindness had improved considerably. By the following morning, the right visual field had again much improved, and he could see a hand move at the periphery. Dr. Beevor then

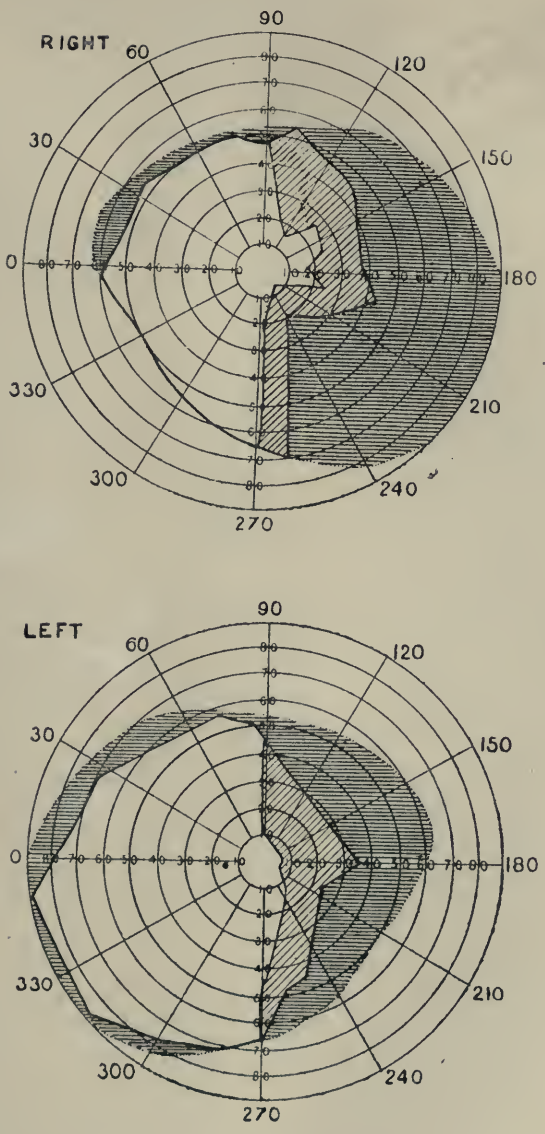


FIG. 12.

W. B.—Partial right hemianopia, persisting after an attack of transient complete hemianopia.—*February 2nd*, 1897.

Fields for white spot 1 cm. in diameter the area of indistinct vision being indicated by lighter shading.

ordered him 30 grs. of bromide of potassium every night, and since then up to the time of writing (June 1) he had not had another fit.

While in hospital he thus had three separate attacks, which on the first occasion resembled ordinary epileptic fits, the two latter being typical Jacksonian convulsions without any loss of consciousness. Each attack was, however, accompanied by complete right hemianopia, which was found during the last attack to be present as soon as slight twitching of the hand had begun, though he was not tested on the first occasion until eighteen hours after the fit, and on the second occasion not until two hours after the commencement of the convulsions. Moreover, the patient is quite certain that he has had similar disturbance of vision accompanying each of the fits he has had at home.

In view therefore, of (1) the probable early occurrence of the hemianopia in all the attacks (2) its long duration, being complete for about twenty-four hours, and then the visual field gradually improving again to its previous condition, and (3) the partial persistent impairment of the right half fields, which is evidence of some lesion in the left half vision centre or visual fibres, it seems probable that in this case again we have an instance of a primary epileptic discharge near the left cuneus, temporarily but completely abolishing its function. In his case this led on some occasions to total loss of consciousness with general convulsion, or an ordinary epileptic fit, while on two occasions Jacksonian fits have followed, an illustration, I think, in the same patient, of the possible variety in the spread of an epileptic discharge originating from the same focus.

An interesting feature of the case was the complete word blindness, and slight word deafness found after the attacks, which, taken with the persistent partial word blindness and agraphia, indicates some lesion of the visual word centre in the left angular gyrus. This might be a separate patch of softening, or what is perhaps more probable, the angular gyrus may be partially damaged by softening, which extends deeply into the white matter, and so has damaged the optic radiations which run in close proximity, thus causing the

partial persistent hemianopia. The primary epileptic discharge may then be assumed to take place in the higher visual centre, and, spreading thence cause the temporary complete hemianopia by paralyzing the half-vision centre on the same side. Word-deafness and word-blindness in association with *petit mal* have been recorded by Dr. Hughlings Jackson (46).

A history resembling those of the previous cases, I met with recently in an out-patient of Dr. Luff's, whom I had the opportunity of examining at St. Mary's Hospital.

A woman, E. W., aged 51, while complaining of some gastric trouble, mentioned that she had numerous attacks beginning with "dazzling in the eyes" on the left side, followed by a partial loss of sight, and by tingling in the left arm and leg, and convulsive twitchings of the left hand and forearm. She then used to feel a curious sensation of swelling in her stomach followed by loss of consciousness.

Though I have not seen her in an attack I have little doubt that her case would prove to be a similar one to those previously related, and that the partial loss of sight, if investigated at the time of an attack, would be found to be left hemianopia.

Two other cases I have seen in which transient hemianopia was associated with unilateral convulsions, and also with severe epileptic fits, in one typical case of general paralysis, and in another obscure case which presented some symptoms also resembling general paralysis.

This latter case, J. H., a man, aged 36, had been discharged from the army in April, 1896, on account of his eyesight and inability to do his work. He had been in the army eighteen years, and had contracted syphilis when he first joined. The sight of his left eye had always been weak. He was fairly well until the end of the second week in November when his left hand and arm became weak, and he noticed that he bumped up against people on his left side. On admission to the Queen Square Hospital under Dr. Ferrier, on November 18, he was found to have marked weakness of the lower half of the left side of his face, and also of his left arm, though less marked. He could

walk fairly well, and there was no weakness of either leg. There was marked tactile anæsthesia and loss of sense of position in the left hand, wrist and forearm; and also complete left hemianopia, and slight left hemianæsthesia, but the exact position of the dividing line could not be ascertained owing to his drowsiness and insufficient answers. His vision was R. V.$=\frac{6}{9}$, L. V.$=\frac{6}{18}$. There was compound hypermetropic astigmatism in each eye quite sufficient to account for the lowered acuity of vision. The optic discs were normal. There was also marked deafness of the left ear, with diminished bone conduction. Knee-jerks increased, L > R. Pupils unequal, L > R, neither reacting to light, though they did so to accommodation. The ocular movements were normal. He had a fit two days after admission, commencing with clonic contractions of the masseters, and movements of the tongue, palate and lips. There was no loss of consciousness, and he could answer questions while the convulsions were going on, which lasted about two minutes. He had numerous fits, closely resembling this one, during his first week in hospital, and then, during two days, he had three batches of strong epileptic fits with early total loss of consciousness and general convulsion, with cyanosis. He was placed on large doses of mercury and iodide of potassium, and had no more fits after the first ten days in hospital. He slowly began to improve, his mental condition became brighter and his left arm and face became again as strong as the right side. At the same time the left hemianopia began to improve gradually, and by the time of his discharge in April, he could tell when a hand was moved quite at the periphery on the left side, and he could count fingers at 45°. As he improved he began to show symptoms suggestive of general paralysis, the pupils remained unequal and inactive to light, his articulation was defective, with a tendency to clip the ends of words, and there was characteristic tremor of the tongue and facial muscles, with overaction of the frontalis and corrugator supercilii on showing the teeth. His manner too became slightly exalted, and he declared he was never better in his life.

The case curiously resembled that of a man, G. S., under the care of Sir William Gowers in the autumn of 1895, an account of which I published in the *Lancet* (23). In each case there was weakness of the left hand and arm, with slight left hemianæsthesia and left hemianopia. Both had localised convulsions affecting the left hand and the tongue, palate and face, and both had also batches of strong epileptic

fits with complete loss of consciousness. Both, moreover, bore a certain resemblance to cases of general paralysis.

In another case of a man, H. S., aged 40, a typical case of advanced general paralysis, I had the opportunity of watching two attacks of localized convulsions on the right side, each being followed by complete right hemianopia lasting for about ten minutes, though there was no hemianæsthesia and only slight motor weakness. He could not answer questions, but there was no palpebral reflex to a finger darted at his eyes from the right side, and he gave no sign of seeing any object approaching him on his right side until it crossed the middle line. In about ten minutes he began to perceive objects again to his right. Dr. Stoddart at Bethlem Hospital, and Dr. Cole of Moorcroft Asylum, have also informed me that they have found hemianopia, each on one occasion, in a case of general paralysis after an attack of unilateral convulsions, the hemianopia being on the same side as the convulsions, and disappearing completely after a short time.

I cannot help feeling, therefore, that if more cases of general paralysis were examined for this point after the fits, hemianopia would not infrequently be found, lasting for a longer or shorter interval. In general paralysis the destruction of the cortex is widespread, and the occurrence of transient hemianopia after the fits would be accounted for by the process beginning to affect the half vision centre, the primary explosion of nerve force originating the fit, whether occurring in the cuneus or spreading to it from the motor area, causing temporary exhaustion of its function. In Jacksonian epilepsy, or localized convulsions due to a limited lesion in the Rolandic area, I should not expect to find transient hemianopia, though I think it would be advisable to test all cases for it. I have not had the opportunity of testing this point in any cases of localized convulsions due to uræmia, in which condition I should think it not unlikely to occur. Amaurosis is well known as a transitory symptom, sometimes associated with uræmic convulsions, and it seems to me quite possible that some cases, if tested, might be found to develop transient hemianopia.

Conclusions.

(1) That hemianopia, rarely binasal, more commonly lateral and left sided, with accompanying constriction of the remaining half fields, may occur as a temporary phenomenon in hysteria.

(2) That hemianopia due to a vascular lesion of the cuneus, of sudden onset, may commence with marked loss of sight, sometimes amounting to complete amaurosis, and due probably to inhibition of the remaining half vision centre.

(3) That the cortical half vision centres are not subdivided into centres for light, form, and colour respectively, and that hemiachromatopia may be due to a lesion anywhere in the visual path between the chiasma and the cortex.

(4) That quadrantic hemianopia, though strongly suggestive of a cortical lesion, may sometimes be due to a lesion in the internal capsule.

(5) That the macular region of the retina is invariably supplied with nerve fibres on the same plan as the rest of the retina, *i.e.*, each side of it from the corresponding side of the brain. That in all cases of absolute transient hemianopia the dividing line between the seeing and the blind halves invariably passes through the fixation point.

(6) That the cortical centre for the macular region in each cuneus is less liable to complete destruction and recovers earlier than the rest of the half vision centre.

(7) That cases of persistent hemianopia in which the dividing line passes to one side of the fixation point, leaving it in the seeing half, are to be accounted for, either (*a*) by the escape or partial recovery of the cortical centre for the macula, or, (*b*) by the acquirement by education of a new fixation point in the retina.

(8) That hemianopic visual spectra of low elaboration, such as red and green lights, or the varieties of scintillating scotoma in migraine, are caused by a discharge in the half vision centre in the cuneus.

(9) That complex visual phenomena of hemianopic type,

such as faces, animals, &c., are elaborated in a still higher visual centre, which possibly is the angular gyrus; their occurrence in the half field only being due to reflex irritation from a lesion generally in or near the cuneus, but which may be in the optic radiations or optic tract.

(10) That double hemianopia does not necessarily cause permanent amaurosis, in many cases the return of a small area of central vision indicating the escape or recovery of the cortical centre for the macula in the cuneus on each side.

(11) That the hemianopia in migraine is due to an epileptic discharge in the half vision centre of one side.

(12) That in many cases an epileptic discharge may originate in, or near, the half vision centre on one side, in some cases proceeding no further, beyond producing temporary hemianopia, in others producing a typical epileptic fit, and again in others giving rise to unilateral convulsions without loss of consciousness.

(13) That transient hemianopia in such attacks may last for twenty-four hours or longer, and may be due to vascular softening adjacent to, but not involving the visual centre or path.

(14) That transient hemianopia is rare in ordinary Jacksonian epilepsy, and is not liable to occur unless the half vision centre be (1) already slightly damaged, or (2) hypersensitive and prone to spontaneous discharge, as in migraine.

(15) That such transient hemianopia not unfrequently accompanies unilateral convulsions in general paralysis, and may possibly occur in uræmia.

(16) That the auditory centre may be similarly paralyzed through spread of the epileptic discharge.

Henschen (14a) has recently published a case of quadrantic hemianopia, the lower quadrants of the field being blind, due to a hæmorrhagic cyst in the posterior part of the pulvinar, which had destroyed the dorsal half of the external geniculate body, but left intact the optic tract and optic radiations, and he infers that the dorsal half of the external geniculate body corresponds to the upper quadrants of the retinæ, just as, in his opinion, the upper lip of

the calcarine fissure corresponds to the same quadrants of the retinæ.

[Since writing the above, the patient, W. B., died suddenly on November 29 of cerebral hæmorrhage. At the autopsy there was found, in addition, a patch of softenin the second temporo-sphenoidal convolution on the left side, about two inches in length, and not quite reaching up to the angular gyrus. The cuneus on each side appears normal, but the brain has not yet been cut, and it is possible that the softening in the subjacent white matter may extend backwards beneath the angular gyrus.]

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NOTE ON MUSCLE-SPINDLES IN PSEUDO-HYPERTROPHIC PARALYSIS.

BY ALBERT S. GRÜNBAUM, M.A., M.D., M.R.C.P.

In 1894[1] Sherrington gave the first account of the structure and function of muscle-spindles based upon physiological experiment. A stimulus was thus given to their more thorough examination in pathological conditions.

Recently Batten has given the results obtained by him in the examination of cases of several nervous diseases, finding, however, except perhaps in tabes, no appreciable changes in the muscle-spindles. So far as I am aware, no account has yet been published of their condition in pseudo-hypertrophic paralysis, and in this disease I have been able to find some changes.

The material was obtained six hours after death, on a cold winter's day, and carefully preserved, but unfortunately, for the most part not histologically examined until over two years later.

In the vascular system there was considerable thickening of the smaller arteries, due, in part, apparently, to hypertrophy of the muscular layer.

In the central nervous system there was marked dilation of the perivascular lymphatics, and also of the blood-vessels. The latter often contained a large number of leucocytes. In the spinal cord were several small hæmorrhages.

In the peripheral nervous system there was degeneration of some fibres of the smaller nerves.

[1] In the last number of BRAIN, both in his historical *résumé* and in the bibliography, Batten erroneously gives the date of Sherrington's first paper as 1895. It should be 1894.

In the muscular system as a whole the changes were of the ordinary character. In the gastro-cnemius very few muscle-fibres remained; the rest were entirely replaced by fat. In other muscles many fibres were atrophied, but some were distinctly hypertrophied. Most of them showed signs of alteration in the surrounding connective tissue, varying from a proliferation of nuclei of the sarcolemma to a deposit of fibrous tissue around the fibre. Several fibres had a distinctly hyaline appearance, and a few showed vacuolation.

The muscle-spindles were for the most part unaffected, but in a few there was a diminution in size of an intrafusal fibre with a deposit of hyaline material around. The nerve fibres supplying them were apparently healthy. In some muscles, *e.g.* gastrocnemius, I could not find any spindles in the sections examined.

Sherrington has shown that division of the nerve supplying a muscle will not produce atrophy or degeneration of the muscle fibres within the spindle. The muscle itself may be totally atrophied, and every nerve fibre going to it have disappeared, yet the muscle fibres within the spindle remain intact; the muscle-spindles are all that are left of the original muscle.

It would therefore appear highly probable that any pathological alteration in such fibres is primary, and consequently also primary in the other ordinary fibres, although commencing much earlier in them. So that, so far as it goes, my observation supports the now generally accepted theory of the intra-muscular origin of the disease.

This theory is also supported by the facts that generally no nervous changes have been detected in the cases examined, and the connective tissue changes do not appear to cause atrophy by compression, for where there is much connective tissue there is a total increase in the size of the muscle. It must be remembered, however, that the nervous and vascular changes, considered to be the primary cause by Erb and Babes respectively, were also present in my case.

I am indebted to Dr. T. D. Acland, under whose care the case was in the hospital, for permission to publish it.

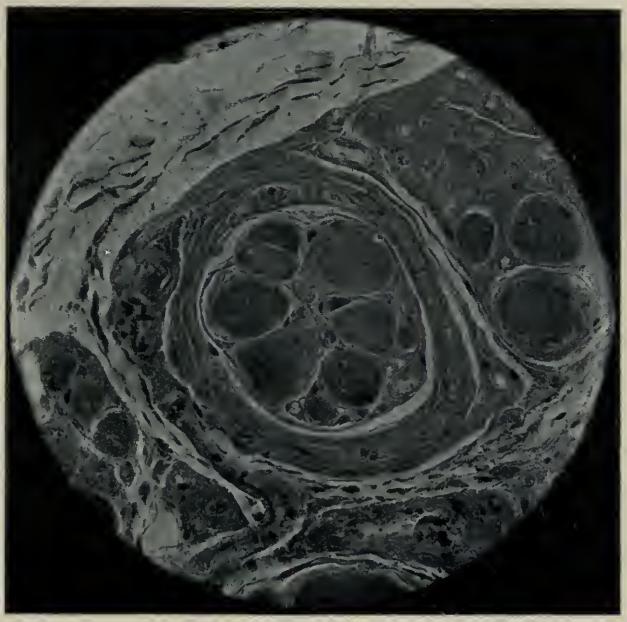

Fig 1.
Muscle-spindle in unaffected portion.

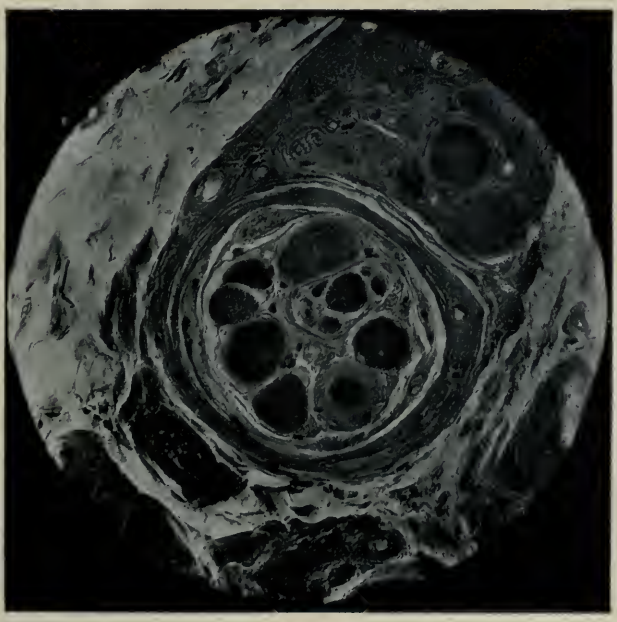

Fig. 2.
The same with affected fibre.

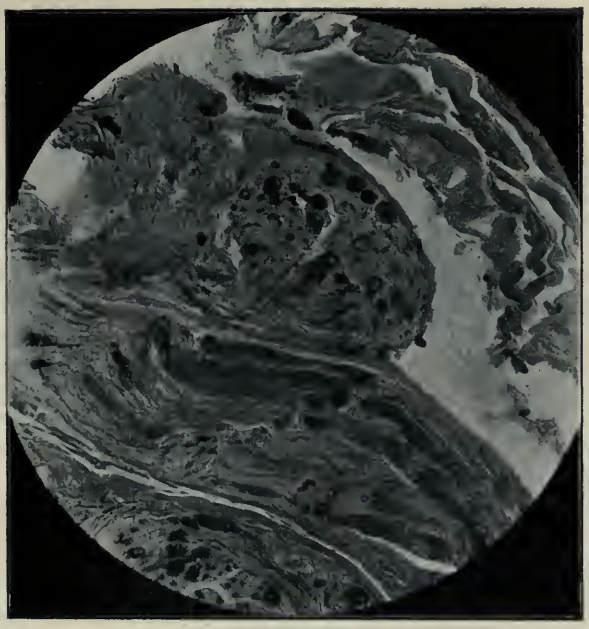

FIG. 3.
Peripheral nerve, partly degenerated.

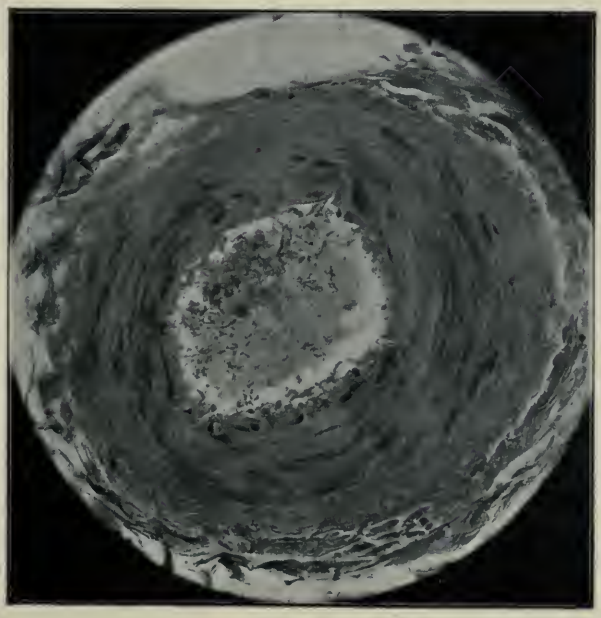

FIG. 4.
Artery with thickened muscular and external layers.

Addendum.

After returning the proof of this article, a paper by Spiller[1] has come into my hands, from which it appears that he has already examined a case of muscular dystrophy (apparently not pseudo-hypertrophic paralysis). Spiller did not succeed in finding any change in the muscle-spindles.

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OBSERVATIONS ON SENSORY NERVE-ENDINGS IN VOLUNTARY MUSCLES.

BY ANGELO RUFFINI.

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THERE are at least three kinds of sensory end organs in voluntary muscles. Of these the most important is the muscle-spindle, and next to it comes the tendon organ; besides these the muscles possess a certain number of Pacinian corpuscles.

I.—MUSCLE-SPINDLES.

From the histological and physiological facts which we now possess regarding muscle-spindles, there can, I think, no longer be a question as to their nature being sensorial. It was already, in 1888, conjectured by Kerschner, but Kölliker in the following year rejected his arguments. In 1892 I myself was able to strengthen the view by contributing for the first time an accurate histological description of the nerve-ending; and in 1893 Sherrington actually demonstrated the existence of sensory nerves in muscle, and in the following year, by indisputable data obtained by experiment and microscopy, finally furnished conclusive proof that the spindle is an organ of sense.

In the past two years I have had the opportunity of preparing a large series of specimens of muscle-spindles from the cat. These have yielded me interesting results, which I will here summarise. In the muscle-spindles of the cat there exist three distinct types of nerve-endings, which I will call respectively the primary, secondary, and *plate* endings.

The Primary Form of Ending.—This is the nerve-ending which I described minutely and figured in 1892, and Sherrington has confirmed my description of it. The large nerve-fibre which goes to form this ending just before it joins the spindle, almost always divides into two or more secondary branches; these branches, after having passed through the capsule of the spindle, divide into tertiary branches, each of which approaches a muscle-fibre in the intrafusal muscle-bundle, and terminates on it. The myelin sheath is lost on the tertiary branches, and each then becomes broad and flat and riband-like, and then either winds spirally round the muscle fibre or runs along one side of it as a longitudinal band from which, from point to point and at varying intervals, troop-like terminal expansions clasp the entire circumference of the fibre. Both these two modifications may co-exist and even lie juxtaposed in one and the same fibre. That is why it seemed, and still seems, to be well to give that ending the name of *annulo-spiral ribbon ending*. At the two extremities of this annulo-spiral ribbon ending the nerve terminates in diverse swollen knobs.

The Secondary Form of Ending.—Besides the primary there is found another form of ending distinct from it in morphological character and in the individuality of the nerve-fibre whence it springs. It has not yet been described in any paper, but I have known of it for some years. The parent nerve-fibre here also divides into secondary branches, but usually only after having penetrated the spindle. The secondary branches soon break up into a number of varicose axis-cylinders, united by very delicate and short filaments. The varicosity of the nerve-fibrils is of various kind, rounded, bifid, triangular, leaflet-like, &c.; the varicosities seem always disposed in such a way as to recall the arrangement of a festoon-wreath of flowers. Hence I call this form of termination the *flower-wreath ending*. The terminal expansions of the axis-cylinders of these nerve-fibres lie on the intrafusal muscle-fibres, but are hardly ever possessed of such long stalks as those of the primary ending.

The Plate Ending.—The size of these endings is extra-

ordinarily variable. One finds some instances smaller than motor end-plates, some equal in size and some much larger; the last-named are the most usual. There is a difference between these and the ordinary motor end-plates; the terminal expansions of these plate-endings are attached to short and extremely delicate filaments, so that they form, as it were, chaplets, in which rounded axis-cylinders and cross-pieces of the finest delicacy succeed each other in turn. The chaplets which compose a plate-ending anastomose one with another by minute filaments, and at the extreme margin of the plate terminate with obvious enlargements that are rounded off, on this side turned away from the plate. It is usual for a single individual fibre to pass to each plate-ending.

I have not met with each of the above-described endings in every spindle; it is for that reason that it seems to me important to distinguish three forms[1] of "spindles": (1) The spindle with complex nerve-ending; (2) the spindle with simpler nerve-ending; and (3) the spindle with simplest nerve-ending. In spindles of the first class there co-exist a primary form of ending, two secondary forms, and as many as twenty or more of the plate-endings. The primary ending and the two secondary are almost always found very near together; the plate-endings are set at various distances, some in the proximal and some in the distal parts of the spindle.

In spindles of the second class there occur a single primary form of ending, a single secondary form of ending, and a number of plate-endings, set as in the spindles of the first class.

In spindles of the third class there exist a single primary form of ending and a few plate endings, always arranged as in the spindles of the two other classes.

From this it is clear that, firstly, the primary form of ending, *the annulo-spiral ribbon*, is met with constantly in all spindles of the cat; secondly, that the secondary form of ending is that which can vary and even be wanting alto-

[1] Accad. Medico-fisica di Firenze, February, 1896.

gether. Hence the nomenclature I have ventured to propose and adopt.

Of the three classes of spindles examples are not met with equal frequency. In the cat, examples of class 1 are the most numerous; examples of class 2 the least numerous.

To the two older views which considered, on the one hand, that the muscle-spindle was an embryonic centre for the development of new muscle-fibres, or, on the other, a pathological product, we cannot, and ought not, to-day to allow anything more than a historical value. They are opinions which we have had, of necessity, to discard as our knowledge of these special nervous organs has progressed. Sherrington, besides having furnished some valuable additions to our knowledge of the structure of these organs, has succeeded in demonstrating, by means of his experiments, that the myelinate nerve-fibres ending in them suffer no damage after the complete destruction of the anterior (motor) spinal nerve-root, and has proved that the nerve-fibres in question take their origin in the cells of the spinal ganglia. If to this experimental result be added now the new data afforded by my anatomical observations, it will be clearly evident that there is an indisputable basis of truth for the new view that the muscle-spindle is of sensorial nature.

II.—Tendon Organs (Golgi Organs).

In 1892 I called the attention of histologists to two forms of nerve-ending that can be found in the tendon organs. The few observations which I then published I have since largely added to, and have further extended my enquiry to the structure of the tendon of the musculo-tendon organ, with the view of throwing light on certain facts which were laid stress upon by A. Cattaneo and Ciaccio. Together with the nerve-fibre proper to the Golgi tendon organ I find in the cat another much thinner myelinite nerve-fibre accompanying the proper fibre. I call this the *concomitant fibre*. This fibre, arrived in contact with the musculo-tendinous

organ, loses the myelin sheath, and begins to undergo subdivision into a number of secondary fibres, and these latter are so tenous that only a very perfect reduction can reveal them in the preparation. These excessively minute fibres do not anastomose one with another, but they do form a plexiform network, more or less extensive, though never very extensive, because the number of their subdivisions is never very great. Generally they follow a direction from the tendinous end toward the muscular end of the Golgi organ, and terminate close to where the tendon passes into muscle-fibre, or in the muscle-fibre, even at some distance from the tendon of the organ. Their mode of ending consists in the formation of an apical beaded chaplet, the end bead being much larger than the more proximal. This ending sometimes can be seen to be not on the muscle-fibre proper, but on the membrane which Sherrington names the axial sheath, because it lies within his periaxial space. It seems, in fact, to me that these concomitant fibres, with their twisting course outside the Golgi tendon organ, adhere to it rather than strictly speaking penetrate it or belong to it. The existence of the above-described plexiform nerve-ending may be really rare, or its rarity may be only a seeming one, on account of the difficulty of regularly obtaining the exquisite and indispensable delicacy of reduction required to make it visible to the eye of the observer. As to the structure of the tendon of the tendon organ, I have succeeded in demonstrating that the tendon is in reality like an ordinary secondary bundle of tendons. In a transverse section it can be seen that the tendon has two strata, an external and an internal. The former is softer and less compact than this latter. The outer layer is of fibre bundles circularly disposed, and from the inner face of this sheath septa pass inwards, which divides the tendon into two or more compartments. These compartments are occupied by tendinous bundles of longitudinal arrangement. In thus demonstrating the characters of the tendon of the Golgi organ, I find my data easily explain the nature of the *ring constriction* of A. Cattaneo, and of the *fibro-elastic band* of Ciaccio.

III.—Pacinian Corpuscles.

In 1892 I described the existence of Pacinian corpuscles in muscle, and Sherrington has since confirmed my discovery by observations of his own. Some of these Pacinian corpuscles lie near to and bear special relation to the tendon organs of Golgi. Sometimes they lie near the muscular and sometimes near the tendinous end of the organ. They have, for the most part, the same direction as the long axis of the tendon organ. By using serial transverse sections I have been able to get an accurate picture of the relations of these Pacinian corpuscles to the Golgi organs.

Just before or immediately beyond the preterminal constriction the nerve-fibre, which up to that place had been simply lying alongside of the tendon organ, turns and pierces the sheaths of the organ. It however only runs to a short depth into the organ, and then proceeds along it close beneath its surface. It then repierces the sheath partly or wholly, and in an outward direction, and terminates in its own small Pacinian corpuscle. This Pacinian corpuscle lies situate among or on the outermost fasciæ of the Golgi organ. In fact, the small Pacinian corpuscle lies embedded in a hollow scooped out of the surface of the tendon organ. This relationship I have studied especially in the cat. I have not been able to satisfy myself that it exists in the rabbit; in the latter animal, to judge by what I have seen, Pacinian corpuscles lie near to but not integrated in the connective tissue of the tendon organs. Typical Pacinian corpuscles are found in the muscles, but those which lie beside the tendon organs differ from the classical type in volume and in development of lamellated sheath. They are quite small, very elongated, and possess only four to eight capsular lamellæ. The axial portion is always thick, and its nerve-fibre myelinate. There is never more than one nerve-fibre, and that bears a well-developed terminal swelling. In the rabbit these corpuscles are rather longer and more lamellated than in the cat.

Pacinian corpuscles are met with frequently also, not actually inside the muscles but outside, in the perimysium.

They are here free of all connection with tendon organs, and are no doubt equivalent to the perimysial Pacinian corpuscles well known in the human perimysium since the descriptions of Golgi and Mazzoni.

Finally, I may take this occasion to state the general conclusions that are to be inferred from these results.

There are in the voluntary muscles nerve-fibres of three kinds : motor, sensory, and vaso-motor.

The motor nerve-fibres end, as is well-known, in the *end plates* of Rouget and Kühne.

The sensorial nerve-fibres possess three quite distinct end organs in man and in all the higher vertebrata. These sensorial end organs of muscle are—(1) the muscle-spindles, (2) the tendon organs (or Golgi organs), and (3) Pacinian corpuscles.

The vasomotorial nerve-fibres form reticular plexuses or true terminal plates (Mazzoni), or terminate simply on the capillary walls with a final apical enlargement (Ruffini).

The function of the motor plates and vasomotorial endings has been known for years. Further, physiological experimentation is now wanted to investigate the functional activities of the three sensorial organs—the spindles, the tendon organs, and the Pacinian corpuscles. In my opinion it is upon these three kinds of sense organ that physiology must turn its attention if it will resolve the problem of the muscular sense.

SHORT NOTE ON SENSE ORGANS IN MUSCLE AND ON THE PRESERVATION OF MUSCLE SPINDLES IN CONDITIONS OF EXTREME MUSCULAR ATROPHY, FOLLOWING SECTION OF THE MOTOR NERVE.

BY VICTOR HORSLEY, F.R.S., F.R.C.S.

As a small contribution to the question of existence of sensory nerve endings in muscle and of the preservation of muscle spindles in muscular atrophy I desire to publish the following photographs:—They were obtained from tranverse sections of the gastrocnemii and in some cases the solei of cats and dogs in which the sciatic nerve had been divided, at varying periods before the animal was killed. These periods varied from three days to one year. At the same time I wish to record the existence of Pacinian bodies which I have also found in the same muscles; and to draw attention in passing to the relative degree to which the gastrocnemius in the cat is composed of red and pale fibres mixed.

The Muscle Spindles.

The muscle spindles of the normal gastrocnemius of both the cat and the dog are as a rule so distended in the equatorial region with lymph that the bundle of muscle fibre, nerve fibre, and capillary occupies only about a third to a quarter of the area of the space as seen in tranverse section. This proportionate volume of the components of the spindle is not altered during the first few days of the atrophy, but as a rule by the seventieth day there is apparently a shrinkage of the spindle, such shrinkage, be it remembered, being parallel to the general shrinking which the atrophy of the muscle gradually undergoes as a whole (see photograph,

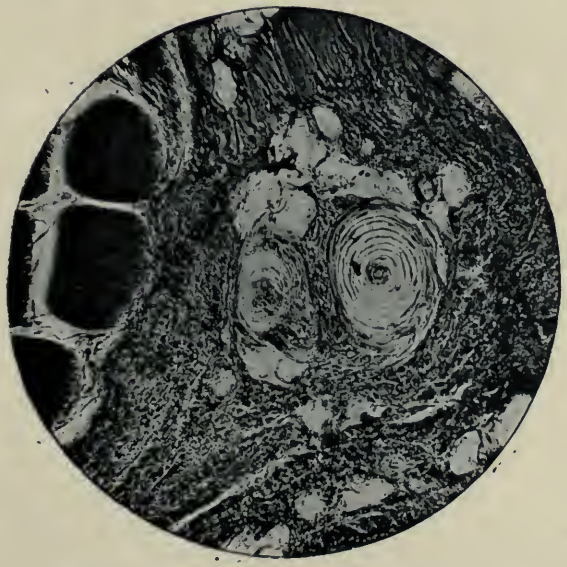
section C). At the same time the muscle fibres are apparently wholly unaltered in character. Very exceptionally a muscle spindle may be found in which no collapse or shrinkage of its cavity has occurred, and this even in the most atrophied specimens.

I must now refer to the condition of the muscle in the cases recorded. In the cases I have examined, and of the cat, the muscle fibres lose their diameter, *i.e.*, their volume, by simple atrophic shrinking, the distinction between the red and pale fibres becomes more defined (see photograph, section D), and the striation is preserved almost to the end. In addition to the loss of substance the particular experiments in the course of which these observations were made, seem to show that after a preliminary increase of metabolic activity there is a steady diminution of the same, parallel with the well-known steady decrease in the force and increase in the duration of a contraction. However, even in the most completely atrophied muscle I have been always able to obtain a very slight movement on direct excitation.

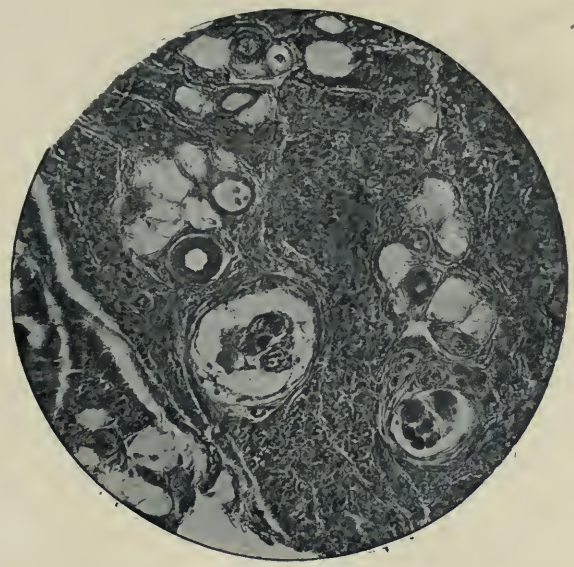
Sensory Nerve Endings in Muscle.

In 1883 I showed that in the sheaths of nerve trunks besides end bulbs or tactile corpuscles there were also true Pacinian bodies. I now wish to report that I have found the same within the gastrocnemius muscle in the cat (see photographs, sections A & B). In one case the body (in this instance a double Pacinian body) lay close to one of the inter-muscular septa of the strong aponeurotic fascia of the muscle, but in another case it was buried in the muscle between the bundles of fibres. These Pacinian bodies are ellipsoidal in tranverse section and have diameters of 104 and 144 μ at their thickest part, and present the ordinary well-known structure. The head of the core in one case at least is trifurcated. (See photo.) Whether in the midst of the totally degenerated muscle or in normal muscle these Pacinian bodies show no difference in structure, and do not exhibit any change, which seems to suggest their possessing a high (? central) nutrition efficiency.

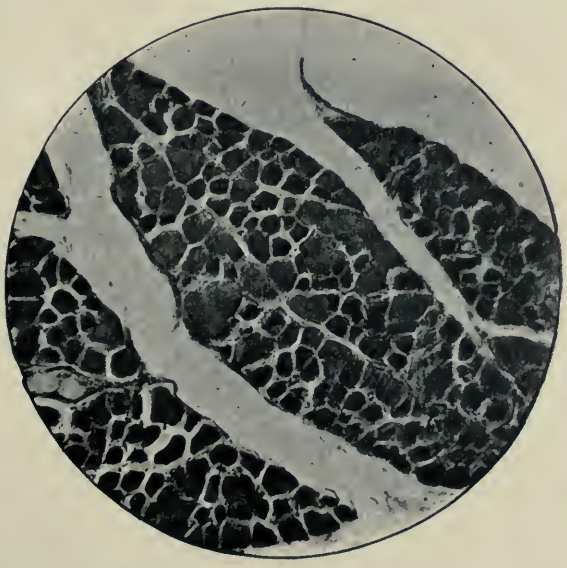
SECTION A.—Tactile body in normal muscle.



SECTION B.—Tactile corpuscle (double).



SECTION C.—Shrinkage of muscle spindles.



SECTION D.—Pale and dark muscle fibres.

A CASE OF ACUTE GRAVES' DISEASE, WITH A DESCRIPTION OF ITS MORBID ANATOMY, AND OF A SERIES OF MICROSCOPICAL SECTIONS.[1]

BY ARTHUR FOXWELL, M.A., M.D., F.R.C.P.

Physician to the Queen's Hospital, Birmingham, and Examiner in Medicine at Cambridge University.

ANYTHING which tends to elucidate the obscure ætiology of Graves' disease is worthy of note. The following case presents a pathological state of the base of the brain not usually described in this condition. This is my excuse for venturing to place this imperfect report before the Section.

Rachel W., 41, was admitted into the Queen's Hospital, Birmingham, on June 21, 1893, complaining of weakness, anorexia and rapid wasting. Her parents were alive and healthy and possessed a comfortable home; four sisters and one brother were also of good constitution; one sister died in childbirth, and two brothers in infancy. There was no history of neurotic tendency in any near relative. Her previous health had also been quite good, except that, ten years ago, she strained her left side whilst rowing and had suffered occasional pain there ever since. There was no suspicion of syphilis or alcoholism. Her present illness commenced at Christmas, 1892, six months prior to her death. Her first symptoms were weakness and a stiffness of the joints on rising in the morning. Except for the slow increase of the weakness and a gradual onset of dyspnœa on exertion, she remained fairly well till March, when she began to suffer from morning nausea without vomiting. In April she first noticed an enlargement of the thyroid, the increase was at first rapid, but it has since varied much in size, sometimes the fulness of her neck

[1] A paper read in the Section of Pathology at the Annual Meeting of the British Medical Association, in August, 1895.

almost vanishing for a week at a time so that she could easily fasten her dress. At this time, too, Dr. Quirke, of Handsworth, first noted undue prominence of the eyeballs, this she had never observed herself though her friends had done so.

For a month previous to admission she had suffered from vomiting and for a fortnight she had been unable to take solid food, though she could not say she had any difficulty in swallowing. For a fortnight she had been too weak to walk; and had perspired freely whenever she fell asleep.

On admission she was found to be a spare but well-formed woman of average size, nervous temperament and dark complexion. She appeared greatly emaciated. Her mentality was quite clear, though there seemed to be much anxiety and mental tension. There was extreme restlessness of one or other portion of the body, though as a whole the body remained still, maintaining the dorsal decubitus. Her cheek bones were prominent, she had a hectic flush, and the temperature was 99·4°. The pulse was 120, small, regular and of low tension; the radial felt healthy. The respirations were 32 to the minute and rather panting in character. She was without appetite, an unpleasant taste being constantly present. The tongue was very raw and vividly red; it was deeply fissured and had scattered patches of dirty fur. The eyes were prominent, but not strikingly so, the sclerotic not being visible above the cornea. The left pupil was larger than the right.

The Thyroid had, just to the right of the mid line, a rounded, hard, walnut-sized nodule; beyond this was a moderately enlarged right lobe over which a systolic murmur was plainly heard. The left lobe was also slightly enlarged. There were no pressure symptoms. The cardiac impulse was best felt in the fourth space within the nipple line, but it was largely diffused from without the nipple line in the fifth space up to the second space close to the sternum. There were pulmonary, tricuspid and mitral systolic murmurs; and, in the veins of the neck, a loud *bruit du diable* with marked systolic pulsation, especially during inspiration. Friction sounds, cardiac in rhythm, were heard over the sternum between the third and fourth cartilages. In the first left space and, to a less extent, in the second space was a curiously circumscribed and definite area of deficient resonance; so well marked was it that my able house physician, Mr. J. A. H. White, and myself, decided that it could only be due to an enlarged thymus gland. At the apex of the right lung the percussion note was slightly deficient and the

expiration lengthened; whilst in the right interscapular region the breath sounds were feeble. Otherwise the lungs appeared healthy. The abdomen was convave and rather hard, the iliac crests being very prominent. The hepatic and splenic dulnesses were normal and neither organ was felt. The urine was fairly normal in quality and quantity.

Treatment and Course of Case in Hospital.—It being very difficult to administer food by the mouth, the patient was fed by nutrient enemata to which was added three times daily a ♏ 40 dose of bromidia, and she was given in addition one teaspoonful of milk every half-hour. On the 24th, as the rectum was exceedingly irritable, two enemata only were given daily, by the long tube, to each of which was added ♏ 20 of laudanum instead of the bromidia. The nausea was now excessive, and a vomit of clear, greenish, strongly alkaline fluid became frequent. On the 25th the patient stated that she had no nausea, but could not avoid almost constantly regurgitating her food. On the 26th she was given ♏ j. of ipecacuanha wine every hour and had no more regurgitation till shortly before death.

On the 27th the pulse rate rose to 168. The restlessness increased. There was a tendency to bed-sores, and "on the hands and feet was slight desquamation." The temperature which had varied from 98·5° to 99·5° now took the higher level of 100° to 101° and so remained till death. On the 28th, 35 ounces of saline fluid were injected into the subcutaneous tissue of the axilla. The pulse, which had been quite uncountable, improved and the restlessness became somewhat less. The next day the pulse and restlessness again grew worse; the emaciation was strikingly rapid; her thirst painful to witness; nothing of any kind could be retained when given by the mouth; and she died exhausted, early on the morning of the 30th.

Briefly, then, the salient features of the case were these:

(1) The three cardinal symptoms of Graves' disease.

(2) Enlargement of the thymus.

(3) Dilatation of the left pupil.

(4) Moderate pyrexia, apparently due to the lung.

(5) Extreme restlessness, rapid wasting, uncontrollable vomiting which was sometimes without nausea or retching. Clinically, it was these last which occupied our attention and made us think there must be some cerebral lesion, the gastric irritability being due to involvement of the vagus.

(6) The rapidly fatal issue, only two months after the enlargement of the thyroid was first noticed.

The post-mortem examination was made thirty hours after death by our pathologist, Dr. Kauffmann, to whom I am indebted for the following excellent report.

The body was rather emaciated with much wasting of the subcutaneous fat.

The thymus extended from the lower border of the thyroid isthmus to the second intercostal space, overlapping the upper part of the pericardium; it was one and a half inches wide, but so thin that it did not weigh half an ounce.

The central portion of the upper lobe of the right lung was of a dirty red colour and contained distinctly less air than the rest. The front border of the right lung was also large and emphysematous. Otherwise the lungs were normal.

The heart was not enlarged, nor was there pericarditis. The aorta was full and distinctly pushed the pulmonary artery to the left. The apex lay beneath the fifth rib, one inch without the costo-chondral joint. The right auricular appendage reached up to the lower border of the second left cartilage. The valves and muscle were healthy.

The kidneys were healthy. Of the liver there is no note, but our memory is that it was healthy. The spleen was small, hard and granular.

The uterus was retroflexed; at its hinder part was a fibroid tumour as large as the body of the womb.

Of the thyroid, unfortunately, there is also no note, but we believe it was in the condition usually found in Graves' disease.

The ganglia of the cervical sympathetic were rather small and wasted throughout, but not harder than normal.

Only the upper portion of the cervical region of the spinal cord was examined; this appeared healthy.

The brain was slightly more adherent to the skull-cap than normal; its surface veins were full, both in front and behind; the arteries at the base were also tolerably full.

There was a patch of yellowish fibrous thickening over

the foremost part of the vermiform process of the cerebellum; this was small in extent but was surrounded by a distinctly thickened, and slightly milky patch of pia of much greater extent. The arteries and veins, chiefly on the upper surface, were markedly full and rather more tortuous than usual.

The third ventricle. While the front commissure appeared fairly normal, the middle and hind ones were much altered. The middle commissure was represented anteriorly by a very thin, grey, gelatinous mass of nerve tissue, no thicker than an ordinary sewing thread; whilst behind this, replacing what should have been the bulk of this commissure, were several delicate, but firm and strong bands of fibrous tissue connecting the adjacent surfaces of the optic thalami, which at these points were thickened, puckered and irregular. The hind commissure appeared thin and supported a dark grey pineal gland which was very friable (owing perhaps to *post-mortem* change).

On the upper part of the opposed surfaces of the optic thalami were two patches of grey translucent softening; these did not extend deeply into the substance of the thalami. The choroid plexus was not congested.

The internal capsule and its surrounding parts, except for an undue number of puncta cruenta, as well as the iter and corpora quadrigemina, appeared healthy. So also did the pons and cerebral hemispheres.

The fourth ventricle presented a remarkable appearance. The whole floor looked thickened and the finer shadings were lost, but the natural hollows were more marked than normal:—*e.g.*, the two depressions on either side of the middle line in front of the auditory striæ which striæ were quite absent. The calamus scriptorius itself was too deep and too sharply marked. The whole looked as if the more delicate distinctions had been swallowed up in an exaggeration of the main lines and a thickening generally of the tissue.

For the microscopic sections of the medulla and pons which I am enabled to show you, I am indebted to the kind skill of Dr. Douglas Stanley, Pathologist to the General

Hospital,[1] and it is with his aid that I have written the following brief account of the appearances noted in them:

(They were all prepared according to the Weigert-Pal method—that is, the material was hardened in Müller's fluid. The sections were stained in hæmatoxylon and then bleached by being placed in a $\frac{1}{4}$ per cent. solution of permanganate of potassium, and afterwards in a solution of $\frac{1}{2}$ per cent. pure oxalic acid and $\frac{1}{2}$ per cent. of sulphite of potash. They were then washed in distilled water, and afterwards in tap water.

The result is to stain the medullated fibres blue, the nerve nuclei a brownish yellow, whilst the neuroglia and other fibrous tissue is almost white.)

The lowest section (*Right hand figure of No.* 6 *slide*) is through the lower end of the olivary body ($\frac{1}{12}$ in. above the horizontal line *d* in Obersteiner's figures). The olivaries themselves were healthy. The vessels are dilated and engorged throughout but especially on the left half. The tissue around the central canal takes the stain badly and has an indistinct, ground-glass appearance; in it are several "amyloid" bodies.

The next section (*Middle figure*) at the level of the Calamus is similarly gorged. The pia is much thickened, and on each side of the central cleft of the floor of the fourth ventricle, it is indistinguishably mixed with the gracilis column; the differentiation of nerve elements being almost effaced, whilst the production of new blood vessels is great.

The third section has still marked congestion though slightly less than the preceding. There is the same thickening of the pia on the floor of the ventricle, and here it passes into and involves the nucleus of the gracilis. The pia and the underlying tissue together form a sclerosed layer about six times the thickness of the normal membrane. The nucleus of the twelfth nerve as here seen is normal.

The fourth section (*Slide* 5, *right hand figure*) at the level of Obersteiner's line *e*, shows the nucleus of the tenth nerve involved in the sclerosis, though to a less extent than

[1] Now physician to out-patients at the Queen's Hospital.

was that of the gracilis, for its nerve cells appear to be but little changed. The ascending root and nucleus of the eighth and the nucleus of the ninth show no morbid change.

Number 4 *slide* (sections 6 and 7, at level of Obersteiner's lines *g* and *h* respectively). The vessels of the pia are much distended and there are one or two spots of hæmorrhage. The pia is rather less thickened, but its thickening extends over a wide area.

Slide No. 3 (with sections 8 and 9 respectively, at levels between Obersteiner's lines *h* and *i*, and *i* and *k*), shows a large excess of vascularity in the nuclei of the eighth and seventh nerves, especially on the left side; but there are no definite hæmorrhages. All the central region of the cord in these two sections has also an excessive vascularity. The pia is still thickened, but now the thickening is less on the median raphe of the floor than at either side.

Number 2 *Slide* has sections 10 and 11 at levels a shade higher than Obersteiner's *m* and midway between his *m* and *n* respectively. The lower section shows several well-marked capillary hæmorrhages beneath the floor of the fourth ventricle on each side of, and in, the median line. Corpora amylacea exist over a wide area, occupying the whole of the tegmen and also extending into the pyramids.

The higher section shows nothing remarkable except excessive vascularity.

Slide No. 1, Section 12, just below the corpora quadrigemina, at Obersteiner's line *o*, shows less vascularity. There is an occasional hæmorrhage but otherwise nothing of note.

In brief, then, the changes found were:

(1) Chronic inflammation-sclerosis of the pia covering part of the vermiform process and of that covering the floor of the fourth ventricle. Also of the middle commissure of the third ventricle.

(2) Acute softening of the surface of both thalami.

(3) Excessive vascularity of the surface of the brain, of the internal capsule, of the cerebellum and of the medulla—in the medulla, at any rate, leading to occasional hæmorrhages.

(4) Sclerotic changes involving portions of the tegmen, the pyramids, the gracilis and their nuclei; and the nuclei of the tenth nerves.

It is difficult to definitely connect any of this morbid anatomy with the clinical appearances manifested by the patient. Bias, no doubt, would tend to make my own imagination run away with me in seeking for cause and effect. Still, the changes described are organic and decided and of sufficient importance, I think, to warrant my bringing the case before this Section.

TRAUMATIC NEURASTHENIA AND HYSTERIA.[1]

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When your Secretary asked me to read at this meeting a paper upon the functional disturbances of the nervous system following accidents, I promptly asked the privilege of changing the title to something more definite. Thanks to the new methods of research the old distinction between functional and organic diseases is fast disappearing, but, were it still maintained in its old-time definiteness, it is a question as to what or how many of the more obscure traumatic nervous affections should be classed under either heading. It was once thought that most of them were due to more or less pronounced changes in the spinal cord, such as we used to call organic, and later it was held by many that they were all of a functional character, but now the current of opinion (1) seems to tend to the belief that a part of them are due to organic changes, although the greater part are probably dependent upon those slighter changes which we call functional, and which can be detected, if at all, only by the recently discovered methods of staining.

Next to the old term of "spinal concussion," no name seems better calculated to add obscurity to the obscure than the new term of "traumatic neurosis." The word neurosis, of itself, is so often understood to mean functional nervous disease that it has a misleading significance. Traumatic neurosis, too, may designate certain cases of chorea, epilepsy, cephalalgia, or paralysis agitans, as well as hysteria and

[1] Read before the Section on Neurology of the New York Academy of Medicine, 11th February, 1897.

other obscure affections. It has added much to our ignorance of mental diseases, for example, to find that ætiology, symptoms, pathology, course, diagnosis, and prognosis have been studied from statistics of cases of amentia, general paralysis, mania, melancholia and paranoia all classed together and regarded almost as a single morbid entity, "insanity." The employment of the term "traumatic neurosis" is almost as confusing. It is only by a careful differentiation of the various affections, and a study of such disorder separately, that we can attain definite knowledge. It may still be impossible to draw a sharp boundary between hysteria and neurasthenia, as it is to draw one between tabes and paretic dementia; it is possible that in the future we may find that there are several distinct affections, all of which we now term hysteria; but, in spite of errors and ignorance, it is better to classify so far as we are able rather than to accept the vague and general term of traumatic neurosis.

Among the commoner traumatic affections of the nervous system, especially in cases where litigation is involved on account of claims for damages, are hysteria and neurasthenia. To determine this point I have classified two hundred cases of the more obscure traumatic nervous diseases seen in hospital and private practice. I have included all the litigation cases of traumatic nervous disease of whatever sort seen by me during that period, but I have not taken into account, among the non-litigation cases, those cases of injury to the peripheral nerves, meningeal hæmorrhage, fracture of the skull or spine, epilepsy, &c., where there was no doubt as to the nature of the trouble. As my object was primarily the study of the obscurer forms of nervous disease such omissions were justifiable, but the percentages here given are too great if we include all cases due to trauma.

Of these two hundred cases one hundred and three were not, so far as I know, the subjects of litigation, and a few of the ninety-seven litigation patients consulted me after all litigation was over simply for advice and treatment, and not to obtain my opinion for use in the settlement of their claims. Seventy cases were classed as hysteria and fifty as

neurasthenia, sixty per cent. of the whole. Of the litigation cases fifty were hysteric and twenty-one neurasthenic, eighty-one per cent. of the whole. Of the non-litigation cases twenty were hysteric, and twenty-nine neurasthenic, thirty-nine per cent. of the whole. The importance of these two affections is therefore evident.

Although injuries of the peripheral nerves and the spine or spinal cord are not uncommon in hospital practice, I have very rarely seen them—and my experience is shared by my colleagues in Boston—among the litigation cases. The only explanation that I can find of this is that, as the diagnosis and prognosis in such cases are usually plain, the opinion of a specialist in nervous diseases is seldom required, and the case, if the liability be admitted, is settled out of court.

The preponderance of neurasthenia in the litigation cases is not very great, and it may be due simply to chance, but the greater percentage of hysteria is another matter, and is worthy of consideration. It at once suggests and seems to strengthen the familiar plea that litigation and the desire for gain are important factors in the genesis of these so-called traumatic affections, producing, under the influence of suggestion, a set of symptoms, real perhaps in themselves, but not the result of the physical injury. Strümpell (2) has lately reiterated this plea with much emphasis. Having for some time urged that the chief factor in the production of "traumatic" or "accident neurosis" was psychical rather than physical, he now goes a step further and considers the chief psychical factor to be the ideas of personal gain which arise as a consequence of the accident. "Shall I obtain the accident pension? How much shall I get? How much am I injured in my working capacity?" With but a slight change in phraseology to suit the differences in our laws, these questions may be asked here. "Shall I get damages? How much shall I get? Shall I be able to earn anything afterwards?" We know well all the maleficent psychical influences which may attend these cases. The desire for gain, the necessity of putting the worst foot foremost, the depressing suggestions, the educating examina-

tions, the worry of litigation, the gloomy prognostications, the law's delay, and, sometimes, the total lack of proper medical treatment during the whole period of litigation put the patient into the worst possible condition and aggravate every symptom. It is no wonder then, that we ask if the physical injury has had anything to do with the subsequent trouble.

Although the whole subject of the so-called "traumatic neuroses" has been much discussed in England, France and Germany, it has awakened very little interest, apparently, south of the Alps (3). If, then, there are no laws in Italy whereby an injured person may recover damages, it may be, granting the truth of the hypothesis, that litigation is the important factor, that the cases are rare in that country, and that, therefore, little attention has been paid to the subject. In answer to my inquiry I have learned from Dr. Seppilli that there are no special laws in Italy relating to railway accidents. When accidents do occur (which is only very seldom the case) the railway companies indemnify, according to the cases, the persons or families for the damages received. The physicians of the company, however, must give their judgment upon the case.

In spite of all this I am disposed to doubt Strümpell's dictum, and to lay much less stress upon these ideas of personal gain and the maleficent psychical influences above referred to in the genesis of the hysterical or neurasthenic symptoms. In the first place, precisely similar conditions have developed in the non-litigation cases, where all these factors are absent; and, in the second place, the symptoms have developed almost immediately after the accident in many of the litigation cases, before these factors could be brought into account. When the symptoms follow immediately upon a period of unconsciousness, profound surgical shock, or marked mental confusion, it is hardly credible to suppose that the first idea to come with the regaining of consciousness is "Shall I get damages?" even though the lawyer's "runner" has reached the scene of the accident before the ambulance. That these factors may have a very bad influence in aggravating or prolonging the disease is

readily conceivable and most probable, but they certainly do not always create it.

I have just referred to the similarity between the litigation and the non-litigation cases. To determine this more accurately I have carefully tabulated the symptoms presented by these two classes of cases, both of hysteria and of neurasthenia. The relative frequency of the symptoms in the two classes of cases was about the same, and the rarer symptoms, observed perhaps in a single case, occurred now in one class and now in the other, as chance ordained. A comparison of individual cases in the two classes showed no striking differences. In other words, from the evidence presented by these hundred and twenty cases of hysteria and neurasthenia, seventy-nine of which were litigation cases and forty-one of which were not, I can find no symptoms which would warrant a diagnosis of "litigation hysteria" or "litigation neurasthenia," as distinct from "traumatic" hysteria or neurasthenia, and no essential difference between the two classes of cases, with one exception. In many, but not in all, litigation cases the patients have dwelt upon and have worried over the prospective law-suit. Worry and the dwelling upon certain ideas are, however, common in neurasthenia and hysteria, no matter what the cause, and in these cases the law-suit has merely furnished the special subject for worry.

It is a fact, however, that these non-litigation cases of hysteria, but not of neurasthenia, have been, as a rule, of a less severe type and have progressed more favourably than the litigation cases. This may be due to the absence of those injurious psychical influences attending litigation, to which I have already referred, and the more prompt inauguration of systematic treatment, but there are other factors which I believe have exercised a marked influence upon the severity of the manifestations.

That hysteria and neurasthenia may be due solely to psychical causes is a well-accepted fact. Even in traumatic cases the physical injury may be insignificant and the psychical shock the only factor. I have seen, for example, a marked somnambulic state develop from spraying the throat.

In emphasising the psychical factor, however, I believe that Strümpell and others have gone to an extreme, and have not given due weight to the physical injury. In order to estimate the importance of such physical injuries, and to find some reason why the percentage of hysterical cases should be so much greater in the litigation than in the non-litigation cases, I have studied the nature of the accident in these hundred and twenty cases.

Of the fifty litigation cases of hysteria here classified, seven were due to collisions of street-cars, seven to being thrown from a carriage, seven to falls on getting on or off the cars, six to railway accidents (in four cases of a grave character), six to electrical shocks, five to elevator accidents, three to falls, and two to blows on the head. Of the twenty non-litigation cases, six were due to falls, four to being thrown from a carriage, two to blows on the head, and two to blows on the arms. Of the fifty litigation cases, eighteen received severe physical injuries and two severe electrical shocks, while sixteen received moderate physical injuries and two moderate electrical shocks; in five cases there was a fracture of some bone. Of the twenty non-litigation cases two received severe, and ten moderate, physical injuries; in no case were any bones broken. From the nature of these accidents, as thus hastily outlined, it may be inferred, and the inference will be confirmed by detailed study, that the psychical factors of fear, horror, excitement and the like, were much greater in the accidents of the first class than in those of the second. In the majority of the non-litigation cases the accidents were of a simple character, unattended with any special terror or scenes of distress; in the litigation cases alarming features were not uncommon.

Turning now to the cases of neurasthenia, I have found that the severity of the symptoms has been, on the average, about the same in the two classes of cases. Of the twenty-nine litigation cases, six were due to railway accidents (in five cases of a grave character), five to falls on getting on or off cars, five to falls, four to blows on the head, and three to being thrown from a carriage. Of the twenty-one non-litigation cases twelve were due to falls, two to being thrown from a carriage, two to blows on the head, and two to strain.

Of the twenty-nine litigation cases eight received severe and nine moderate physical injuries, and one a moderate electrical shock; in three cases bones were broken. Of the twenty-one non-litigation cases four received severe physical injuries, one a severe electrical shock, and eleven moderate physical injuries; in three cases bones were broken.

From these facts it seems possible that, apart from any questions of litigation or suggestions arising from the desire of gain, there were reasons why there should be a larger number of cases of hysteria in the litigation series, and why they should be of greater severity. Litigation and the desire for gain are also present in the neurasthenic cases, but the severity of the accidents was about equal in the two classes, and we find a corresponding equality in the number and severity of the cases. The only conclusion, therefore, to which we can come is that litigation and the desire for gain play only a subordinate part in the genesis of these morbid conditions, and that the physical as well as the psychical shock of the accident is a very important factor.

I have already said that psychical shock alone may produce hysteria or neurasthenia—a fact repeatedly demonstrated by the researches of Guinon (4), Janet (5), and Breuer and Freud (6). The question naturally arises, can physical shock alone act as a cause? A satisfactory answer cannot be given. Even in the simplest accident, a fall on the ice, a strain in lifting, or a sharp blow on the arm against a piece of furniture, there may be some element of fear or apprehension to arouse a strain of morbid mental conditions. When a man is rendered unconscious by a sudden blow on the head, and later has no memory of the accident, it may be that, in the interval "between the saddle and the ground" on which the theologians once laid stress, there was some suggestion of future evil which hypnotism might reveal. One of my colleagues, a young physician, who in his hospital life had become somewhat familiar with accidents and scenes of distress, has suffered for a year and a half from neurasthenia, the result of a serious railway accident in which several persons were injured. He felt a jerk, lost consciousness for a second, and recovered his senses

to find himself standing in the aisle of the car facing to the rear, while the car next behind was crashing into the one he was in. He was uninjured and spent some time at the scene of the accident caring for the wounded. He stated that he was not frightened at all, and doubted whether there was any period of momentary fright of which he was not conscious. The sensation was exactly as if he had been struck a very heavy blow in the back of the neck with a sledge hammer. Such a statement from a trained observer I believe to be of some importance in deciding the question, and my own belief is that in a part of the cases the psychical shock, although it can not be wholly excluded, is distinctly a subordinate factor in the genesis of the trouble.

Of the other factors in the ætiology it is hardly necessary to say very much. With regard to age four of the cases of hysteria were under twenty, one fourteen, two sixteen, and one (the only litigation case) eighteen. Three neurasthenics, all of them litigation cases, were under twenty, one seven, one fourteen, and one seventeen. Women are much more subject to these affections than men. Thirty-eight men out of one hundred and thirty-seven (twenty-eight per cent.) had hysteria, and twenty-seven (nineteen per cent.) had neurasthenia. Thirty-two women out of sixty-three (fifty-one per cent.) had hysteria, and twenty-three (thirty-six per cent.) had neurasthenia. Thirty-seven out of forty litigation cases and eighteen out of twenty-three non-litigation cases among women had one or the other of these affections. A neuropathic heredity, upon which the French especially have laid much stress, I have found to be of minor importance. I have obtained a history of a questionable heredity (nervous disease, tuberculosis, &c.) in eight cases of hysteria and seven of neurasthenia. A previous history of poor health was elicited in twelve cases of hysteria and ten of neurasthenia. A history of previous syphilis or of abuse of alcohol was only rarely obtained. Many of the litigation patients, however, might be disposed to conceal any hereditary or pre-existing weaknesses.

Traumatic hysteria and traumatic neurasthenia have many symptoms in common, so that the designation of the

former affection as hystero-neurasthenia is not unwarranted, and the boundary between the two affections is not very distinct. Headache, nervousness and irritability, depression, insomnia, vertigo, failure of memory and mental power, pain in the back and limbs, paræsthesia, palpitation, dyspnœa, a rapid pulse, loss of appetite, dyspepsia, constipation, diminished endurance, inability for physical or mental application, general muscular weakness, and exaggerated knee-jerks, are common to both.

Of the symptoms common to the two affections a few are worthy of note on account of their objective value. In over one-half of the hysteric cases, and in over one-third of the neurasthenic cases the knee-jerk was distinctly exaggerated, being often attended with the patellar twitch and front-tap contraction, and very rarely with a spurious ankle clonus.

In a large majority of the cases, both of hysteria and neurasthenia, there was a decided increase in the rapidity of the pulse, it being over eighty and frequently weak, irregular or intermittent. Congestion, coldness of the extremities, flushing, excessive or localized sweating, and other vaso-motor disorders were not uncommon, but œdema was rare and cardiac murmurs and enlargement of the heart were exceptional. In a very few cases there was an abnormally slow pulse.

Mannkopf's test, the increase in the rapidity of pulse on pressure upon an alleged tender region, I have found of distinct value. Instead of an increased rapidity the pulse may become weaker or irregular under such conditions. In forty-five out of forty-nine cases the test has given definite information, but when there was other conclusive evidence of the disease this test, on account of its painful character, was not employed. Not only is it of value as a test of tenderness, but it is also a test of muscular effort. When a patient executes a slight movement, lifting the leg, closing the hand, stooping forwards, only by an apparently great effort, if we find that the pulse is materially quickened we have proof that the effort really was great. Flushing of the face and an outbreak of sweat may also be noted under such conditions. The test may also be used for analgesia. If the

pulse rises in consequence of a painful stimulus in a healthy area and does not rise when the same stimulus is applied to the alleged analgesic area, we have proof of the genuineness of the analgesia.

The chief distinction, so far as our present knowledge goes, between neurasthenia and hysteria is to be made by the detection of the various hysterical stigmata.

Of the various hysterical stigmata pronounced amnesia, after the initial period of amnesia due to shock, or pronounced aboulia was distinctly rare. An enfeeblement of the will power and a failure of memory corresponding to the general asthenic condition can hardly be classed as distinctly hysterical. Hallucinations of sight and hearing, temporary delirium, and temporary confusion were occasionally met with both in hysterical and neurasthenic cases, usually in the period immediately following the accident. Attacks of one sort or another were noted in sixteen cases, twelve of the litigation cases and four non-litigation cases. In none of these cases did these attacks manifest the typical features of the " grand attack " in its four stages, but occasionally they were severe enough to show the *arc de cercle* or other striking phenomena. In no case could I detect any hysterogenous zones. Hemiplegia was noted in twenty-five cases, menoplegia in ten, and paraplegia in one, contractures in five, tremor in twenty-four, ataxia in eight, mutism in three, and somnambulism in one.

The most frequent stigmata were those in the sensory domain. Hypoæsthenia was noted in sixty-four per cent. of the cases, analgesia in forty-nine per cent., and distinct anæsthesia in twenty per cent. Thermoanæsthesia was less often sought for, but was noted in seven out of thirteen cases. Many patients, fifty-seven, complained of numbness or other abnormal sensations, chiefly in the affected area (7).

In a few of the cases presenting other hysterical stigmata no disturbances of the cutaneous sensibility were manifest, but in most of these cases the examination was hurried and the opportunity for prolonged observation was lacking. Of course, in rare cases hysteria may occur without any disturbances of cutaneous sensibility or any contracture of the

visual field (8). In a considerable number of cases the loss of tactile sensibility was very slight. The patient could feel the slightest touch upon the affected area of the skin, but it was not felt quite so distinctly as on the corresponding healthy area. It required careful examination to detect the difference, so that in many cases it might be readily overlooked. In some very severe cases there was only this slight difference. In some cases the affected side is distinctly colder to the touch, especially in the hand or foot, and the patients complain of an increased sensitiveness to cold. Further valuable information, as confirming the genuineness of the sensory disturbance, is afforded by the condition of the cutaneous reflexes. The plantar reflex is frequently diminished or lost on the anæsthetic side, and the other reflexes sometimes show a similar change. Rosenbach long ago held that the abdominal reflex was not affected in cutaneous anæsthesia, unlike the other skin reflexes, but this is incorrect, as in no less than four cases it was diminished or lost on the affected side, and in one case it was diminished on one side when the plantar reflexes remained equal. The knee-jerks, too, often show an inequality, but this seems to have less relation to the side affected. It is rather more apt to be exaggerated on the affected side, as seen in eight cases, but in five cases it was exaggerated on the sound side.

As is well known analgesia may be present when the cutaneous sensibility is normal, so that many cases may be stated to show normal sensibility merely from lack of thorough examination. I have found the best test for sensibility to pain to be by means of the faradic current, applying Erb's cutaneous electrode to different regions of the skin, and measuring the strength of the current required to produce pain by Edelmann's faradimeter. A current of fifteen to twenty volts will usually cause pain in normal areas, while pain will be felt in analgesic areas only with a current of from thirty-five to eighty volts.

With regard to the distribution of these disturbances of cutaneous sensibility, in forty-two cases one side of the body was affected, but in many of these cases the area of anæs-

thesia was not sharply defined nor did it affect the complete half of the body; the face was often exempt, the anæsthesia did not reach fully to the median line, and there were some areas on the affected side where sensation was as good as on the healthy side. In eight cases the anæsthesia was confined to a single limb. In two cases the legs alone were affected. In six cases both sides of the body were affected. In one case the anæsthesia was very variable, now occurring in irregularly distributed areas, now affecting nearly one-half of the body, and, a day or two later, shifting to the other side. In a part of the cases the special senses were affected on the anæsthetic side, in some cases only one or two of the senses, and in others they were unaffected. A diminution in the sense of smell on one side was noted more frequently than of the other special senses. Achromatopsia or dyschromatopsia was noted in a few cases, but was not observed in others.

Contraction of the visual field was noted in forty-four cases, but in no case of examination with the perimeter was a normal field found, and in several cases examined in other ways, where the field was apparently normal, the perimeter subsequently showed some contraction, so that it is fair to suppose that contraction of the field is one of the most common sensory stigmata. In only a few cases, however, was the contraction marked, usually the field extending to about forty degrees on the nasal side and seventy on the temporal. The "shifting type" of field, although often sought for, was only rarely found. Reversal of the colour fields, also, was only rarely noted.

In accepting these stigmata as the distinctive symptoms of hysteria as opposed to neurasthenia, we are not free from the possibility of error. In the first place the stigmata may not appear until some time after the injury. In a case at present under observation the visual fields were normal, the cutaneous sensibility unimpaired and there were no motor symptoms five months after the injury; the only possible stigma was a loss of smell on one side. Two months later, however, contraction of the visual fields with peripheral exhaustion ("shifting type") had developed, with right

hypoæsthesia and analgesia, and these symptoms have persisted, with others, for nearly a year and a half. Furthermore, with the progress of hysteria, an existing anæsthesia or hypoæsthesia may wholly disappear, leaving behind only a slight analgesia which may readily escape notice unless careful electrical measurements be made; and finally, all disturbances of cutaneous sensibility may be wholly wanting. In some of these cases the visual field may remain contracted, but in others it may enlarge to normal. It may thus happen that, in ignorance of past conditions or of the future development of the case, we may regard a case as simply neurasthenic that in the past or in the future had shown or will show clear evidences of hysteria. Whether a contraction of the visual field, without any other stigmata, is sufficient to warrant a diagnosis of hysteria, is still open to question. Charcot (9), on the one hand, holds that it does not occur in uncomplicated neurasthenia, but Wilbrand and Sänger (10), on the other hand, whose work on the visual field is of great value, and also Löwenfeld (11), claim that it may occur in such cases. In four cases which I have classed as neurasthenic, there was some contraction of the field without other stigmata of hysteria, and in the absence of other stigmata I have not ventured to class the cases as hysterical. It is true, however, that in non-traumatic neurasthenia I have not yet found contraction of the visual fields, but a careful perimetic examination was only rarely made.

Contracture of the visual field, by itself, may undoubtedly occur in various affections. I have noted it in cases of brain tumours without as well as with optic neuritis, in the early stages of optic atrophy with as regular an outline as in hysteria (contrary to Charcot's statement (12) as to the fields in tabes), in spinal sclerosis, in tobacco amblyopia, in oculomotor paralysis, in melancholia, and rarely in epilepsy, not as a transitory phenomenon following the attack (13), and in other cases where the idea of hysteria was not entertained. This fact, however, is well recognized, and it has been fully demonstrated by the researches of Wilbrand and Sänger.

When, in a given case, we find disturbances of the cu-

taneous sensibility, not following the distribution of the peripheral sensory nerve areas or the spinal root areas, associated with contracture of the visual field, are we justified in assuming that the case is one of hysteria? If the sensory disturbance affect one half of the body the probability of hysteria seems greatest. Patrick (14) has laid considerable stress upon a sharply defined boundary of the anæsthetic area as characteristic of hysteria. This may be the case, and in a very few cases it was noted, but in the great majority of cases, including many that were undoubtedly cases of hysteria, this sharp outline did not exist. Charcot (15) has claimed distinctly that in hysteria alone do we find profound hemianæsthesia involving the special senses, and Gilles de la Tourette (16), following Charcot's teachings, holds that hemianæsthesia is due either to a capsular lesion or to hysteria. Of course, instead of a capsular lesion there may be a focal lesion elsewhere in the sensory tract. The wider knowledge of hysteria, due to the investigations of the French neurologists, have rendered the conclusions of Thomsen and Oppenheim (17) somewhat unsatisfactory, and further investigations are desirable. If in alcoholism, lead poisoning, multiple sclerosis or epilepsy, we find hemianæsthesia with contracted fields it may often be true that there is a coexisting hysteria, but it is scarcely logical to claim that all such cases are probably hysterical (18), or boldly to assert with Souques (19) that because the patient presents these stigmata he is therefore hysterical. It is not long since it was held that hysteria and syringomyelia were very often associated, because the sensory disturbances in syringomyelia did not follow the regular spinal distribution, but later inquiry has shown that the distribution of anæsthesia in lesions of the cord itself is very different from that in lesions of the spinal roots. It is not at all improbable that careful research will prove that hemianæsthesia is not pathognomonic of hysteria. In migraine I have occasionally heard patients complain of a subjective feeling of numbness over one half of the body during the attack, and Dr. J. J. Putnam tells us that he has noted an actual diminution of sensibility on one side during an attack of supraorbital

neuralgia. I have also seen hypoæsthesia over one half of the back in a case of mitral disease and phthisis. Hemianæsthesia in focal lesions of the brain is, of course, not uncommon. Not long ago I saw for a few moments only a man who had had epileptic seizures in childhood. Three years before he was thrown from a wagon in a collision with a railway train, and after that he was no longer able to do hard work. Two years later the fits returned. When I saw him he had atrophic paralysis of the right shoulder and upper arm, tremor of the hands, exaggerated knee-jerks, and right hemianæsthesia. Not long after he died of pneumonia. The autopsy showed a very peculiar and extreme atrophy of the brain cortex, of unknown origin, involving the whole left ascending parietal convolution and the upper part of the right ascending parietal, but not extending beyond those convolutions.

It becomes a question, therefore, which is still undetermined as to whether we can regard even hemianæsthesia as indicative of hysteria. The implication of the special senses, sometimes noted in hysterical hemianæsthesia, is by no means decisive. In a part of the cases there was a distinct diminution of all the special senses on the anæsthetic side, with dyschromatopsia; in others the difference was very slight, affecting one sense only, especially the sense of smell; in other cases still, which were clearly hysterical, the special senses were wholly unaffected, or, if affected at all, difficulty was due to some local disturbance, a refractive error, old middle-ear disease, or an occluded nostril. In certain cases, too, the colour sense was perfectly normal, and the colour fields showed no special change. In a few instances the colour fields were reversed. This study of the colour fields which, I regret to say, was undertaken in only a few cases, confirms the conclusions of Mitchell and De Schweinitz (20), who found that a reversal of the colour fields was only rarely seen. It is clear, however, that the absence of disturbance of the special senses cannot exclude hysteria, in cases where there is a cutaneous hemianæsthesia, and, even, if such disturbance be noted, we must be sure that it is not due to local causes.

If it be a question whether the disturbances of cutaneous sensibility which affect one half of the body can be regarded as an absolute indication of hysteria, the doubt becomes still greater when we have to deal with the cases, sixteen in number, where the disturbance affected one limb, both legs, or much of the body. The segmental character of anæsthesia affecting one limb, in the gauntlet, sleeve or stocking type, once thought characteristic of hysteria, is now known to occur in syringomyelia and other affections of the cord. Only a few days ago I observed such an anæsthesia of the foot and lower part of the leg, in almost precisely the area covered by a sock, in a typical case of tabes, and the boundary was as well defined as is often seen in hysteria. When the anæsthesia effects the legs alone, or most markedly, and gradually grows less as it goes up the body, fading away without any definite boundary into areas of normal sensitiveness, the hysterical nature of the disturbance is certainly questionable.

On the whole, therefore, it seems probable that the majority of cases which present disturbances of the cutaneous sensibility and contracted visual fields are hysterical; but, where these symptoms exist without the corroborative proof of other hysterical stigmata, it is doubtful whether we can assume that the case must therefore be hysterical, and it is a distinct begging of the question to claim, when a patient is clearly suffering from some other disease, that hysteria must also be present because he has hemianæsthesia and contracted fields. It is undoubtedly true that many of the symptoms which were once thought to be absolute evidence against hysteria, such as ankle clonus, facial or oculomotor paralysis, immobile pupils, incontinence of urine, and even reaction of degeneration, are said to occur in hysteria (21). Nevertheless, in spite of the great advances made in our knowledge by the French, I am disposed to agree with Rumpf (22) that the concept of hysteria has been extended to cover a much wider field than is justifiable. Souques has criticised very sharply the views of Buzzard (23) regarding the simulation of hysteria by organic disease, but it is nevertheless true that there are cases of disseminated or

diffuse sclerosis where, for a considerable period, the symptoms may seem simply those of hysteria or neurasthenia, and where it is a very difficult matter to determine whether we have to do with hysteria or a slowly progressive sclerosis. The data furnished by clinical examination may be throughout the whole course of the disease quite inconclusive, and we may have only that general impression, so impossible to describe in words yet of such value in estimating the condition before us, that the case is one of progressive structural disease of the central nervous system.

Before leaving the consideration of these various symptoms presented by neurasthenic and hysteric patients, a word must be said as to the hypothesis recently advanced by Strümpell in his remarkable article that many of them, including anæsthesia, contracted fields, exaggerated knee-jerks and an excitable pulse, are due simply to suggestion. Oppenheim's (24) answer that, in a large number of the cases, many of these symptoms are not evident except upon very careful examination, agrees fully with my own experience, and I have found in many cases that the patients had no idea that such symptoms existed. It is true that by suggestion we can often excite or remove symptoms in hysterical cases. If, however, a certain number of persons are subjected to accidents of a similar character, and, as a result, they all become nervous and suggestible, and are all subjected to examinations of a similar character, why should some present anæsthesias, contractures, paralyses and contracted fields, and others present none of these symptoms? Or, if, as Mendel claims (25), the æsthesiometer and perimeter, and careful and repeated examinations suggest and develop these hysterical stigmata, why, in a well-conducted clinique where all patients are thoroughly examined, or in the private practice of men who habitually make thorough examinations, should not these symptoms be more common?

We are not yet in a position to say why one patient becomes hysteric, and another neurasthenic, as a result of an injury, but it is certainly more reasonable to seek the difference in the patient's previous condition or in factors connected with the accident itself than in the events which

follow some time later after some indications of disease have already appeared, and it is most unreasonable to ascribe these symptoms simply to the ordinary methods of examination.

The prognosis of these two affections still remains uncertain, and our judgment in any given case is liable to error. I have made some effort to collect definite statistics concerning a considerable number of cases in order to obtain accurate knowledge. It is needless to say that the task is difficult, and I can here give only my own knowledge of the subsequent history of a certain number of cases of hysteria, as the information collected with regard to the neurasthenic cases is still scanty. I can speak of twenty-eight cases of hysteria, eighteen of which were litigation cases and ten of which were non-litigation cases. Of the litigation patients one has recovered completely, five have improved considerably, but still complain and show hysterical symptoms, seven have not improved, and five have died. Of these five, one died from unknown causes, one had considerably improved but died of intercurrent pneumonia, one was steadily growing worse and died of a low form of pneumonia, and two died of exhaustion due to hysteria. Of the non-litigation patients, three have completely recovered, four have improved, two have not improved, and one has died of intercurrent pneumonia. The one litigation patient who made a complete recovery, grew somewhat worse after the case was settled, and then, after a good many months, had a bad carbuncle on the back of the neck. On recovering from this he made a complete recovery from the hysteria. It will be seen from these figures that, so far as my own investigations have gone, the remarkable cases that recover as soon as damages are paid do not appear, although we must admit the possibility of a sudden disappearance of some or all the symptoms in a genuine case of hysteria. It is well, too, to accept with reserve the statement that a patient is well because one or several trustworthy persons have seen him walking about and attending to his affairs—a truism which would hardly seem necessary to utter to any one at all familiar with nervous diseases. If a patient still presents

the stigmata of hysteria, even though he make no complaint and attend to his affairs, he cannot be considered as a healthy man.

The much more favourable outcome of the non-litigation cases is due in part to the absence of the disturbing factors attending litigation. It must be borne in mind, however, that these cases were for the most part of a milder character, due to a less severe injury, and that the patients presented themselves for treatment early in the progress of their trouble. Even under these circumstances, however, some of the cases have not improved, but, on the contrary, have shown a progressive deterioration.

The guarded nature of the prognosis which must be rendered in a given case of traumatic hysteria is therefore evident, and it is confirmed by the long duration which we note in individual cases, a number of the cases tabulated having continued for from six to twelve years, often with some improvement, but still presenting distinct symptoms and not having regained their previous health.

There seems to be no very definite relation between the severity of the symptoms in hysteria and the gravity of the prognosis. Certainly there is no connection between the intensity of the stigmata and the subsequent history of the same. One of my fatal cases and several very protracted and obstinate cases presented stigmata so slight as to be detected only after careful examination. I am therefore much more inclined to believe that the prognosis of hysteria depends, as Binswanger states (26) of neurasthenia, not so much upon the intensity of the symptoms as upon their course of development and duration. The longer, therefore, hysteria lasts the less likelihood is there of complete recovery.

I have already stated that I am as yet unable to give definite statistics as to the subsequent history of a considerable number of cases of neurasthenia. Some of the cases certainly progress more favourably under treatment than do the cases of hysteria; and, on the whole, I think the prognosis more favourable. But, on the other hand, I have known of cases of long duration (eight, twelve, fifteen and even twenty years), so that the possibilities of grave results should always

be borne in mind. Von Hösslin (27), indeed, speaks of the prognosis in traumatic cases as much graver than in cases of a non-traumatic nature, but this seems distinctly doubtful.

In the discussions held last summer in Germany, the general opinion was that the best treatment in these conditions was to keep the patient at work. It is undoubtedly true that in our enthusiasm for the rest-cure we are prone to overlook the fact that in a considerable number of neurasthenic and hysteric cases a work-cure is much more indicated. In the majority of cases it is also true that if the patient be allowed to sit or lie unoccupied and to brood upon his troubles, to make no effort, to study every symptom and to receive the suggestions and sympathy of his friends, his condition will rapidly become worse; but this is not the rest cure. In more than one case I have seen the condition aggravated by ill-advised attempts to work or by the necessity of daily labour to obtain a living. The good results in a number of non-litigation cases seen at the hospital were due directly to the prompt inauguration of treatment, the enforced isolation and the complete rest. With this, of course, should go a proper psychical treatment, constant encouragement, occupation and diversion, and a very guarded attempt to resume the normal occupations of life. It is often said that no improvement is likely to take place while litigation is pending. This is in a measure true, as the worry attendant upon litigation acts as a constant source of irritation; but I have in spite of this seen patients who were put under proper treatment make a very considerable gain in spite of the continuance of litigation. Within a very short time I saw a patient who was under very bad psychical influences and was steadily growing worse. Removal to the hospital, where he could be isolated and kept from these bad influences, produced a remarkable improvement, although the trial of his case was imminent.

The admitted evil influence of litigation, however, indicates to us a duty which we owe to the victims of accident. The claimants for damages have a bad name, and they are generally regarded by the outside public as swindlers and extortioners. The facts, however, show that in the great

majority of cases this stigma is undeserved. Very few of them are simulators, most of them are genuine sufferers, a considerable number of them never fully recover, and, in spite of the sensational reports from Chicago which have appeared in a recent number of a popular magazine, the amount which they receive in damages is, in my experience, inadequate, especially after the expenses of litigation are deducted.[1] It is, moreover, admitted by the majority of recent writers that the prognosis in these affections becomes graver the longer the disease lasts. In one case under my observation the injury was received in March, 1890; the case was tried in January, 1892, and it is still pending before the Supreme Court on appeal. Another case, when the patient was injured in September, 1892, has just come to trial. Such cases are perhaps extreme, but it rarely happens that a case which comes to trial in Boston, ends within two years from the time of injury. These delays are a direct source of harm to the patients and undoubtedly serve to turn the scale against recovery in a certain number of cases. It is, of course, hopeless to expect any reform in legal procedure, but it still remains our duty to protest against its evils, and to seek to obviate them by urging a prompt settlement of the patient's claims out of court, in every case when it can be accomplished without too great a sacrifice.

[1] In twenty cases of hysteria and ten of neurasthenia under my observation the average award was £1,100, the highest being £4,500. Of this it is probable the patient generally gets less than half.

REFERENCES.

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(2) STRÜMPELL. "Ueber die Untersuchung, Beurtheilung and Behandlung von Unfallkranken," Munich, 1896.

(3) The most important, and indeed almost the only Italian article upon the subject of which I have any knowledge is the excellent critical digest by SEPPILLI in the *Rivista Sperimentale di Freniatria*, XVII., p. 70, 1891.

(4) Guinon. "Les agents provocateurs de l'hystérie." Paris, 1889.
(5) Janet. "État mental des hystériques." "Les stigmates mentaux." "Les accidents mentaux." Paris, 1892, 1894.
(6) Breuer and Freud. "Studien über Hysterie." Leipzig, 1895.
(7) Dana. *American Journal of Medical Sciences*, October, 1890. "The Traumatic Neuroses." "Hamilton's System of Legal Medicine," II., p. 340.
(8) Pitres. "Leçons cliniques sur l'hystérie et l'hypnotisme," I., p. 125.
(9) Charcot. "Leçons du Mardi," II., p. 138.
(10) Wilbrand und Sänger. "Ueber Sehstörungen bei functionellen Nervenleiden," p. 73.
(11) Löwenfeld. "Pathologie und Therapie der Neurasthenie und Hysterie," p. 154.
(12) Charcot. *Op. cit.* II., p. 15.
(13) Charcot. *Op. cit.*, II., p. 31.
(14) Patrick. *New York Medical Journal*, February, 1896.
(15) Charcot. "Clinique des maladies du système nerveux," I., p. 27.
(16) Gilles de la Tourette. "Traité clinique et thérapeutique de l'hystérie," I., p. 218.
(17) Thomsen und Oppenheim. *Archiv. fur Psychiatrie*, XV., p. 559.
(18) Charcot. "Leçons du Mardi," II., p. 469. "Clinique des maladies du système nerveux," I., p. 294.
(19) Souques. "Contribution à l'étude des syndromes hystériques 'simulateurs' des maladies organiques de la moelle épinière," p. 7.
(20) Mitchell and de Schweinitz. *American Journal of the Medical Sciences*, November, 1889.
(21) Patrick. *Art. cit.*
(22) Rumpf. *Neurologisches Centralblatt*, June 15, July 1, 1896.
(23) Buzzard. "On the Simulation of Hysteria by Organic Disease of the Nervous System."
(24) Oppenheim. "Der Fall N."
(25) Mendel. *Neurologisches Centralblatt*, October 15, 1896.
(26) Binswanger. "Die Pathologie und Therapie der Neurasthenie," p. 338.
(27) Müller. "Handbuch der Neurasthenie," p. 197.

PROCEEDINGS OF THE SOCIETY.

At a Pathological Meeting held on October 28th, the following Communications were made :—

Drs. Sanger Brown and Adolf Meyer.—"Morbid Anatomy in a case of Hereditary Ataxy of the type described by Sanger Brown." (Communicated by Dr. J. A. Ormerod.) The following changes were described :—

(1) Excess of corpora amylacea in the posterior roots and posterior half of the cord, and in the superficial parts of the anterior columns.

(2) Increase of neuroglia in the subpial layer, and in the degenerated parts of the cord.

(3) Certain minute changes in the cells of Clarke's columns (not shown in present specimens).

(4) Degeneration (limited to the upper parts of cord) in *certain portions of the posterior columns*, viz., near mesial septum, superficial parts of Goll's columns, borders of Burdach's and Goll's columns; in the cerebellar tracts, and slightly in the antero-lateral tracts.

(5) Similar degeneration in the lower part of medulla.

(6) In some districts of cerebellar cortex, decrease in the number of Purkinje's cells.

Drs. Samuel Gee and H. H. Tooth.—"Case of Hæmorrhage in the region of the pons Varolii almost entirely on the right side."

The lesion has caused complete destruction locally of—(1) The right lemniscus, with many transverse fibres of the pons but not the pyramids. (2) The right posterior longitudinal bundle completely, and partially (? by pressure) the left. (3) Cranial nerves right v. motor, vi. and vii.

Secondary degeneration (1) Of the right lemniscus brainwards to the optic thalamus, also to a lesser degree of the left. (2) Of the posterior longitudinal bundles brainwards to the optic thalamus, showing also a crossing of the fibres in the third nucleus; posteriorly of the same bundles to become continuous with, or identical to, the anterior ground bundles of the

lower medulla, the post-pyramidal decussation of the lemniscus being undegenerated. (3) Of the white matter of the flocculus of the cerebellum.

Dr. W. A. Turner.—"Symmetrical Softening of the Pyramids and Inter-olivary Strata." From a case of cerebral syphilis, arising probably from blocking of a branch or branches of the basilar artery. The softening extends from the lower end of the pons Varolii to the decussation of the pyramids.

Dr. F. E. Batten.—"The Effect of Marchi's fluid on Nervous Tissue which has undergone *post-mortem* change." The author showed that under the ordinary *post-mortem* conditions no changes take place which are liable to be mistaken for pathological appearances.

Dr. James Taylor and Mr. C. A. Ballance.—"Case of Tumour of the Right Lateral lobe of the Cerebellum." Forced movements to the same side, and symptoms of intense intra-cranial pressure.

Dr. Risien Russell.—(1) Tumour of the lumbro-sacral cord, with degeneration of the posterior columns on both sides, and the antero-lateral ascending tract on one side. (2) Degenerations in the spinal cord after removal of a large tumour from one cerebral hemisphere. Though degenerate fibres were present in the crossed pyramidal tract on the side of the lesion, none could be seen passing to it at the decussation of the pyramids, where all the degenerate fibres appeared to cross to the opposite lateral column. The degeneration in the direct pyramidal tract on the side of the lesion was traced into the sacral cord.

BRAIN.

PART IV., 1897.

Original Articles and Clinical Cases.

THE ORIGIN AND DESTINATION OF CERTAIN AFFERENT AND EFFERENT TRACTS IN THE MEDULLA OBLONGATA.

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CONTENTS.

(1) INTRODUCTION.

THE experimental researches on which this paper is based were carried out in the Pathological Laboratory of University College, when I was Research Scholar to the British Medical Association, and during the time that the

laboratory was under the directorship of Professor Victor Horsley; I therefore take this opportunity of thanking him for the privilege which I then enjoyed. The microscopical examination of the central nervous system of the animals then operated on has been made subsequently.

The question with which I specially intend to deal is that with regard to the existence of efferent tracts in the spinal cord other than those belonging to the pyramidal system.

In addition to this question of efferent tracts in the spinal cord, the subject of afferent tracts degenerating in the medulla, pons and mesencephalon, after various lesions of the medulla, will also be discussed, in view of the results which have been obtained in this connection.

I do not propose to refer in detail to the extensive literature relating to the questions about to be dealt with in this paper, but intend to allude to those investigations, more especially recent ones, which bear directly on the points which I have investigated. This will be done in the discussion which will be found in connection with each part of the paper after the results of the experiments have been detailed, and also in a more general discussion on the question of efferent tracts in the spinal cord. The results of Biedl[1] will, however, be rather more fully dealt with, in that this observer has called into question certain of my negative results in connection with ablation of the cerebellum,[2] which have a direct bearing on the questions about to be discussed in this paper.[3]

(2) Methods of Research.

Dogs and monkeys (Macacus rhesus and sinicus) were alone employed in the investigations which form the subject of the communication. In every instance the animal was

[1] Biedl, *Neurolog. Centralblatt*, 1895, vol. xlv., p. 434.

[2] Risien Russell, *Phil. Trans. Roy. Soc.*, 1894, vol. clxxxv., B., p. 819.

[3] Since this paper was written an important monograph by Dr. André Thomas has appeared, entitled "Le Cervelet, étude anatomique, clinique et physiologique," Paris, 1897. This observer confirms Marchi's results as regards the existence of an efferent antero-lateral tract in the spinal cord, derived directly from the cerebellum.

rendered unconscious by the administration of ether by inhalation, and narcosis was maintained throughout the operation. The hair was shaved from the back of the head and neck, the skin then cleansed by soap and water, and subsequently thoroughly disinfected by perchloride of mercury lotion (1 to 1,000). A median incision was made from the superior occipital protuberance to about the mid-cervical region posteriorly, and the muscles were then scraped from their attachments to the occipital bone and to the upper cervical spines and arches. Bone forceps were then used to expose the posterior portion of the cerebellum, the medulla and upper part of the cervical cord, up to which point instruments first sterilised by boiling, and subsequently kept in carbolic lotion (1 to 40), were used, and the wound was washed with perchloride of mercury lotion (1 to 2,000), sterilised cotton wool being used instead of sponges. As soon as all bleeding had been arrested and the dura mater was about to be opened, boiled normal saline solution was used to freely wash out the wound, and from this point in the procedure no antiseptic lotion was employed, the instruments and the cotton wool used for sponges being both kept in boiled normal saline solution. On the importance of this I have sufficiently insisted elsewhere[1] which makes it unnecessary for me to do so again here. The posterior part of the middle and one lateral lobe (in every instance the left) of the cerebellum were gently raised by means of a small elevator, and if my object was to divide the inferior peduncle of the cerebellum as high up as possible, or to cut out Deiters' nucleus, then the cerebellum had to be raised considerably more than in those instances in which a lesion to the lateral region of the medulla, or to the posterior columns and their nuclei, was to be produced. In the case of the lateral medullary lesion it was necessary to gently displace the medulla laterally towards the opposite side, and to rotate or raise it slightly to enable me to produce the lesion sufficiently ventrally to avoid injury to the restiform body. Every care was, of course, taken not to injure the spinal accessory or hypoglossal roots. A delicate,

[1] *Loc. cit.*

narrow-bladed knife was used to divide the peduncle, to cut out Deiters' nucleus, and to divide the posterior columns and their nuclei; this was also used to divide the lateral tracts of the medulla in some instances, but on other occasions the lesion of the medulla was produced by inserting a needle just dorsally to the position of the inferior olive, pushing it about two to three millimetres towards the cephalic end of the medulla and then moving the point of the needle so inserted from side to side in different directions so as to destroy the region into which the needle point had entered, every care being, of course, taken to prevent injury to the pyramids, both when the knife was used and when the needle was substituted.

The skin wound was subsequently sutured by means of sterilised horse-hair, and an antiseptic dressing applied after further washing of the skin surface with an antiseptic lotion. Three weeks to a month after this the animals were killed by an overdose of chloroform, administered by inhalation, the central nervous system at once removed and placed in Müller's fluid, and subsequently prepared for microscopical examination by means of the Marchi method, which is now too well known to call for special description here.

(3) Plan of Research.

The following experimental procedures were adopted in attempting to arrive at the facts with regard to the origin and destination of some of the afferent and efferent tracts in the medulla oblongata.

(1) Section or destruction of the lateral region of the medulla oblongata between the ascending root of the fifth nerve and the inferior olive.

(2) Division of the restiform body.

(3) Division of the direct sensory cerebellar tract of Edinger.

(4) Severance of Deiters' nucleus from its connection with the medulla.

(5) Section of the posterior columns and their nuclei in the medulla.

Fig. 1.

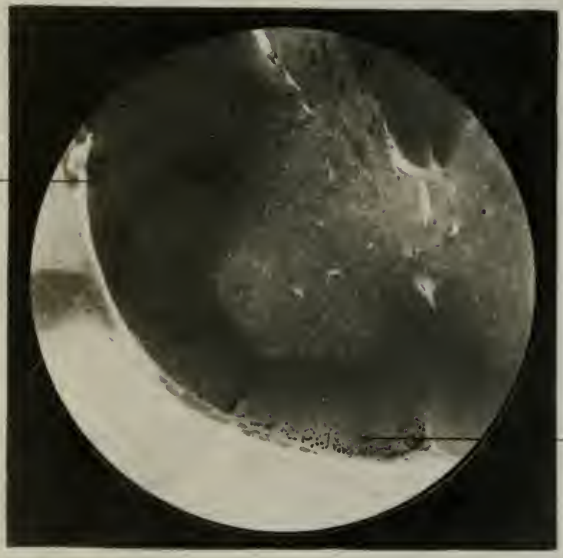

Fig. 2.

Part I.—Section or Destruction of the lateral region of the Medulla Oblongata between the Ascending Root of the Fifth Nerve and the Inferior Olive.

As may be seen by reference to fig. 1, the lesion produced in the lateral region of the medulla is so situated that the inferior olive has just escaped injury and there is no sign of any injury to either pyramid; indeed, the way in which I produced the medullary lesion precluded any possibility of injury to the pyramid without first injuring the olive. In the section from which the photo-micrograph has been taken the only degeneration present, other than that in the direct neighbourhood of the lesion, is a well marked degeneration of the opposite fillet seen lying immediately dorsal to the opposite pyramid, a degeneration obviously due to interruption of the arciform fibres, by the medullary lesion, in their passage from the posterior column nuclei on the side of the lesion to the opposite fillet. As will subsequently be seen, the absence of injury to the opposite pyramid is of even more importance in relation to the subject under discussion than is the fact that the pyramid on the side of the lesion is intact.

(*a*) *Efferent Tracts.*—From the lesion in the lateral region of the medulla a well marked descending degeneration may be traced into the spinal cord; that this extensive band of degeneration consists of two distinct efferent tracts will subsequently become evident, but in the cervical region of the spinal cord from which the photo-micrograph represented in fig. 2 was taken, the degenerated fibres apparently constitute a single extensive tract, which, commencing dorsally in a well marked triangular area of degeneration, situated just ventrally to the crossed pyramidal tract and internally to the anterior part of the direct ascending cerebellar tract and the posterior part of the afferent antero-lateral tract of Gowers, sweeps round the periphery of the antero-lateral region of the cord to the anterior median fissure. Continuous as this area of degeneration appears to be, there are two regions in which it is more extensive than elsewhere; one of these is its posterior limit, where it is

triangular in shape, the apex of the triangle pointing inwards towards the gray matter of the lateral horn, and ventrally at a point just external to the anterior fissure where the band of degeneration becomes nearly twice the depth of the narrow band situated at the periphery of the cord and connecting these two points of more extensive degeneration. As may be seen in fig. 2, the degeneration does not extend to any extent into the region of the direct pyramidal tract as it abuts on the anterior median fissure, or indeed into any part of the area of the cord occupied by the direct pyramidal tract in man, as I hope to be able to show by comparison subsequently. A cursory examination of fig. 2 suffices to convince one that the crossed pyramidal tract is encroached on by the degenerated fibres forming the posterior limit of the triangular area of degeneration already alluded to.

As the thoracic region of the spinal cord is reached it becomes more and more evident that the area of degeneration under consideration is really composed of two tracts; the narrow band of degenerated fibres which connects the posterior triangular area with the area situated just external to the anterior tip of the anterior median fissure, becomes gradually thinned out (see fig. 3) until a breach of continuity becomes evident, separating the area of degeneration situated ventrally from that situated dorsally to it. The dorsal area of degeneration, while still retaining some evidence of the triangular form so well seen in the cervical region of the cord, now becomes much more scattered and thinned out, a few degenerated fibres are scattered towards the region of the crossed pyramidal tract, but by far the greater tendency of spread is in the shape of a narrow band occupying the periphery of the cord external to this tract. Very little alteration is to be noted in the area of degeneration close to the anterior tip of the anterior median fissure, which occupies an area of almost the same breadth as in sections taken from higher levels, and shows no more evidence of extending along the margin of the anterior fissure or in any other way encroaching on the domains of the direct pyramidal tract. Evident as was the separation

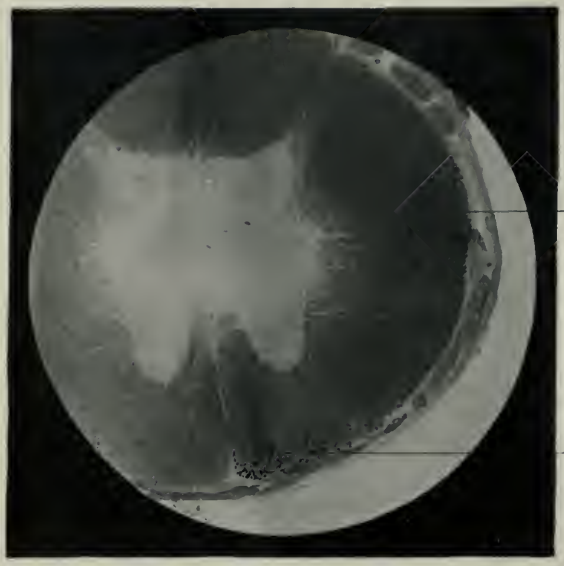

FIG. 3.

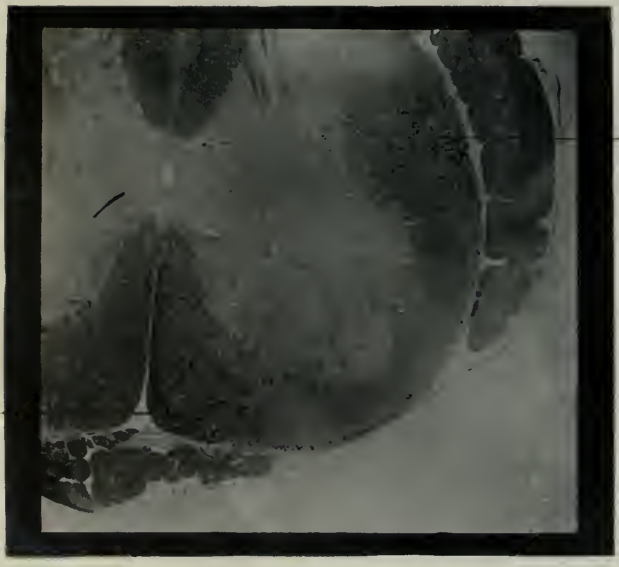

FIG. 4.

between the two tracts on examination of the thoracic cord, the picture presented by sections taken from the lumbar region of the cord, and shown in fig. 4, leaves no possible room for doubting that we are dealing with two separate and distinct tracts of degenerated fibres. The whole of the narrow band of degeneration occupying the periphery of the cord, so well seen in the cervical region connecting the two major areas of degeneration, has now completely disappeared and there remain two distinct and separate tracts, the one situated in close proximity to the crossed pyramidal tract, and the other at the periphery of the cord close to the anterior median fissure. The posterior area now forms a much more compact bundle than was the case in the thoracic region, and it again assumes most distinctly the triangular form so characteristically seen in the cervical region of the cord; but instead of the base of this triangular area of degeneration being separated from the periphery of the cord by a distinct band of undegenerated nerve fibres, as was the case in the cervical region, it now comes quite to the surface in the greater part of its extent, only a limited portion of the tract being separated from the margin of the cord posteriorly by undegenerated fibres. The apex of the triangular area, as before, points inwards towards the grey matter of the cord. So nearly does this area of degeneration in the lumbar cord correspond to the region occupied by the crossed pyramidal tract, that there must be considerable intermingling of the two systems of fibres.

As regards the ventral tract situated close to the anterior median fissure, there has been a marked thinning out of its fibres, and while the chief seat of degeneration is that which it occupied at higher levels, there is, nevertheless, a little more tendency for the degenerated fibres to spread along the margin of the anterior fissure, but they only extend to rather less than half way to the anterior commissure, and even up to that point are by no means plentiful.

Before considering this anterior tract further it will be convenient to dispense with the lateral tract that has occupied our attention. Throughout its course in the spinal cord degenerated fibres may be traced from it to the

grey matter of the anterior horn, the bulk of these fibres apparently passing to the part of it known as the lateral horn. No evidence has been obtained that these fibres decussate at any point in their course from the lesion in the medulla; they maintain a direct course on the side of the lesion throughout the spinal cord, and only pass to the grey matter of that side of the cord; none of the fibres could be traced across to the opposite side even in the grey matter.

That the tract under consideration is probably identical with that which was described by Boyce[1] in the cat after hemisection of the mesencephalon, in which his lesion passed between the anterior and posterior quadrigeminal bodies, is rendered extremely probable by a comparison of fig. 2 with fig. 5, plate 3, in Boyce's monograph on the subject. The position, shape, and general characters of the tract, as seen in the cervical region, are practically identical with that described by Boyce; there is, however, this difference with regard to our observations, Boyce could find no evidence of the existence of his tract caudal to the upper thoracic region of the spinal cord where it appeared to end, whereas, as has been seen, the tract to which I am now calling attention has been traced throughout the spinal cord to the lowest part of the lumbar region. This may indicate a fundamental difference between the two tracts, as is further suggested by the fact that the tract described by Boyce is one originally derived from a decussating system, whereas at no point in its course from the medulla throughout the spinal cord have I been able to find any evidence that the tract I am describing decussates. In spite of these apparent differences I am inclined to regard the tract I have found as belonging, at any rate, to the same system of fibres as that described by Boyce, even if it cannot strictly be regarded as the continuation and termination of that tract.

A similar tract has been described by Ferrier and Turner[2] as degenerating from the nucleus lemnisci lateralis, leaving the lateral fillet at the level of the motor nucleus

[1] *Phil. Trans. Roy. Soc.*, 1895, vol. clxxxvi., B., p. 321.

[2] Ferrier & Turner, *Phil. Trans. Roy. Soc.*, 1894, vol. clxxxv., B., p. 719.

of the fifth nerve, and traced by them as far as the sixth thoracic segment of the spinal cord.

Boyce found that the degenerated fibres which formed the tract in question passed from the side of the lesion to the opposite side in Forel's decussation and then passed caudalwards, and Held[1] speaks of fibres originating in the red nucleus, which pass caudalwards to form the lateral column. It is quite conceivable that an "internuncial" system, even of long fibres, may only extend for a limited distance of the full length of that system, and that there should be a constant accession of fibres to the system originating at levels further caudal to the level at which the highest fibres arise, and extending further caudalwards than these fibres, though not of necessity longer than the fibres arising farthest cephalwards and which belong to the same system.

That this is not too great an assumption is made evident by the fact that even in the pyramidal system such an accession occurs; thus in a cat whose nervous system I examined microscopically[2] and in whom the pyramid was completely absent on one side, as seen in sections through the mesencephalon, pons and upper part of the medulla, fibres entered this system from lower levels, and were not derived by decussation from the opposite intact pyramid. The evidence obtained in this animal was to the effect that there is an accession of fibres to the pyramidal system as lower and lower levels are reached, and that all the fibres contained in the pyramids are not derived from the cerebral hemispheres. The same condition has been found to occur in man.

If such a state of things is possible in connection with a system like that of the pyramidal fibres, how much the more likely is it to occur in connection with an "internuncial" system, such as that which we have been considering.

That a system of fibres other than pyramidal ones existed in close relationship to the crossed pyramidal tracts was

[1] Held, *Archiv. fur Anat. u. Physiol.*, 1892.
[2] Brain, 1895, vol. xviii., p. 37.

surmised by Loewenthal,[1] who performed hemisection of the posterior part of the medulla of the kitten at the level of the decussation, with the result that there was atrophy of large fibres upon the ventral and external aspect of the pyramid, which he believed did not belong to this structure. Bechterew[2] has shown that a system of fibres in this situation myelinate earlier than the pyramidal fibres. So, too, Bouchard[3] noticed that in man the descending degeneration in the lateral column was greater after injury to the medulla than after injury higher up, and that the medullary fibres were situated at the lateral border of the pyramidal tract. It is clear, however, that, as we have already seen, the fact that there is a constant accession of fibres to the pyramidal system as lower and lower levels are reached, makes it probable that some of the fibres which attracted the attention of these observers were derived from this source, as well as from the system which I am dealing with, for the pyramid was of course injured in the instances from which their observations were derived.

It will, I think, be well to defer the discussion of the direct antero-lateral tract which engaged our attention earlier in the paper, until we have considered the results of cutting out Deiters' nucleus; but before leaving this part of the subject it may be well to emphasise one or two points in connection with this tract. It ought to be clearly remembered that its position is one which does not encroach on the true limits of the direct pyramidal tract to any extent, that its fibres do not extend to any extent along the margin of the anterior median fissure, that it extends throughout the spinal cord to the lowest part of the lumbar region, and that its fibres pass to the grey matter of the anterior horn of the same side, without any evidence of their decussating in the anterior commissure or by any other route. It will be found of extreme importance to keep all these points clearly in mind when we come to further discuss this tract in connection with an apparently similar tract which has been derived from other sources.

[1] Loewenthal, *Rev. Med. de la Suisse Romande*, 1886.
[2] Bechterew, "Die Leibungsbahnen," 1894, p. 47.
[3] Bouchard, *Archiv. gén. de Méd.*, 1886.

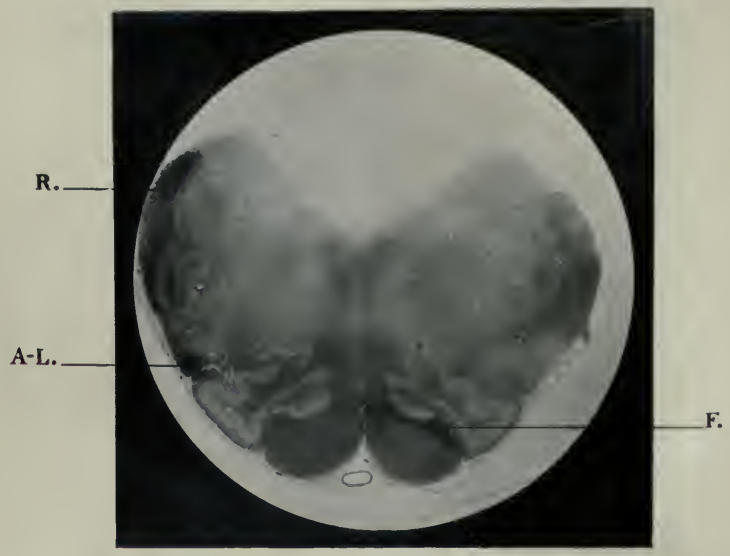

FIG. 5.

FIG. 6.

(*b*) *Afferent Tracts.*—There still remains to be described the afferent tracts which were found degenerated after a lesion to the lateral medullary region. Close to the seat of lesion nearly the whole of the periphery of the lateral border of the medulla is occupied by degenerated fibres, but on passing farther towards the brain this more or less complete band of degenerated fibres becomes more and more separated into two distinct groups, until they eventually are no longer connected with each other. The most dorsal of these groups of degenerated fibres occupies the confines of the restiform body, while the ventral group of fibres occupies the position of the afferent ventral cerebellar tract (see fig. 5). The former group naturally passes to the cerebellum in the restiform body, some of the fibres to the middle lobe to end on the same side, and others through this to the opposite side, to end eventually on the ventral aspect of the vermis in the region of the nuclei globosi.

The ventral tract of degenerated fibres passes through the pons as a well defined bundle occupying the region external to the superior olive, and being situated on the ventral side of the emergent root of the seventh cranial nerve, and further forward immediately on the ventral side of the emergent root of the fifth nerve. These degenerated fibres pass round the fifth nerve and course along the inner side of the transverse fibres of the pons (middle cerebellar peduncle), and eventually reach the middle lobe of the cerebellum by turning back over the superior peduncle of the organ.

This tract follows so closely the course which the ventral cerebellar tract has been described as taking, that it is probable that they are identical; accordingly these results confirm the observations of Loewenthal,[1] Mott,[2] Auerbach,[3] and Tooth.[4]

But, in addition to the portion of this ventral cerebellar tract which reaches the cerebellum, another portion has yet

[1] Loewenthal, *loc. cit.*
[2] Mott, Brain, 1892 and 1895.
[3] Auerbach, *Virchow's Archiv.*, Bd. 121, 1890, p. 199.
[4] Tooth, Brain, 1892, p. 397.

to be described, which lies internally to this more compact bundle, whose course has been already traced, as seen on transverse section at the levels of exit of the seventh and fifth cranial nerves. Though forming a distinct tract of degeneration, these fibres are fewer in number and more scattered than the other bundle, from which they are pretty clearly separated at the level of the exit of the fifth nerve. At this level they are close to the outer end of the fillet, which is otherwise free from degeneration on this side, and in their further course towards the mesencephalon they remain closely associated with this system of fibres. In the region of the posterior corpora quadrigemina they form a well marked though scattered band of degeneration, following more or less the distribution of the fillet, and in the region of the anterior quadrigeminal bodies they are still to be seen as a well marked though scattered tract chiefly occupying the region of the dorsal end of the fillet. Indeed, a few of the most dorsal of these fibres more scattered than their fellows, can be seen coursing inwards towards the middle line, and can be traced to a point a little external to the position of the descending root of the fifth. It will thus be seen that this result is in entire accord with the observations of Mott[1] with regard to this portion of the ventral cerebellar tract, and is quite opposed to the view expressed by Patrick[2] who was unable to find any such degeneration in the anterior quadrigeminal region, and accordingly concluded that Mott's results were probably due to an artifact.

In that the lesion interrupted a considerable proportion of the arciform fibres in the formatio reticularis, well marked degeneration of these fibres passing to the opposite inter-olivary layer was found. On being traced forward through the pons these degenerated fibres were found to occupy both the median and lateral fillet. The degeneration of the fillet is well marked, and can be traced with ease through the mesencephalon to the optic thalamus.

In addition to these arciform fibres which pass to the opposite inter-olivary layer, other fibres are interrupted in

[1] Mott, *loc. cit.*
[2] Patrick, *Journal of Nervous and Mental Diseases*, February, 1896.

their course through the formatio reticularis, notably those passing to the inferior olives, which fibres are, of course, degenerated and can be clearly seen passing to these structures.

Part II.—Degenerations Consequent on Division of the Restiform Body.

The description of the cerebellum, as having three peduncles, appears to me to be erroneous and misleading. The possibility of distinguishing four peduncles by which this organ is connected with contiguous structures is so clear that it is difficult to see why two of these paths of connection should be considered as one. The direct sensory cerebellar tract of Edinger is a structure entirely separate and distinct from the restiform body, and ought to be so regarded. Anatomically they stand out clearly and distinctly as two definite structures, having little if any resemblance, and having connections totally distinct from each other. Embryologically it has been found that the fibres of the direct sensory cerebellar tract receive their medullary covering at a different period to the fibres of the restiform body. Experimentally I have shown that ablation experiments on the cerebellum are followed by degeneration in the medulla oblongata limited accurately to the confines of the restiform body strictly so-called, and as opposed to the direct sensory cerebellar tract which lies to the inner side of the restiform body; and I hope to bring forward further evidence later in this paper in support of the same contention. So, too, in all the experimental evidence there is with regard to ascending degeneration reaching the cerebellum after interruption of afferent tracts in the spinal cord, the degenerated fibres have been accurately limited to the confines of what ought strictly to be looked on as the restiform body, and do not in any way encroach on the area occupied by the direct sensory cerebellar tract of Edinger, or become in any way connected with this tract.

Throughout this paper, then, I shall, in referring to the restiform body, mean the structure "R," shown in fig. 6,

as opposed to the direct sensory cerebellar tract "E," seen in the same figure lying to the inner side of the restiform body.

Looked at in this way, the cerebellum must be regarded as having four peduncles—an anterior, a middle, and two posterior, or, as I should prefer to regard it, a posterior (restiform body) and an inferior (direct sensory cerebellar tract).

With this conception of the connections of the cerebellum clearly before us, the degenerations consequent on section of the restiform body (posterior cerebellar peduncle) are as follows :—

Of course, a well-marked degeneration of afferent fibres (passing to the cerebellum) follows such a lesion, but with their precise distribution in that organ we are not at present concerned. The points to which I wish rather to direct attention are in connection with efferent fibres, which degenerate after division of the restiform body. These degenerated fibres are strictly limited to the confines of this tract at first, as after ablation experiments on the cerebellum. In passing caudalwards they occupy more and more of the periphery of the medulla, external to the ascending root of the fifth nerve, owing to some of the fibres becoming situated more and more ventrally as lower levels of the medulla are reached. Fibres from this system pass to end apparently in the formatio-reticularis of the same side of the medulla, while others pass to the olive on the same side and to that on the opposite side. No degenerated fibres could be found passing to the spinal cord by this route, an observation which is in harmony with the results of Ferrier and Turner[1] after division of the restiform body. It will thus be seen that the degenerated fibres met with in this system conform as regards distribution in all respects with those which leave the cerebellum by way of the restiform body after ablation experiments conducted on that organ.

Biedl[2], in supporting Marchi's view that a descending antero-lateral tract degenerates in the spinal cord after

[1] *Loc. cit.* [2] *Loc. cit.*

cerebellar lesions, considers that the fact that I met with a few scattered degenerated fibres in the upper cervical region of the spinal cord is in itself evidence against my contention that no such antero-lateral tract, derived directly from the cerebellum, exists. Now, the exceedingly few degenerated fibres which I described as having been met with in the upper cervical cord were only found in some instances, and knowing how easy it is to get slight involvement of Deiters' nucleus in cerebellar lesions, I prefer to look to such a slight accidental complication as accounting for the existence of the insignificant number of degenerated fibres met with in the upper part of the spinal cord. Certainly nothing could be more erroneous than any interpretation of my results of ablation of the cerebellum which finds in them evidence of a descending antero-lateral tract in the spinal cord having its origin in the cerebellum. The contrast between these results and those to be presently described as resulting from injury to Deiters' nucleus is so striking that it is difficult to believe that this tract has its origin in the cerebellum.

Biedl also appears to be under the impression that I denied the existence of fibres degenerating from the cerebellum by way of the restiform body to the opposite inferior olive. The comment in my paper on the subject was that the number of fibres passing to the opposite olive was much smaller than was expected, and did not form so large a tract as that which appeared to have been met with by Ferrier and Turner.

Part III.—Degenerations following Section of the Direct Sensory Cerebellar Tract.

There appears to be little room for doubt that the degenerations met with after section of the direct sensory cerebellar tract are in reality due to the implication of Deiters' nucleus by this lesion. Imbedded as it is, in the fibres of the direct sensory cerebellar tract, it is an almost impossible task to divide this tract without injury to the nucleus. The only way to accomplish division of the tract without injury to the nucleus is to make the incision as

close to the cerebellum as possible, a procedure attended in its turn by the risk of injury in the cerebellum of structures other than the tract in question. In only one of my experiments am I able to satisfy myself that section of the direct sensory cerebellar tract has been accomplished without apparent injury to Deiters' nucleus (see fig. 7); in some the injury to the nucleus is slight, in others well marked, and belonging to the latter category are the cases in which I deliberately attempted to sever this nucleus from its connections with the medulla. The more obviously Deiters' nucleus was injured, the more marked were the degenerations about to be described, and the slighter the damage to the nucleus the less evident were these degenerations.

In that the only degenerations met with are precisely those which followed the cutting off of Deiters' nucleus from its connections with the medulla, it would be superfluous to describe these now, as they will have to be described in the next section of the paper, which deals directly with lesions of Deiters' nucleus.

No afferent fibres were found degenerating in the direct sensory cerebellar tract between the nucleus of Deiters and the nucleus globosus in the cerebellum, a fact which supports the view of Ferrier and Turner[1] that this is an efferent tract from the middle lobe of the cerebellum to Deiters' nucleus.

Part IV.—The Results of severing the connection of Deiters' Nucleus with the Medulla.

The lesion produced in this case is well shown in fig. 8, from which it will be seen how clean the line of incision was —free from any inflammatory complication and without any damage to neighbouring parts, such as the restiform body or the sixth nucleus, &c. Of the degenerations which result from such a lesion, that which concerns us in the first instance is the degeneration of a tract which leaves the seat of lesion and passes as a well-marked band of degeneration through the formatio reticularis, about midway between

[1] *Loc. cit.*

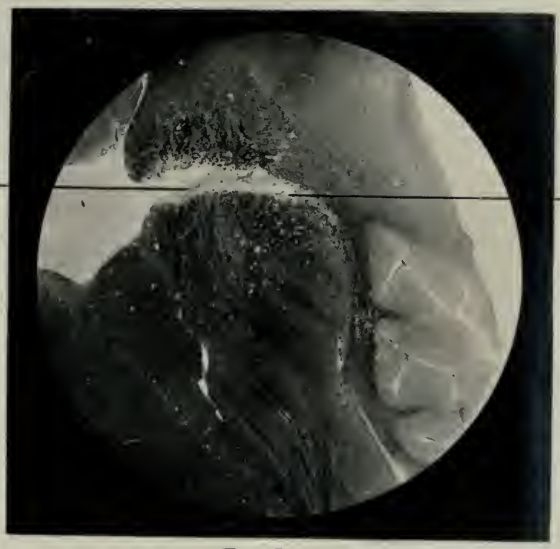

FIG. 7

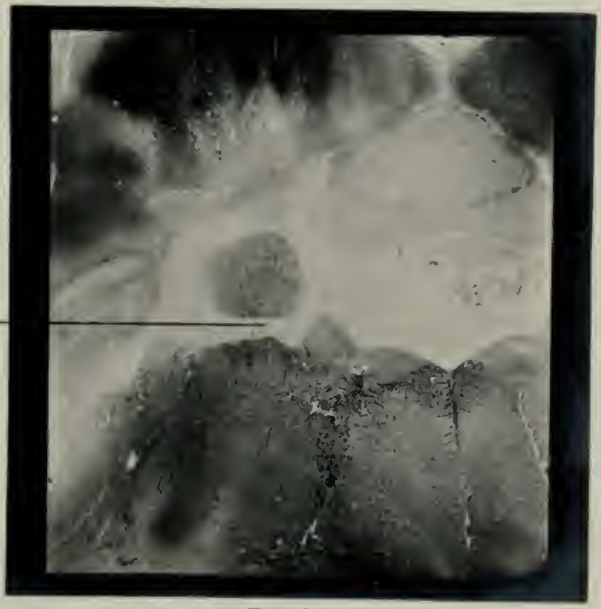

FIG. 8.

the ascending root of the fifth nerve and the raphe, and which occupies a considerable vertical extent between the dorsal and ventral aspects of the medulla, as seen on transverse section (see fig. 9). In passing caudalwards they are to be found forming a well-marked group of fibres, situated external to the inferior olive and internal to the nucleus lateralis. When the upper cervical region of the spinal cord is reached they are to be seen as a well-marked antero-lateral band of degeneration, occupying the periphery of the cord and extending forward to within a short distance of the anterior median fissure where the band ends; degenerated fibres from this source do not extend to the anterior median fissure, nor do they encroach on the region ordinarily supposed to be occupied by the anterior direct pyramidal tract (see fig. 10). On being traced further caudalwards in the spinal cord, however, this tract becomes blended with another, which we shall subsequently find is derived from fibres which reach the spinal cord by way of the posterior longitudinal bundle on the same side, and these two tracts together form an antero-lateral tract occupying the periphery of the cord, and extending down the margin of the anterior median fissure, thus encroaching on the area of the anterior direct pyramidal tract. The degenerated fibres become much more scattered in the thoracic cord, especially those in the anterior column and those at the periphery of the antero-lateral region of the cord, while still forming a well-marked band, are more scattered, so that the band is broader than that seen in the cervical region. In the lumbar cord there are next to no degenerated fibres left at the periphery of the antero-lateral region of the cord, but a well-marked band of degenerated fibres is situated along the margin of the anterior median fissure.

In their course through the spinal cord these degenerated fibres pass to the grey matter of the anterior horn of the same side. Evidence of the decussation of some of them in the anterior commissure to reach the anterior horn of the opposite side was found.

From certain considerations to be afterwards dealt with, it is probable that the degenerated fibres met with in the

lumbar region of the cord are derived from this system of fibres which we traced through the formatio reticularis in the medulla, and that none of those which pass to the anterior columns by way of the posterior longitudinal bundles reach the lumbar cord.

It is of interest to note that the degeneration as seen in fig. 10, while occupying the periphery of the antero-lateral region of the spinal cord does not become specially dense close to the anterior tip of the fissure, as was the case after a lesion to the lateral region of the medulla, and shown in figs. 2, 3 and 4.

The tract corresponds in position and general characters to that described by Marchi, as degenerating after ablation of the cerebellum.

Ferrier and Turner[1] failed to confirm Marchi's observation, but found that such a degeneration resulted when Deiters' nucleus was injured. In a previous investigation[2] I also failed to confirm Marchi's observation. It is interesting and instructive to note that while after ablation of one lateral lobe of the cerebellum, there is an entire absence of any degenerated fibres occupying the position of the tract seen in the formatio reticularis after a lesion of Deiters' nucleus, there is nevertheless a well-marked degeneration of fibres in the restiform body and peripheral region of the medulla occupied by fibres derived from that system, a region free from degeneration after injury to Deiters' nucleus. Further, after ablation of one lateral lobe of the cerebellum with well-marked degeneration in the restiform body, an antero-lateral efferent tract in the spinal cord was not found, while a well-marked tract as is to be seen in fig. 10, was obtained after the lesion to Deiters' nucleus, in which the restiform body was free from degeneration.

Mott,[3] when cutting off the posterior column nuclei from the arciform fibres, found that in some instances in which the lesion extended rather far forward and too deeply, causing injury to ground fibres and to some of the ocular nuclei

[1] *Loc. cit.* [2] *Loc. cit.* [3] Mott, Brain, 1895.

Fig. 9.

Fig. 10.

situated in the floor of the fourth ventricle, there was degeneration of an antero-lateral tract similar to that described by Marchi, which passed down to the lumbo-sacral region of the spinal cord, most of the fibres decussating in the anterior commissure to go to the opposite anterior horn.

More recently Biedl,[1] dividing the restiform body in cats, considers that he has completely confirmed Marchi in that he obtained degeneration of this antero-lateral tract in the spinal cord, which he believes is derived from the cerebellum. Further consideration of this antero-lateral tract, about whose origin there is so much difference of opinion, is best deferred until we are in a position to discuss the whole question of what tracts exist in this region of the spinal cord, and what are the most probable sources from which they are derived.

Passing to the consideration of other tracts, which degenerate after cutting out Deiters' nucleus, we find that running inward from the seat of lesion are well marked strands of degenerated nerve fibres which cross the medulla towards the raphe and in their course pass on the ventral aspect the nucleus of the sixth nerve, without any evidence of their entering this nerve or its nucleus at any point (see fig. 9). Some of these degenerated fibres enter the posterior longitudinal bundle of the same side, while others pass across the middle line at the raphe to enter the posterior longitudinal bundle of the opposite side. Having entered the posterior longitudinal bundles, some of these degenerated fibres pass in these structures cephalwards, while others pass caudalwards, so that both an ascending and a descending degeneration results in the posterior longitudinal bundles after a lesion such as that with which we are now dealing. In tracing these degenerated fibres caudalwards it is difficult to say that one posterior longitudinal bundle contains more of them than does its fellow (see fig. 11). When the cervical region of the spinal cord is reached they are found to occupy a position on the ventral aspect of the cord scattered

[1] Biedl, *loc. cit.*

in the anterior columns on each side of the anterior median fissure, and being least scattered in the region of the anterior columns nearest to the anterior commissure and grey matter of the anterior horns. As has already been said, further caudalwards in the cord the fibres of this system on the side of the lesion become intermingled with those of the tract which we traced from the site of the lesion through the formatio reticularis of the medulla to the antero-lateral region of the spinal cord. Judging from the degenerated fibres in the opposite anterior column, which have no other tract of degenerated fibres to become intermingled with, and whose further course through the spinal cord can consequently be followed with much greater certainty, the fibres of this system do not appear to pass far caudalwards in the thoracic cord, but become thinned out and lost. In the lumbar cord no vestige of this tract of fibres can be seen on the side opposite to the lesion, it is, therefore, probable that the anterior tract of degenerated fibres which is to be found in this region of the cord on the side of the lesion is derived solely from the tract traced through the formatio reticularis of the medulla from the seat of lesion.

The fibres which pass to the cord in the posterior longitudinal bundles end in the grey matter of the anterior horn of their own side, and, as far as can be seen, none of these fibres decussate in the anterior commissure to reach the opposite anterior horn.

The degenerated fibres which pass cephalwards in the posterior longitudinal bundles are shown in fig. 12, and can be traced to the quadrigeminal region, where they appear to terminate. All the fibres which pass across the medulla from the seat of lesion do not enter the posterior longitudinal bundles, some of them pass beyond the opposite posterior longitudinal bundle, course along the ventral aspect of the opposite sixth nucleus to end in the formatio reticularis of that side apparently; while others turn forward before they reach the raphe, and seem to terminate in the nuclei of the formatio reticularis on the side of the lesion.

Some degenerated fibres pass from the seat of lesion and arch forward in the formatio reticularis, pass dorsally to the

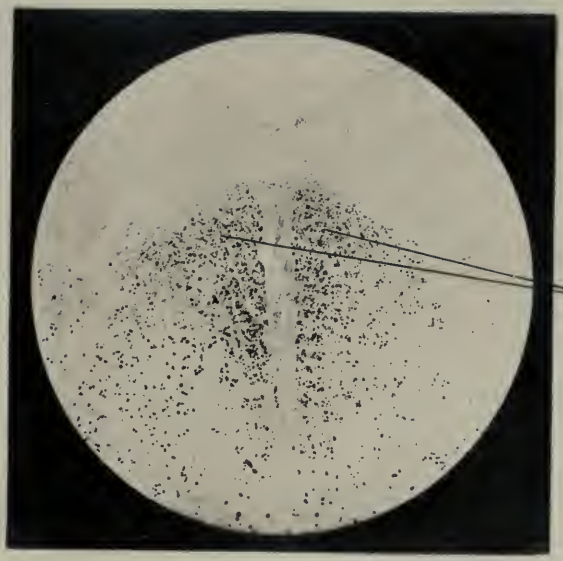

FIG. 11.

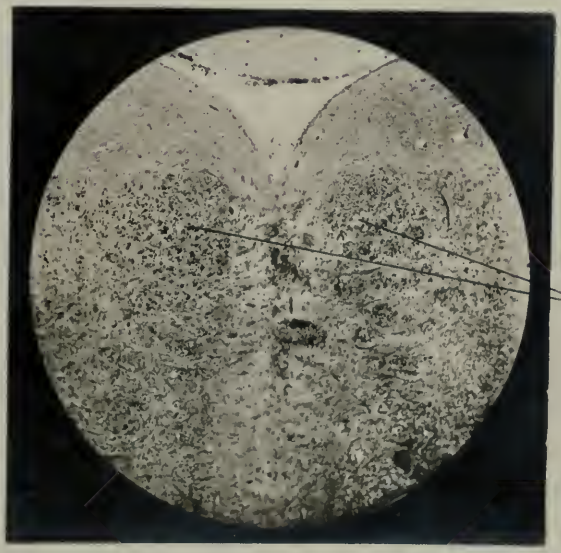

FIG. 12.

superior olive of their own side, and the pyramid on both sides to the neighbourhood of the opposite superior olive, some of them appearing to enter this structure.

Other fibres which course through the formatio reticularis in this manner and cross the raphe become intimately associated with the opposite fillet, and pass with this structure to the anterior quadrigeminal region. They occupy the position of both the mesial and lateral fillet. Though many of these fibres which cross the raphe are mingled with the trapezoid fibres, the latter are perfectly intact, and show no evidence of degeneration.

Part V.—Degenerations Resulting from Transverse Section of the Posterior Column and their Nuclei on one side in the Medulla Oblongata.

My object in undertaking this part of the investigation was to exclude the possibility of any of the results obtained after the lesions already described, more especially that in the region of Deiters' nucleus being due to accidental injury to the posterior column nuclei, for it will be remembered that after a lesion in the region of Deiters' nucleus there resulted degeneration of a tract which occupied the position of the fillet on the side opposite to the lesion, and whose fibres followed the course of the fillet through the pons and mesencephalon. To have undertaken this part of the research with any other object would have been superfluous in view of the admirable work that has been done by Mott[1] with regard to the relationship which exists between the posterior column nuclei and the mesial and lateral fillets.

With the exception of such degenerations as resulted from inclusion of part of the restiform body in the lesion, in some cases, the only degenerations which resulted were of fibres entering the cerebellum by the restiform body, and the arciform fibres which passing from the posterior column nuclei course through the formatio reticularis cross the middle line, and thus reach the opposite inter-olivary layer.

[1] Mott, Brain, 1895, p. 1.

These degenerated fibres pass forward through the pons, occupying the position of the median and lateral fillets, to the mesencephalon. The amount of degeneration of the fillet which results is only slight. This is only what was to be expected when we take into consideration how slight is the damage of the posterior column nuclei produced by a lesion passing transversely through them. So small in amount was the degeneration that it did not appear likely to be profitable to attempt to trace these fibres further forward than the anterior quadrigeminal region of the mesencephalon. The results as far as they go are in accord with those of Mott, Ferrier and Turner, and others. With regard to the additional degenerations present when some part of the restiform body was included in the lesion it is unnecessary to say anything here in that the subject of the degenerations consequent on lesions of the restiform body has already been dealt with. But, apart from this complication, degenerated fibres passed to the cerebellum in the restiform body. No efferent tract was found degenerated in the spinal cord after the lesion to the posterior columns and their nuclei, even when complicated by lesion to part of the restiform body.

(4) Discussion Relating to the Origin of the Direct Efferent Tracts met with in the Spinal Cord.

We are now in a position to discuss the possible or probable origin of the direct tracts met with in the ventral region of the spinal cord, but before doing so, it may be well to briefly summarise the various tracts to be dealt with. They are :—

(1) The antero-lateral tract, said to be derived from the cerebellum.

(2) The antero-lateral tract, derived from the region of Deiters' nucleus.

(3) The antero-lateral tract found degenerated after injury to ground fibres and some of the cranial nerve nuclei.

(4) The anterior column fibres which degenerate after hemisection of the mesencephalon.

The evidence that there exists, at any rate in man, a direct anterior pyramidal tract, is clear; but it is equally clear that this is not the only efferent tract which occupies this, the ventral region of the spinal cord. Less certain is it, however, as to how many additional efferent tracts occupy this region of the cord; nevertheless, the evidence at our disposal on this point suggests the probability that there are at least two such additional systems of fibres derived from very different sources. As to the origin of the one, there is little in dispute. Boyce[1] found that after hemisection of the mesencephalon the lesion passing between the anterior and posterior corpora-quadrigemina, fibres degenerated in both anterior columns, and that those to the opposite anterior column crossed from the seat of lesion to the opposite side of the mesencephalon in Meynert's decussation; and Held[2] states that Meynert's decussating fibres have their origin in the superior corpora-quadrigemina, and take part in the formation of the antero-lateral column. According to Boyce, these degenerated fibres reach the anterior columns of the spinal cord by way of the posterior longitudinal bundles, and cannot be traced beyond the lower part of the cervical region of the spinal cord.

The behaviour of those degenerated fibres which pass to the anterior columns of the spinal cord by way of the posterior longitudinal bundles, after injury to Deiters' nucleus is so similar to that of the fibres described by Boyce, that though taking their origin at different levels of the central nervous system, they nevertheless appear to belong to the same system of "internuncial" fibres.

The other efferent system of fibres which occupy the anterior columns of the spinal cord is a well-marked antero-lateral tract, about the source of origin of which there has been much dispute. Thus Marchi described this tract as degenerating in the spinal cord after ablation of the cerebellum; while my own observations were in accord with those of Ferrier and Turner, who failed to confirm Marchi's observations; these observers, however, found this tract

[1] *Loc. cit.* [2] *Loc. cit.*

degenerated when Deiters' nucleus was injured. Mott, on the other hand, met with this tract degenerated after injury to ground fibres, and some of the ocular nuclei ; while Biedl, dividing the restiform body 1—1½ cm. above the calamus scriptorius, found this tract degenerated, and believed that his observation confirmed that of Marchi, and concluded that the tract in question is derived from the cerebellum.[1] My own most recent experimental observations on this point are, however, entirely at variance with Biedl's results, for I find that this tract only degenerates when Deiters' nucleus is injured, or severed from its connections with the medulla oblongata. I have elsewhere[2] called attention to the possible errors which may be responsible for Marchi's results, and need not repeat myself in this connection here, except to say that it is my belief that the degeneration of this tract was probably due to accidental circumstances, and that it is probable that such a tract does not degenerate after a lesion absolutely limited to the cerebellum. So careful was I not to injure the medulla or contiguous structures when extirpating portions of the cerebellum, that I preferred to leave a thin layer of cerebellar tissue between my lesion and the medulla, in most instances, rather than run the risk of injury to the medulla. In no instance in which there was no injury to the medulla did I find this tract degenerated. The only possible source, then, from which this tract could be derived is from this fragment of cerebellar tissue left, but I feel convinced that the amount of this was too insignificant to give rise to a tract of such importance. Nevertheless, in a controversy of this kind, in which I am most anxious that the true origin of the tract in question should be decided on, I think it right to put forward all the possibilities of error that occur to me. It is a significant fact, however, that when I removed one lateral half of the cerebellum I obtained a well-marked degeneration of the restiform body, properly so called, and that the degenerated fibres from this

[1] As has already been said, the researches of Thomas also confirm Marchi's observation.

[2] *Loc. cit.*

source occupied the periphery of the medulla at lower levels, and were only seen in the formatio reticularis in their passage to the olives, and possibly to the nuclei of the formatio reticularis, while when I injured the nucleus of Deiters, or severed it from its connections with the medulla, I obtained no such degeneration in the restiform body proper, or in the peripheral region of the medulla, where such a well-marked degeneration existed when the restiform body was degenerated; but, instead, get a well-marked tract of fibres degenerating caudalwards from the region of Deiters' nucleus through the formatio reticularis to the antero-lateral region of the spinal cord.

In attempting to compare my results with those of Biedl, the first possibility that suggests itself to me is that this observer looks upon the restiform body and the direct sensory cerebellar tract of Edinger as one structure, and that what he regards as restiform body I regard as restiform body plus direct sensory cerebellar tract. Let us, therefore, consider what possibility there is that a lesion of the direct sensory cerebellar tract could be made without injury to Deiters' nucleus, which, as we are all aware, is intimately imbedded in this tract. Certainly this object will not be obtained by a lesion dividing the restiform body and ascending root of the fifth nerve 1—1½ cm. above the calamus scriptorius. A far more likely result of such a lesion would be to interrupt any fibres that might be passing from Deiters' nucleus caudalwards, or any derived from the direct sensory cerebellar tract which do not end in Deiters' nucleus (if there are any such fibres), but pass it to reach some region of the medulla caudal to it. The only lesion of the direct sensory cerebellar tract followed by degeneration in the medulla and spinal cord caudal to Deiters' nucleus that can be regarded as indicating that the fibres so degenerated are derived from the cerebellum is the section of the tract between Deiters' nucleus and the cerebellum high enough up, that is, near enough to the cerebellum to avoid injury to Deiters' nucleus. So difficult is this that I have never accomplished the task without some injury to the nucleus in question, except in one instance, despite the fact that I invariably

attempted to divide the tract as close to the cerebellum as possible without at the same time producing any damage to the cerebellum itself as opposed to this strand of fibres connected with the organ. This much, however, is certain, and that is that the more obvious the lesion to Deiters' nucleus, the more marked was the tract degenerating in the formatio reticularis and passing down the antero-lateral region of the spinal cord, and the less evidence there was of any injury to the nucleus, the less degeneration could be found passing through the formatio reticularis to the spinal cord; indeed, in the one instance in which the nucleus appeared to have escaped injury no sign of degeneration of the antero-lateral or any other tract was found in the spinal cord, so that the evidence seems strong, that the antero-lateral tract originates in Deiters' nucleus, and is not derived directly from the cerebellum.

The observation of Von Monakow,[1] that the cells of Deiters' nucleus atrophy after hemisection of the spinal cord in the cervical region of a new-born rabbit is worthy of careful attention in this connection. Of no less importance are the observations of Held based on the myelination method, for he finds that the antero-lateral ground bundles and lateral limiting layer of the spinal cord are connected with Deiters' nucleus by means of the longitudinal fibre systems of the reticular formation.

But to return to the results obtained by Biedl, we must remember that his lesion was situated at a point 1—1½ cm. above the calamus scriptorius, that it penetrated sufficiently deeply into the medulla to divide the ascending root of the fifth nerve as well as the restiform body, and that as a result of this lesion not only was degeneration of an antero-lateral tract in the spinal cord met with, but also a direct tract in the lateral region of the spinal cord closely associated with the crossed pyramidal tract, as far as position is concerned. Now, I have already shown in an earlier part of this paper that this is precisely the condition of things met with after a lesion of the formatio reticularis, external to the inferior

[1] V. Monakow, *Archiv. f. Psychiat u. Nervenkrank.*, xiv., 1883, p. 1.

olive, and in the region of the nucleus lateralis. Further, I have called attention to the fact that the lateral tract corresponds closely with a similar tract described by Boyce as degenerating after hemisection of the mesencephalon in the region between the anterior and posterior corpora quadrigemina.

With such evidence before us it is impossible to escape from the supposition that Biedl's lateral tract is in reality none other than that met with by Boyce and myself after the lesions just described, and that the degeneration resulted from its interruption in the formatio-reticularis. A reference to Boyce's, fig. 4, p. 329, will show that his so-called lateral columnar tract is so closely related in position to the ascending root of the fifth that any lesion to the latter structure would be more than likely to also involve these lateral columnar fibres, and would account for the degeneration met with by Biedl in the lateral column of the cord.

The antero-lateral tract of Biedl in all probability results from interruption of fibres passing from Deiters' nucleus in the formatio reticularis, or possibly from injury to this nucleus itself. That this is the probable origin of the degenerated fibres met with in this tract, and that it does not simply indicate an interruption of Boyce's anterior columnar-fibres is shown by the fact that the fibres traced by Boyce to the anterior columns of the cord pass in the posterior longitudinal bundles well out of reach of Biedl's lesion, and that the latter observer describes degenerated fibres in the formatio reticularis in precisely the position in which they have been met with by me after lesions of Deiters' nucleus.

It must not be lost sight of in this discussion that however much difference of opinion there may be with regard to the origin of the antero-lateral efferent tract, it is the only one met with by me after complete section of the restiform body proper together with the direct sensory cerebellar tract, and that absolutely no evidence of any direct lateral tract situated near the crossed pyramidal tract was met with. Mott's result is of importance in this connection, for his lesion involved the sixth nucleus chiefly,

and would thus almost certainly interrupt the fibres passing from the region of Deiters' nucleus to the antero-lateral region of the cord; and in accordance with my results he met with an antero-lateral tract only, and with no lateral tract in the region of the crossed pyramidal tract.

(5) General Conclusions.

The results obtained in connection with the various experimental lesions that have been described in this paper, justify the following conclusions:—

(1) The descending antero-lateral tract which degenerates in the spinal cord after a lesion of the lateral region of the medulla is probably the same as that met with after injury to Deiters' nucleus, such slight differences as exist between the two tracts being possibly accounted for by the fact that some of the fibres which degenerated after the lesion to Deiters' nucleus may have escaped injury in connection with the lesion to the lateral region of the medulla. This tract is probably identical with that described by Marchi, as degenerating after lesions of the cerebellum; Mott, after injury to ground fibres and some of the cranial nuclei; and Biedl, after section of the restiform body and ascending root of the fifth nerve; its real source of origin being Deiters' nucleus as contended by Ferrier and Turner.

(2) The degenerated fibres which reach the anterior columns of the upper portion of the spinal cord through the posterior longitudinal bundles after a lesion of Deiters' nucleus, are quite distinct from the above tract, and probably belong to some system of inter-nuncial fibres similar to those traced by Boyce to the anterior columns of the spinal cord, by way of the posterior longitudinal bundles, after hemisection of the mesencephalon.

(3) The direct descending tract of degenerated fibres met with in the spinal cord, in close relationship to the fibres of the crossed pyramidal tract, after a lesion of the lateral

region of the medulla, is probably identical with a similar tract described by Boyce, after hemisection of the mesencephalon, and by Biedl after section of the restiform body and ascending root of the fifth nerve, the proximity of Boyce's columnar tract to the latter structure accounting for its probable inclusion in Biedl's lesion. The fact that Boyce only traced these columnar fibres to the cervical region of the cord, while I was able to trace them throughout the whole length of the cord, probably indicating that the system of fibres is one to which new fibres are constantly being added as levels more and more caudally situated are reached in passing through the pons and medulla. On this hypothesis, there is nothing peculiar in Boyce's fibres derived from the mesencephalon terminating in the cervical cord, while my interruption of the tract in the lower region of the medulla resulted in degeneration of fibres to the lumbo-sacral region of the cord. The most important objection to looking on Boyce's tract, and that which I have described as belonging to the same system of fibres, is the fact that Boyce's fibres originate in a decussating system, while there is no evidence that the tract I have met with decussates at any point in its course.

(4) That the direct tract in the region of the crossed pyramidal tract is in no way derived from the pyramids is abundantly proved by the fact that in my lesions both pyramids were left absolutely intact, not the slighest injury to either of the structures being detected on most careful microscopical examination.

(5) Fibres derived from the restiform body proper and degenerating caudalwards, after section of this structure, occupy the lateral peripheral region of the medulla in passing further and further caudally, do not form a descending tract in the spinal cord, but pass to the formatio reticularis, and to both inferior olives.

(6) The " direct sensory cerebellar tract " of Edinger is a totally distinct structure from the restiform body, and ought not to be confounded with it. This tract is not an afferent tract, but on the contrary all the evidence obtained on this question points to its being an efferent tract from the nucleus

globosus of the cerebellum to Deiters' nucleus in the medulla, as has been contended by Ferrier and Turner.

(7) Fibres which degenerate after section of this tract (with injury to Deiters' nucleus) have a different course in the medulla oblongata to those derived from the restiform body.

(8) There is an indirect efferent path from the cerebellum to the spinal cord, through the "direct sensory cerebellar tract" of Edinger and Deiters' nucleus.

(9) One of the tracts met with after a lesion of the lateral region of the medulla corresponds so closely in position and distribution with the afferent antero-lateral tract of Gowers, that it is highly probable that they are identical, and accordingly that the observations of Mott and of Auerbach are correct in this connection.

(10) Another afferent system of fibres which degenerates after the lesion of the lateral region of the medulla, and which is related to the fillet in its course to the quadrigeminal region, is probably the same as that described by Mott as a distinct part of the antero-lateral tract of Gowers, and is certainly no artifact as has been suggested by Patrick.

(11) After injury to Deiters' nucleus, a tract of degenerated fibres can be traced in close association with the opposite fillet to the anterior quadrigeminal region.

(12) So, too, degenerated fibres forming an afferent system pass in both posterior longitudinal bundles to the quadrigeminal region of the mesencephalon after injury to Deiters' nucleus.

(13) None of the degenerations found after section of Edinger's direct sensory cerebellar tract can be ascribed to the severance of fibres of this tract, but all appear to depend on concomitant injury of Deiters nucleus.

(14) Injury of the posterior column nuclei is responsible for none of the degenerations met with in this inquiry, with the exception of a well-marked degeneration of the arciform fibres to the opposite inter-olivary layer with consequent well-marked degeneration of the fillet and a degeneration of fibres passing in the restiform body to the cerebellum.

DESCRIPTION OF FIGURES.[1]

FIG. 1.—Lesion of the lateral region of the medulla oblongata (L) produced by means of a needle. Degenerated fibres, stained black, are seen in the neighbourhood of the lesion at the periphery of the medulla, and also in the fillet (F) immediately dorsal to the opposite pyramid. The inferior olive (O) and both pyramids (P) are absolutely intact.

FIG. 2.—The efferent tracts which degenerate after a lesion such as that represented in figure 1, and which form a continuous band in the cervical region of the spinal cord, from which region the photomicrograph was taken. Two chief areas of degeneration, the one situated ventrally (A-L) and the other close to the crossed pyramidal tract (L.C.), are united by a narrow band of degenerated fibres which occupy the periphery of the antero-lateral region of the cord.

FIG. 3.—A photomicrograph of a section from the thoracic part of the same cord as that whose cervical region is shown in figure 2. The degenerated fibres are more scattered, and the ventral (A-L) and lateral (L.C.) tracts are no longer so continuous with each other as was the case in the cervical region.

FIG. 4.—The two efferent tracts are seen completely separated from each other in the lumbar region of the same cord from which figures 2 and 3 were taken.

FIG. 5.—The afferent tracts which degenerate after a lesion such as that shown in figure 1. Two dark areas of degenerated fibres are seen at the periphery of the medulla, the one irregularly circular (A-L) situated just outside and dorsal to the inferior olive, and the other forming a band which occupies the position of fibres which pass to or from the restiform body (R). F = Fillet.

FIG. 6.—A photomicrograph of a section showing the restiform body (R) with its fibres divided transversely, some of them being degenerated in consequence of a lesion of this structure caudal to the point shown in the figure, and the direct sensory cerebellar tract of Edinger (E) with its fibres undegenerated and cut longitudinally.

FIG. 7.—The line of incision (L) passing through Edinger's direct sensory cerebellar tract is shown in this figure. The dorsal part of the restiform body (R) external to Edinger's tract has been slightly injured, but all other adjacent parts of the medulla and cerebellum have escaped injury.

FIG. 8.—The lesion (L) in a case in which the nucleus of Deiters was severed from its connections with the medulla oblongata.

FIG. 9.—A photomicrograph of the tracts of degenerated fibres which result from a lesion such as that represented in figure 8. One darkly stained band of degenerated fibres is seen occupying a considerable area of the formatio reticularis midway between the ascending root of the fifth nerve and the raphe (A-L). Other degenerated fibres are seen passing on the ventral aspect of the sixth nucleus (VI.) to reach the posterior longitudinal bundles (P.L.B.)

[1] The figures are from photomicrographs, some of which were taken for me by Dr. E. S. Worrall, and others by Mr. L. B. Fleming.

FIG. 10.—A photomicrograph to show the distribution of the degenerated fibres in the upper cervical region of the spinal cord after the lesion shown in figure 8; they occupy both anterior columns (A), and the periphery of the antero-lateral region of the cord on one side (A-L) as well.

FIG. 11.—The efferent fibres which degenerate in the posterior longitudinal bundles (P.L.B.) after the lesion shown in figure 8.

FIG. 12.—The afferent fibres which degenerate in the posterior longitudinal bundles (P.L.B.) after the lesion shown in figure 8.

AN EXPERIMENTAL INVESTIGATION OF THE DIRECT PYRAMIDAL TRACT.[1]

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Introduction.

Before entering upon the subject of my paper, I wish to express my thanks to Professor Vaughan Harley for allowing me to carry out this investigation under favourable circumstances at the Laboratory of Pathological Chemistry at University College, and to Mr. Victor Horsley for his assistance, especially during the earlier part of the research, and for the kind and valuable criticism which he was always willing to afford.

The fibres of the crossed pyramidal tract are well known to conduct impulses from the cerebral cortex of one side to muscles upon the opposite side of the body. It has been the object of the present research to determine to what extent the same statement is true for those motor fibres which, at the decussation of the pyramids in the medulla oblongata, do not cross to the opposite side of the cord, but continue on the same side under the name of the "direct pyramidal tract."

[1] The expenses of this research were defrayed by the British Medical Association.

Method of Investigation.

Animals employed.—Inasmuch as the direct pyramidal tracts form a larger part of the cord in monkeys than in lower animals (and the difference is yet more marked in the case of the *anthropoid* apes), I had hoped and attempted at the outset to carry out this investigation in monkeys. But the necessary severity of the operations and the consequent shock were so great that these animals could not live to the end of the experiment. The same remark applies to cats and to rabbits. The results obtained, therefore, in the present research, are to be taken as applying to dogs only.

In a preliminary note in the *British Medical Journal* of July 18, 1896, allusion was made to an experiment on a monkey which apparently gave positive results. Microscopical examination of the medulla oblongata in that case, however, showed the experimental division to be incomplete.

The following was the method of research :—

(*a*) The dogs were anæsthetised with ether in all cases except the first, when chloroform was used. In no case was the animal allowed to recover from the anæsthetic.

(*b*) A preliminary tracheotomy was performed in every case.

(*c*) The skull was opened over both excitable regions of the cerebral hemispheres without opening the dura mater. All hæmorrhage being arrested, the flap of skin was replaced in order to keep the cortex warm during the progress of the ensuing part of the experiment.

(*d*) The arches of the atlas and axis were exposed by a median incision and removed. By means of a small trephine, an aperture was made in the thin plate of the occipital bone covering the vermis, and this aperture was enlarged and made continuous with the foramen magnum by removing the bridge of bone, which separated the two.

(*e*) The dura mater thus brought to view was opened in its whole length, and the cerebellum, lower part of the medulla oblongata, and uppermost end of the spinal cord well exposed. The fourth ventricle was then carefully opened. The posterior median fissure could now be seen

extending down the cord from the point of the calamus scriptorius, with the columns of Goll standing out prominently on each side of it. The cerebellum was raised slightly by means of an aneurism needle and the lower part of the fourth ventricle exposed to view. A von Graefe's cataract knife was then inserted in the middle line just above the point of the calamus scriptorius, and thrust forward and upward towards the anterior surface of the medulla. The incision thus begun was carried in the middle line down the whole length of the medulla, and part of the spinal cord exposed, care being taken that the point of the knife should not quite reach the anterior or ventral surface of these structures. This part of the operation was then completed by withdrawing the knife and inserting into the incision a blunt flat seeker and pressing it against the posterior surface of the basilar portion of the occipital bone, and the posterior surfaces of the bodies of the vertebræ. This proceeding was found to be necessary because, in those cases where one was fortunate enough to strike the middle line in front, the basilar artery became longitudinally divided if the knife alone was used throughout.

(*f*) At this stage the animal ceased breathing, but was restored by performing artificial respiration for about ten minutes.

(*g*) Attention was now again turned to the cortex cerebri. This was exposed by again reflecting the flap of skin made at the earlier part of the experiment, and opening the dura mater over each hemisphere. Flaps of dura mater were formed and turned upward, so as to expose on each side the crucial sulcus and neighbouring gyri.

(*h*) With a weak faradic current, the excitability of the cortex was ascertained by stimulation of the facial area. Satisfied on this point, one next proceeded to systematically stimulate the motor area in all its parts, and to note what movements were obtained as a result of such stimulation.

(*i*) Finally, the animal was killed, and its brain removed with the upper part of the spinal cord for subsequent examination. The relative accuracy of the division of the decussation was ascertained by macroscopical examination

of the medulla at the time and by subsequent microscopical examination of transverse sections of the same.

Experiments.

In the following list of experiments I make no reference to those in which, owing to death of the animal from hæmorrhage, shock, &c., inexcitability of the cortex, or very incomplete division of the decussation, the result was valueless. Unfortunately, owing to the many difficulties of the investigation, much of my work was lost by accidents of this nature.

No. 1.—Mesial Section.

In this case, the incision passed longitudinally down the left pyramid and divided all the fibres going from the right hemisphere to the left half of the cord; while the division of those fibres which passed from the left hemisphere to the right side of cord, was incomplete.

Movements obtained.—Flexion of the opposite hip was obtained from stimulation of either cortex.

Flexion of the right fore-limb at the shoulder was obtained from stimulation of the left hemisphere; but no movements could be obtained in the left fore-limb by stimulation of either cortex.

No movements of the trunk could be obtained, nor were there any movements on the same side of the body as the excitation.

No. 2.—Mesial Section.

The incision was absolutely in the middle line completely dividing the decussation.

Movements obtained.—Flexion of the opposite hip was obtained from excitation of either cortex. It was equal on the two sides.

There were no movements of the fore-limbs, of the trunk, or of the same side of the body as the excitation.

No. 3.—Mesial Section.

The incision passed down the outer side of the left pyramid. It divided all the fibres going from the right

hemisphere to the left side of the cord, but apparently few of those going from the left hemisphere to the right side of the cord.

Movements obtained.—Flexion of the opposite hip was obtained on excitation of either cortex.

Flexion of the right fore-limb at the shoulder was obtained on stimulation of the left cortex.

No movements of the left fore-limb were obtained, or of the trunk, or of the same side of the body as the stimulation.

No. 4.—Mesial Section.

The incision passed down the outer side of the left pyramid, and was such that very few motor fibres were divided.

Movements obtained.—Powerful movements were obtained in all four limbs. I include this faulty experiment, because it not only serves as a control observation, but also because the tail deviated to the same side as the cortical excitation at a spot just above the leg area on each side (*vide Experiment No.* 6).

No. 5.—Mesial Section.

The incision was median and divided the decussation completely.

Movements obtained.—Flexion of the hip, ankle, knee and toes of each side resulted from stimulation of the opposite cortex.

No movements were obtained of the fore-limbs, of the trunk (including the tail), or of the same side of the body as the cortical excitation.

No. 6.—Mesial Section.

The incision passed very slightly to the right of the middle line and divided the decussation completely, except for a few fibres which passed from the left cerebral hemisphere to the right side of the cord at the lower end of the incision.

Movements obtained.—On stimulation of the left cortex a slight movement of the right fore-limb digits was obtained. This was, however, soon lost.

Flexion of the hip, knee, ankle and toes was obtained on stimulation of the opposite cortex on each side.

The tail deviated to the same side as the excitation.

No trunk movements were obtained.

No. 7.—Mesial Section.

The incision was median and completely divided the decussation.

Movements obtained.—The only movements obtained in this animal were slight flexion of the toes on each side on excitation of the opposite cortex; a deviation of the tail to the same side as the excitation. The deviation of the tail was more marked to the left than to the right side.

No movements were obtained of the fore-limbs, or of the trunk, or on the same side as the excitation, with the exception of the tail.

No. 8.—Mesial Section.

The incision was median and completely divided the decussation. At the lower end, however, it deviated to the right and apparently divided nearly all, if not all, of the fibres of the direct pyramidal tract on that side.

Movements obtained.—Slight flexion of the toes was obtained on the right side, but no movements of the left, on excitation of the opposite cerebral cortex.

The tail deviated to the left on stimulation of the left cerebral cortex. No deviation to the right could be obtained.

No movements were obtained of the fore-limbs, or of the trunk on either side.

No. 9.—Mesial Section.

The incision was median for the greater part of its length, but deviated slightly towards the left at the upper end of the decussation, and a few fibres at the upper end of the decussation passing from the left cerebral hemisphere to the right half of the cord escaped division.

Movements obtained.—Excitation of the left cortex gave well-marked flexion of the right hip and knee, slight flexion of the left hip, arching of the trunk laterally with the concavity to the left side and deviation of the tail to the right.

Excitation of the right cortex gave slight flexion of the left hip, knee, ankle and toes, and deviation of the tail to the left.

No movements of the fore-limbs were obtained.

No. 10.—*Mesial Section.*

The incision was oblique so that it deviated slightly to the left at the upper end, while at the lower end it encroached slightly on the right direct pyramidal tract. A few fibres passing at the upper end of the incision from the left hemisphere to the right side of the cord escaped division.

Movements obtained.—Excitation of the left cortex gave slight flexion of the hip, knee and ankle on the right side, and contraction of the right back muscles so that the trunk was arched laterally with its concavity to the right.

Excitation of the right cortex gave extension of the hip, knee and ankle on the right side. No movements were obtained on the left side.

There were no movements of the fore-limbs.

No. 11.—*Mesial Section.*

The incision was median and the decussation was completely divided.

Movements obtained.—Flexion of the hip, knee, ankle and toes was produced on each side by excitation of the opposite cerebral cortex, while the tail deviated to the same side as the excitation.

There were no movements either of the trunk or of the fore-limbs.

No. 12.—*Mesial Section.*

The incision was median and completely divided the decussation.

Movements obtained.—The results were precisely the same as those in the last experiment, except that no movements of the tail were obtained.

Remarks.

There is considerable uniformity in the results obtained in the above experiments, although No. 9, and in part No. 10, appear to be anomalous in many respects.

In cases 2, 5, 7, 11, and 12 the incision precisely carried out the desired conditions. No. 8 went farther, inasmuch as the only tract left intact was the direct pyramidal tract of one side. In none of these cases was any movement obtained either in the fore-limbs or in the trunk. On the other hand, flexion was obtained, on the opposite side to that of the excitation, at the joints of the hind-limbs in all the cases—hip, knee, ankle, and toes in three, toes only in two, hip only in one.

In three of these cases there was deviation of the tail to the same side as the excitation. Experiments 4 and 6 are also in accordance with this observation; while in the anomalous No. 9 the tail deviated to the opposite side.

In all the other experiments, except No. 4, the crossed pyramidal tract was completely cut off from one side of the body only; and in all these cases the results for that side are in accordance with the above observations.

The movements obtained in the limbs were in no case as powerful as those in a dog where the pyramids are untouched. That this is not merely due to shock is shown by facts observed in No. 4 and in several of my unpublished cases. In some of these the medulla was longitudinally divided; but the pyramids were absolutely untouched. In all such cases the movements of the limbs, obtained on excitation of the cortex, were quite powerful.

No importance is to be attached to the fact that flexion rather than extension of the limbs was so uniformly obtained; because the former movement is so much more readily brought about by excitation of the cortex than the latter.

Conclusions.

(1) The number of fibres (channels) in the direct pyramidal tract does not appear to be the same for all individuals of the same species.

(2) Nearly all the fibres of the so-called direct pyramidal tract ultimately cross to the opposite side of the cord.

(3) Its fibres convey some of the impulses from the cortex cerebri to the hind-limb of the opposite side.

(4) A few fibres remain on the same side of the cord to supply the tail muscles of that side.

(5) The direct pyramidal tract conveys no impulses to the fore-limbs or to the trunk.

A METHOD OF EXAMINING FRESH NERVE-CELLS; WITH NOTES CONCERNING THEIR STRUCTURE, AND THE ALTERATIONS CAUSED IN THEM BY DISEASE.

BY JOHN TURNER, M.B.

Assistant Medical Officer, Essex Lunatic Asylum.

Ever since Nissl introduced his method of staining the chromophilic material of the nerve-cell by methylene blue, considerable doubts have been entertained, at least by English histologists, as to whether the appearances observed were not artificially produced during the somewhat severe processes through which the section had to pass in preparing and staining. Dr. Robert S. Cook,[1] by a modification of the method in which neither alcohol or heat was employed, did much to remove the doubts entertained as to the genuineness of the chromophilic material considered as a constituent of the normal cell structure; still, his method involves the freezing of the cell and its fixation in osmic acid, &c. Recently, Dr. Mott has expressed the opinion that the rod-like and granular arrangements seen in the cell in Nissl's preparations might be due to coagulation by the alcohol, &c., of the albuminous principles of the cytoplasm. That, however, the appearances here referred to are quite genuine, and exist as such in the dead cell, unaltered by reagents, can with certainty be shown by examining fresh cells in the following way.

A small and thin slice of cortex is placed direct in a 0·5 per cent. watery solution of methylene blue (Grubler, B.x.), and is left from three to twelve hours; three hours is sufficient, but the pieces do no harm after twelve hours' soaking.

[1] Eighth Annual Report, New York State Hospital, 1894.

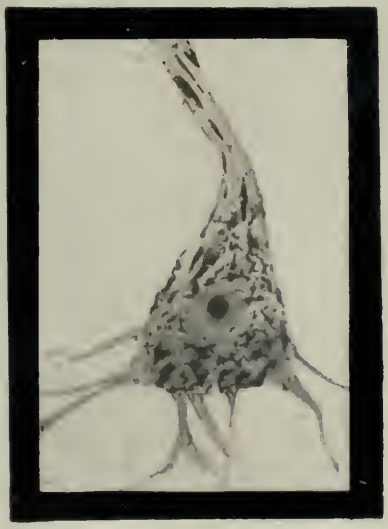

FIG. 1.

FIG. 2.

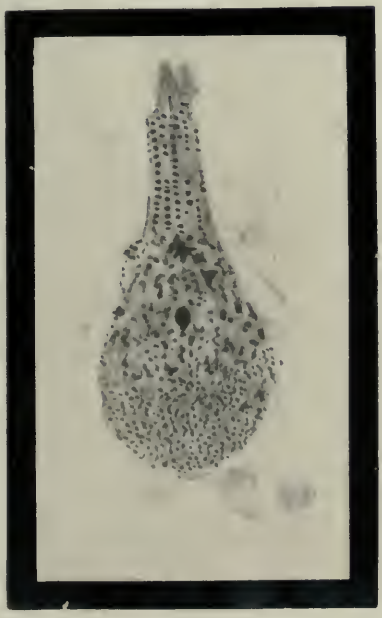

FIG. 3.

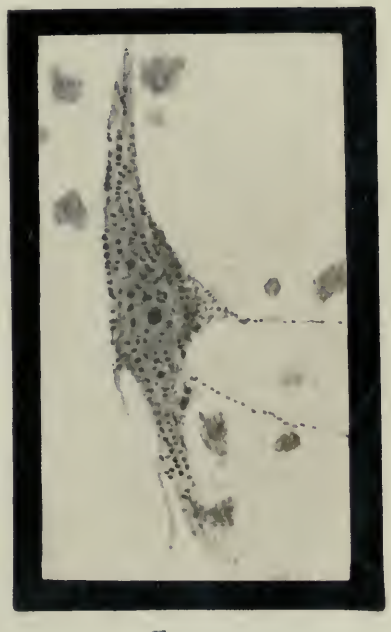

FIG. 4.

When ready a minute portion is removed with a scalpel from the surface of the piece, through the whole depth of the cortex, the smaller the piece the better. The fragment is laid on a slide and covered with a drop of Farrant's solution (the Farrant is not essential—water suffices—but with the former the preparations keep longer), and covered with a cover-glass, and gentle, uniform pressure, made with two mounted needles, to spread out the fragment, so that its several parts maintain, as far as possible, their relative position to one another.

When the film is sufficiently thin to transmit light, the slide is removed to the stage of the microscope, and further flattening out done with a mounted needle, watching the cells meanwhile through a half-inch objective. Remarkably beautiful preparations of nerve-cells are thus obtained, which are best viewed by artificial light. As a rule these preparations do not keep more than ten days, but they can be preserved for a few days longer, and rendered sharper, if the film is passed to and fro through a flame till the slide is just too hot to bear on the back of the hand. By this means the colour in the cells deepens, and that in the matrix fades quicker than it does if left unheated. Occasionally, for what reason I know not, the films will keep for a long time. (Fig. 2 was photographed from one which had been prepared two years previously.)

These preparations are at their best a few days after first made, as by that time nearly the whole of the blue colour (pink by artificial light) of the matrix has faded, and the blue cell stands out sharply against an almost colourless background.

By the above means one is enabled to study nerve-cells in their original condition, and unaltered by any re-agents (beyond the artificial colouring). A true estimate of their size is formed, and the intimate structure of the cyto-plasm is clearly seen. Pigmentation, when present, is distinctly shown; it is generally of a light yellowish green colour, or of a light brownish red. Sometimes the cells (especially the giant pyramids of the motor region) are enormously distended with it, so that they bulge out to more

than their original size, the collection of pigment being larger than the rest of the cell. The pigment is most usually deposited at the base or along one side of the cell, but it may intervene between the body and apex, apparently blocking off completely the former from the latter.

The nucleolus is darkly stained and sharply defined; the nucleus does not show except in certain pathological conditions. One observes the great elasticity of the cells; with pressure on the cover-glass, their long flexible processes bend about in all directions. The body spreads out and withstands considerable squeezing, returning to its former shape when the force is removed. In senile cells, and those altered by disease, this elasticity is often lost, and very slight pressure will part the processes from the body and cause the latter to break up.

The shape of the cells is seen to be very different in different cases, those from young subjects, &c., are angular with 7, 10 or more processes, while those from the old or degenerated are globular and with few or perhaps none. I am speaking now of the pyramidal cells of the third and fourth layers of frontal and motor cortex, and the large scattered pyramids of the occipital cortex.

Lugaro[1] refers "to the condition which has been termed sclerotic degeneration, in which the cell appears shrunken, intensely stained, and homogeneous," and this he considers "artificially produced by the action of hardening agents under circumstances not well understood."[2] However, that this appearance and condition does occur in certain pathological states, and is not due to artificial means is evident, as such cells are met with by the present method; they generally appear small and badly shaped, and are tough, very considerable pressure on the cover-slip being required to break them down.

The chromophilic material exists normally in the body of the cell in the form of short, thick rods, triangular and spindle-shaped bodies, and in the more interior parts of

[1] *Rivista di Pathologia Nervoso E Mentale*, Aug. 1896.

[2] Quoted from Dr. F. Robertson's Abstract, *Journ. Mental Science*, July, 1897.

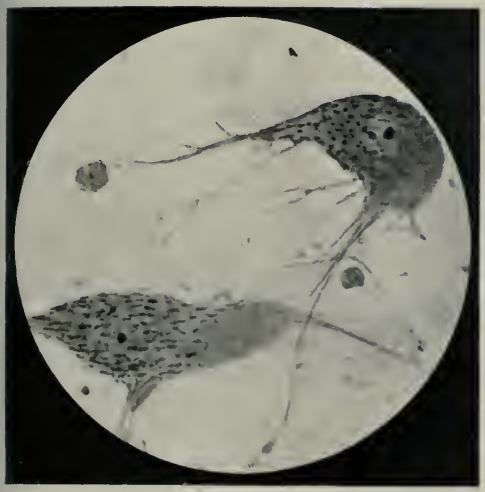

FIG. 5.

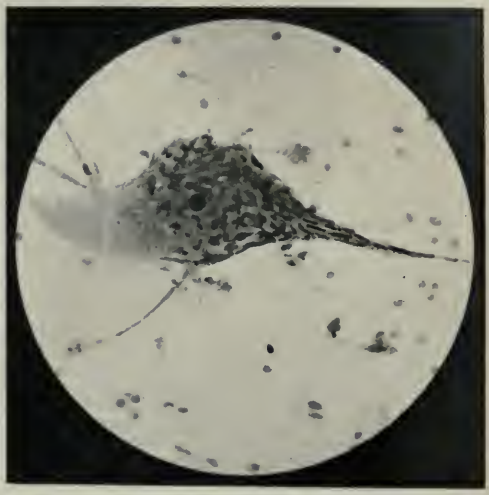

FIG. 6.

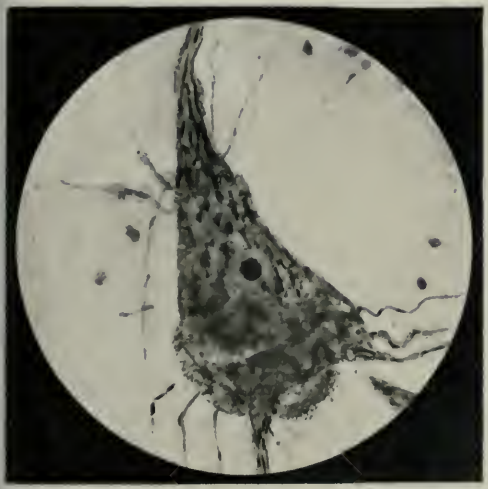

FIG. 7.

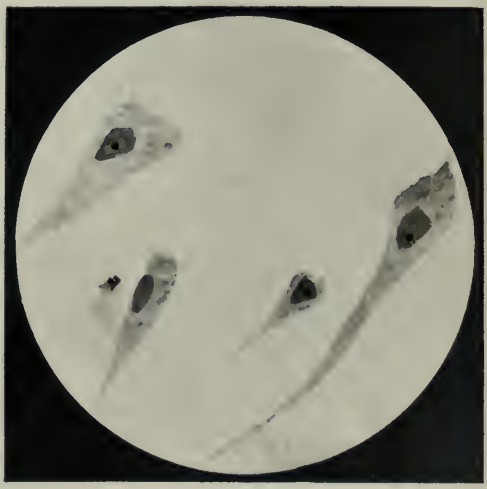

FIG. 8.

the cytoplasm as irregular ill-defined masses; in the apex and processes as somewhat slender, often spindle-shaped threads with their long diameter, corresponding to that of the process. These threads are from 10-20μ long. Such an arrangement is found among the cells (presumably in this respect normal) in the brains of those dying with recent insanity; and to a less extent in those whose mental symptoms have been of longer duration, but in which gross degenerative changes have not taken place (see figs. 1, 5, 7, 9).

An early indication of morbid change is the breaking up of the threads in the processes into a series of small granules or cubes, which, however, are still linearly disposed (see figs. 3, 4 and 10).

Aggregation of several particles into large masses is also apparently an early change (see fig. 11), at least it is found in cells which in other respects appear healthy.

In senile and certain other prolonged degenerative conditions (*e.g.*, advanced general paralysis) it is common to find the chromophilic material arranged in the form of rather large irregular granules distributed sparsely throughout the cell and in its processes, in which latter are no longer any threads to be seen (fig. 12).

In advanced general paralysis one sometimes (probably usually) meets with a complete disappearance of the chromophilic material; film after film from different parts of the cerebrum may be examined, all with a negative result. In these cases the cells often stain rather deeply and diffusely, and are tough and difficult to break. They are, except that often there is no apparent shrinking, in the condition that Lugaro refers to as sclerotic.

Sometimes with general disappearance of the chromophilic material the cytoplasm stains lightly and homogeneously, and is exceedingly fragile, so that it is almost impossible to spread out the film without fracturing processes, and often destroying the contour of the cell. In such cases one generally finds the nucleus well defined, rather deeply stained, round, oval, or triangular, with rounded edges; but although it may be found to have a shrunken appearance in fresh Purkinje cells, it is rarely if

ever so seen in the cells of the cerebrum. The appearances just described are sometimes found in advanced general paralysis (fig. 8).

In the Purkinje cells of the cerebellum the chromophilic material is scattered uniformly throughout the cytoplasm, in the form of short rods, triangles, or granules, but in certain morbid conditions it is common to find an aggregation of it around the nucleus, while the remainder of the cell contains little or none. This appearance has been described by Dr. Fleming in the multipolar cells of the anterior cornua in rabbits and dogs after ligature of the sciatic nerve, etc.[1]

The processes stain either very faintly or not at all except with certain pathological conditions; thus, those of senile cells, stain often densely.

The cerebellum on account of the more or less uniform size of its elements is a favourable site from whence to obtain specimens for a comparison of fresh and hardened cells.[2]

The shrinking of the Purkinje's cells and their processes in alcohol varies a great deal; in the case of those in which presumably no marked degenerative processes have taken place it amounts to about one-third of the original bulk,

[1] BRAIN, vol. xx., Nos. 77-78.

[2] The following modified method of Nissl I have found gives good results, and as it is far simpler than the original process, it may be worth while to describe it: Small, thin (4—5mm. thick) slices of fresh brain are placed in absolute alcohol, where they remain 24-36 hours, care being taken that each lies perfectly flat and not in contact one with another. The alcohol should be changed twice or three times as soon as it becomes turbid. The pieces are then trimmed so as to be no thicker than 3 mm., and placed in a 2% watery solution of methylene blue B.x. They remain in the solution all night, standing on the paraffin bath, which is kept at 56°—58° C. The following morning the pieces are removed from the staining fluid and put into absolute alcohol, which is changed once or twice during the three hours in which they remain in alcohol; they are next wiped and transferred to chloroform, where they remain three hours, and are then put into paraffin of a melting point of 52° C., where they remain 3—4 hours. It is essential that they be passed through two lots of paraffin, otherwise they will be too soft to cut satisfactorily. The sections are examined either immediately after removing the paraffin from them, or, better still, after they have been fixed to the slide by capillary attraction. In the former case they shrink on removing the paraffin, but in the latter they expand. If fixed they must be flattened, and the surplus water removed from the slide as quickly as possible, as it abstracts colour from the section, and they must be thoroughly dried before the paraffin is washed out. If these instructions are carried out they keep well. Sections prepared in the above manner may subsequently be stained (on the slide) by Beneke's method for showing neuroglia structure with good results.

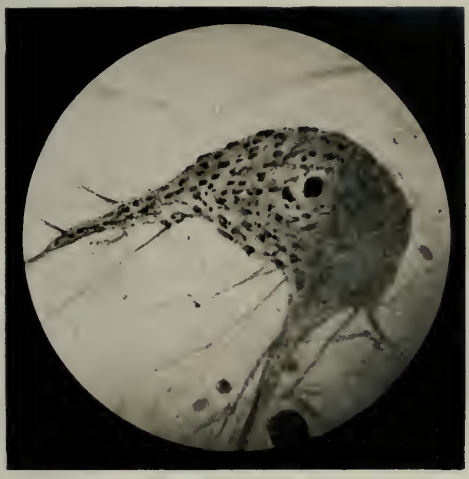

FIG. 9.

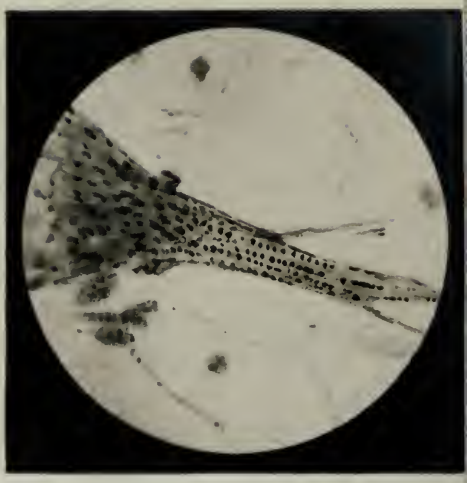

FIG. 10.

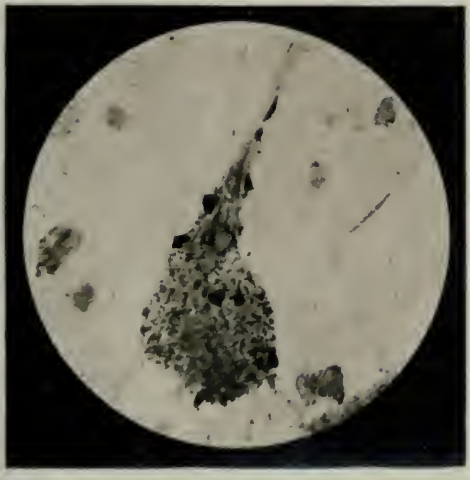

FIG. 11.

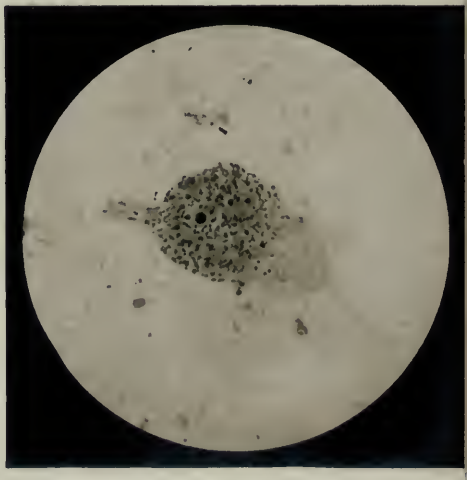

FIG. 12.

in senile and otherwise degenerated cells to nearly a half. This is probably due to the dehydration of the section, the supposition being that in the latter the cytoplasm is largely occupied by some watery matter which the alcohol removes. A variation is indeed noted in the size of fresh cells, between those of the young and recent cases and the old and degenerated, as the following table shows:—

No.	Sex.	Age.	Diameter in μ of Purkinje's cells (mean of 10) Long.	Trans.	Form of Mental Disorder.	Duration.
1	F.	64	37	30	Senile mania	Many years
2	F.	69	41	31	Senile mania	Doubtful
3	F.	33	42	32	Organic dementia	Over a year
4	M.	40	42	35	Dementia general paralysis	Some years
5	M.	45	47	33	General paralysis	Within a year
6	M.	39	54	30	Advanced general paralysis	Some years
7	M.	63	53	31	Chronic mania	Many years
8	F.	47	47	36	Chronic mania	Many years
9	M.	45	50	40	Chronic mania	Many years
10	F.	56	50	40	Chronic melancholia ...	Two years
11	F.	20	54	38	Acute mania	Recent
12	F.	30	52	43	Advanced general paralysis	Over a year
13	F.	33	56	45	Acute mania	A few weeks

But, in all cases, the latter in contradistinction to hardened cells retain their plump form and never appear shrunken. The processes also which in degenerated cells after hardening appear attenuated and darkly stained, do not in the fresh state, beyond the taking up of more colour, show any marked departure from a normal condition as regards calibre.

This shrinking of cells with alcohol, &c., in certain abnormal conditions produces vacuoles, which are never met with in a fresh state either in the cytoplasm or nucleus, and are with great probability artificially produced by dehydration. Nevertheless, they are a valuable sign of a pathological state of the cell in which its protoplasm is altered so that it contains an excess of watery matter.

No. 3 in the table was an instructive case as regards the formation of vacuoles. The cerebellum was very firm and with fresh films few Purkinje cells were to be seen.

Hardened preparations showed a great increase and prominence of the radial fibres and a layer of large connective tissue elements in the region of the Purkinje cells giving off fibres running straight upwards in the cortex; only a few Purkinje cells remained and these were small, surrounded by leucocytes and often contained a large round or oval vacuole (13-22μ in diameter). None were seen in the fresh cells.

The study of the intimate structure of the cytoplasm revealed by Nissl's process is of the greatest importance in the pathology of the nerve cell. It already allows us to observe changes which were formerly quite beyond our ken, and no doubt it will in the future, when the more exact nature of the chromophilic material is known, be a valuable aid in our efforts to understand the meaning of these changes. Its importance in the pathology of the nervous system can hardly be over-estimated.

FIGURES.

FIG. 1.

Frontal cell from a woman, age 33 ($\frac{1}{12}$ oil, with ocular). Cell $51 \times 39\mu$; nucleolus 5×5. No Pigment. $\frac{500}{1}$.

FIG. 2.

Occipital cell from a woman, age 42, acute mel. ($\frac{1}{8}$, with ocular). Cell 40μ wide; nucleolus $4 \times 4\mu$; nucleus darker than cytoplasm and indistinct. $\frac{350}{1}$.

FIG. 3.

Occipital cell, man, aged 76 ($\frac{1}{8}$, with ocular). Lower half occupied by pigment; chromophilic threads breaking up into segments in apex. $\frac{350}{1}$.

FIG. 4.

Occipital cell, woman, age 20, acute mania ($\frac{1}{12}$ oil, with ocular). Cell 24μ wide; chromophilic matter segmented. No pigment. $\frac{500}{1}$.

FIG. 5.

Two giant cells, woman, age 33 ($\frac{1}{2}$ in., with ocular). Upper one $120 \times 75\mu$; nucleolus 12μ; clump of pigment at lower end; lower one 210×72; nucleolus 9μ. Pigment at right end. $\frac{160}{1}$.

FIG. 6.

Giant cell, woman, age 30, acute mania ($\frac{1}{8}$, no ocular); $186 \times 84\mu$; nucleolus $14 \times 12\mu$. Mass of pigment occupies and distends left end. $\frac{200}{1}$.

FIG. 7.

Giant cell, same woman ($\frac{1}{12}$ oil, no ocular). $\frac{277}{1}$.

FIG. 8.

Occipital cells with pale cytoplasm, no chromophilic matter and well-defined dark nuclei; man, age 63. Chronic mania.

FIG. 9.

Upper cell of fig. 5 more highly magnified.

FIG. 10.

Apex of cell in fig. 6, showing segmentation of chromophilic threads.

FIG. 11.

Motor cell, man, age 47, G.P.; large masses of chromophilic material at base and apex of cell; threads still seen in apex.

FIG. 12.

Senile cell, woman, distended with pigment; chromophilic matter exists as small, sparsely scattered granules, apex broken off.

A STUDY OF A CASE OF ACUTE HÆMORRHAGIC (NON-SUPPURATIVE) ENCEPHALITIS.[1]

BY ALFRED WIENER, M.D.

Adjunct Professor of Nervous and Mental Diseases in the New York Polyclinic.

(*From the Clinic of Prof. B. Sachs.*)

THE occurrence of two distinct attacks of this disease in the same individual, with a complete recovery after the first attack, and with a quickly fatal result after the second, has induced me to put on record my observations in this case, including a report of the microscopic examination of the brain and the membranes. The study of this form of encephalitis is still in its infancy. There is no doubt that many of the cases which heretofore have been diagnosticated as meningitis, were cases of encephalitis. There is a great similarity in the clinical symptoms of these two diseases. In a recent monograph upon this subject, Oppenheim admits that the diagnosis of encephalitis can seldom be established with any certainty, and goes so far as to say, that a diagnosis of this disease is at best only a probable one. It was formerly believed that when this disease was present a fatal issue was inevitable, and that if the patient recovered, he in all probability had not suffered from encephalitis. The cases published by Strümpell-Leichtenstern, Fürbringer and Oppenheim, have shown the fallacy of this view.

In contra-distinction to the polio-encephalitis of Wernicke, which is due to multiple punctiform hæmorrhages in the region of the nuclei of the eye muscles, in the walls of the third ventricle, in the central grey matter of the floor of the fourth ventricle, and in the aqueduct of Sylvius,

[1] Read before the American Neurological Association, at Washington, May 5, 1897.

there is the form described by Strümpell in 1890, and afterwards by Leichtenstern and Fürbinger. This form is due to an acute primary inflammation of the brain, showing on *post-mortem* examination, a hyperæmic condition, with small capillary hæmorrhages scattered here and there in the hemispheres, centrum ovale, in the basal ganglia, &c.

The clinical symptoms of these two types will differ, in consequence of the peculiar localisation of the lesion in each, viz., either in the hemispheres or in the brain axis. Again their etiology is likewise the same, both forms occurring as they often do, after infectious diseases, ulcerative endocarditis, chronic alcoholism and other toxic infections. It is my impression that they are after all only different forms of the same disease. Cases have been reported in which a combination of the symptoms of both forms exist, which certainly very materially strengthens this view. The case of Freyhan published in the *Deutsche Med. Wochenschrift*, 1895, No. 34, is a particularly good example.

Infectious diseases seem to play a very important role in the etiology. Its occurrence during influenza epidemics, as Leichtenstern, Königsdorf, Schmidt and myself (two cases) have lately observed, and the case of Dr. Putnam's after mumps should keep us on the alert, and teach us to consider the possibility of its presence, and thus prevent confusion between it and meningitis. Influenza bacilli have been found in a case reported by Pfuhl, and in one reported by Nauwerck.

The history of the case which I wish to report in this paper is as follows:—

J. J. was two years and nine months of age when he was brought for the first time to the clinic of Dr. B. Sachs. This was in the month of August, 1893. The history given at that time by the mother of the little boy, was that he had fallen down a cellar stairway, striking on the stone steps with his head. When he was picked up he did not appear to be any the worse off for his fall. This happened three weeks previous to the time that he was first brought to the clinic. No visible injury was apparent at the time. On the same day of the accident, the

child played with his brother and sister, and nothing was noticed in its behaviour to excite any alarm. On the following day the child began to become fretful and peevish. On the third day he began to exhibit a tendency to destroy things in general. He tore up his handkerchief and even the dress that he wore. On the fourth day a difficulty in his speech was noticed for the first time. The child also began to totter and fall. On the fifth day speech was much worse and the child could no longer walk; he was becoming more and more drowsy and cried very much. On the evening of the sixth day a convulsion took place. The child was at times in a constant state of restlessness alternating with stupor and screaming attacks. Then again it would have distinctly lucid intervals. Such was the history given by the mother, and on examination the following important points were brought out. Father and mother are both living, apparently healthy although somewhat ill-nourished; father is a shoemaker, mother has had chorea when a young girl, otherwise the family history in regard to any neurotic taint is negative. The family consisted when all were living of seven children, four boys and three girls. One boy died of scarlet fever with convulsions, oldest girl has had chorea. There have been no miscarriages. J. was the fifth child, born at term and with no difficulty at the labour. Learned to talk and walk at the usual time, and was perfectly healthy and well up to the time when he met with this fall. On examination we found a well nourished child, unable to walk or talk and in a constant state of restless activity, tossing itself first to one side and then to the other, moving its head forward and backward, crying very much and drooling constantly at the mouth. Child would make no attempt to stand or walk; when put on its feet, and an effort was made to compel it to walk, it did so in the form of jumps from one spot to another, showing that it still possessed power to move its limbs, but only in a very awkward and atactic manner. Patellar reflexes were present and exaggerated on both sides. Superficial reflexes were also exaggerated. Sensation was normal over entire body. Left also appeared somewhat more affected than right side. Child paid no attention when spoken to. When a strong light was held before its eyes, it made no attempt to follow it. However when the child was watched it was noticed that the eyes could be moved in all directions. Pupils responded to light and accommodation. No paralysis or paresis of the cranial nerves was observed. Urine was free from albumen and sugar. Pulse varied between 60, 80, and 120; temperature was of intermittent

type. The child was seen and examined on several occasions after the first examination, but no change was noticed in its condition until December of the same year when a gradual improvement began to set in. In February of the following year "1894," the child began to walk again. Speech showed no signs of returning until March. The child first began to utter words of few syllables; gradually its vocabulary increased, sentences were formed again, so that by the end of six months the child was almost completely restored to its former good health. I could detect no trace of its former illness. . . . I took the pains to examine the child on several occasions and always with the same result.

The child remained perfectly well until the spring of 1896, when it suffered from an attack of scarlet fever and diphtheria. I did not have occasion to see it during this illness, but I understand that the child made a good recovery. From September 15 to December 9, the child attended school; its reports from school were of the best, and the child seemed to take great pleasure in being allowed to attend. On December 9, the child complained of feeling tired, had pain in its head and back, besides this there were present catarrhal symptoms in its nose and throat. It appeared drowsy, and contrary to its former delight in being able to attend school, it was now inclined rather to stay at home and lie down; it soon became fidgety and restless again, running aimlessly about and at the same time apparently anxious and frightened. Speech disturbances again appeared. For ten days it continued in this manner, growing worse each day, until finally the mother recognizing that the child was again drifting into a state which closely resembled the first attack, brought it once more to our clinic for treatment. The child was sent home, a thorough anti-phlogistic course of treatment was advised and carried out, but was of no avail. On December 23, the fourteenth day of the illness, the child had a severe convulsion, then followed attacks of delirium, alternating with stupor and lucid intervals. There was again the constant drooling of the mouth, the child had the habit of incessantly protruding and retracting its tongue. Twitchings of the various muscles in the arms and legs were frequently noticed, probably a little more on the left side than on the right. It would make attempts to get out of bed, and had to be restrained; the head was again being tossed in all directions. On the 24th another severe general convulsion took place. On the 26th several convulsions occurred, and the stupor which followed was now more complete. Pupils on

examination were unequal, left smaller than right, and no response to light. On the 27th there was delirium, stupor, coma, convulsions and finally death. Temperature during all this time was again irregular, and the pulse was constantly rapid. No proper examination of the optic discs could be made.

Autopsy.—This was made ten hours after death. I found the abdominal organs in perfect condition. Thorax normal, excepting slightly œdematous lungs, and a congested appearance of the upper respiratory tract. The heart was normal; the skull was symmetrical; the diploe was thick and hyperæmic over the left and right parieto-occipital regions. Inner surface of skull was perfectly smooth; the superior longitudinal sinus contained a small parietal thrombus. The dura mater appeared normal to macroscopic appearances; its inner surface was smooth and shiny, nowhere was it thickened. The pia could be stripped off in patches from the cortex. At the bottom of the sulci some difficulty was experienced in detaching it, and often pieces of cortex would come away with it. The lateral ventricles contained only a very small amount of a reddish serous-looking fluid. The choroid plexus was filled with blood; the ependyma was smooth; the third and fourth ventricles and the cerebellum were of normal macroscopic appearances. Several cuts were made through the brain substance, showing it to be markedly hyperæmic and in some places soft and œdematous. The whole brain was placed in Formalin to harden and then prepared for further examination.

Microscopic Examination.—Figs. 4, 6. The blood vessels in the pia were markedly congested and filled with blood. Here and there were small hæmorrhagic infiltrations. There was also noticed an abundant production of cells somewhat resembling the cells which normally coat the surface of the pia. Besides this, a reticulum of very fine fibres covering the entire surface of the pia, which appeared to be fibrinous. This was the appearance of the pia when stripped from the cortex. In some places on cross section, I found it closely adherent to the cortex, and presenting the same pathological picture above described. Pieces of cortex taken from the hemispheres, and sections made from the basal ganglia, pons, and medulla, all show a very characteristic picture. There were many dilated and overfilled blood-vessels (fig. 5). The walls of the blood-vessel, both veins and capillaries, are infiltrated with round cells. The intima does not appear to be affected. The media and adventia coats seem to bear the entire burden. Along the walls of the blood-vessels, and in the perivascular spaces, numerous wandering leucocytes

FIG. 1.

Blood-vessels filled with blood and their walls infiltrated with round cells.

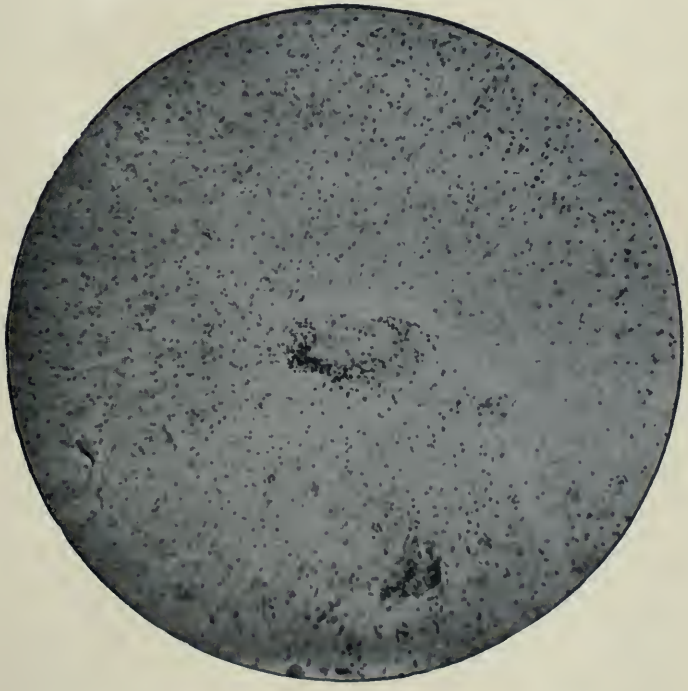

FIG. 2.

Small hæmorrhage with round cell infiltration. Sub-cortical.

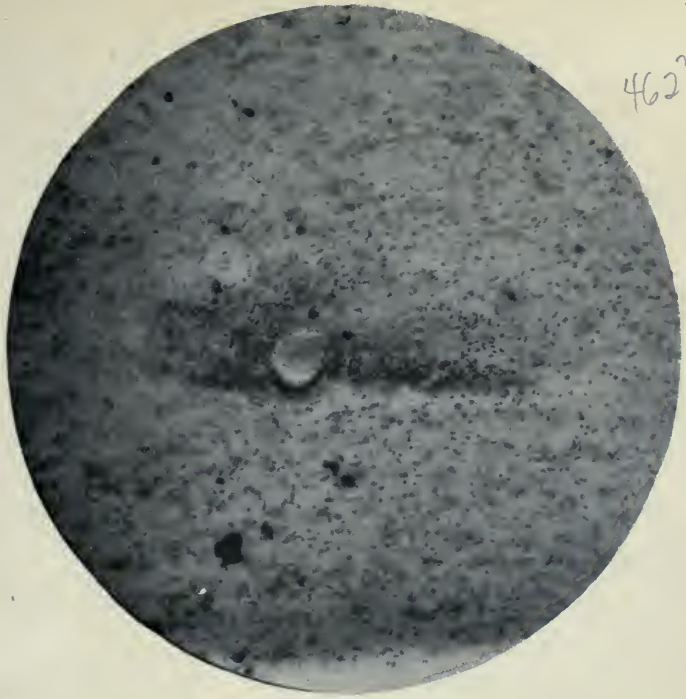

FIG. 3.

Ruptured blood-vessel. Hæmorrhage. Round cell infiltration.

FIG. 4.

Pia as stripped from the cortex. Fibrinous mass covers the entire surface. In the centre of the picture a dilated blood-vessel is visible.

can be seen, together with red blood cells (figs. 1, 3). In the region of the third left frontal convolution (fig. 2), in the anterior extremity of the first temporal, and in the olivary body in the region of the twelfth nerve, capillary hæmorrhages could be distinctly seen. No necrosis of tissue, nor actual destruction of nerve elements was observed anywhere. In regard to the ganglion cells which were examined after the method of Nissl, I found that those which were situated in the neighbourhood of hæmorrhages and very much diseased blood-vessels would not take on the stain very readily. Some looked swollen, and so blurred that neither the nucleus or nucleolus could be recognised. The whole appearance resembled the picture which we recognise as cloudy swelling (fig. 7). The peri-cellular spaces were distinctly enlarged. Although several sections were stained for the purpose of bacteriological examination, no positive results were obtained. Such were the pathological changes present in this case. An acute inflammatory process in the pia and in the brain substance, with a tendency toward capillary hæmorrhages. A picture which is now familiar to us and which we recognise as "Acute Hæmorrhagic Encephalitis," or as I would prefer to say in this case, "Acute Hæmorrhagic Meningo-Encephalitis."

Let us review the clinical features of this case, in the light of these pathological facts.

A perfectly healthy child, 3 years of age, meets with an accident in falling down a stone stairway. On the following day symptoms gradually begin to appear, and at the end of a week the child has suffered from a convulsion, lost its speech, and cannot walk; its mental condition is seriously affected, the child is restless and peevish, there are attacks of screaming alternating with stupor, an irregular temperature, and a pulse at one time slow and at other times rapid, but at no time irregular. There appears to be not so much loss of power in the limbs as an awkward use of the same. The constant tossing of the head from side to side, and the occasional grasping the same with its hands, suggest the probability of headache. No attention is paid to light or sounds. No paralysis of the cranial nerves was observed. Urine was free from albumen and sugar. Optic discs and pupil reflexes were normal. Patellar and superficial reflexes were exaggerated. Such an array of symptoms we were naturally inclined to believe, when we saw this child for

the first time, to be due to a meningitis of the convexity, following trauma. The child gradually began to improve and was well again at the end of six months. In the following three years, with the exception of an attack of scarlet fever and diphtheria, the child remained perfectly well. It developed physically and mentally to the same degree as other children of the same age. Suddenly one day it began to suffer from catarrhal symptoms of the upper respiratory tract, and began to complain and present a picture resembling almost exactly its previous attack. The child now rapidly developed the same symptoms which it had suffered from in its first attack, and in three weeks the disease proved fatal.

With regard to the etiology of the first attack, it would appear that although there was nothing present on *post-mortem* examination to show the effect resulting from a trauma upon the skull, or its membranes, nevertheless this first attack and the trauma must have been very closely associated with each other. Within twenty-four hours after the accident, the first symptoms appeared. The clinical picture that followed closely resembled that of the second attack; the autopsy proved that the second attack was due to an acute hæmorrhagic encephalitis.

That the child inherited a tendency to neurotic troubles, is apparent from the presence of chorea in the mother and in one sister. Taking these facts into consideration, we need not hesitate to attribute the first attack, if not directly at least indirectly, to the injury preceding it. Cases of this kind have been reported by Mauthner, Birdsall and Dinkler. I am somewhat at a loss to account for the second attack; it occurred, however, at a time of the year when influenza was epidemic in New York. This child began to suffer with catarrhal symptoms of the upper respiratory tract, and its general condition suggested the possibility of an attack of influenza. Again this child having had one attack of encephalitis, was naturally predisposed to this disease. This child was also beginning to attend school, and was subjected to the first mental strain.

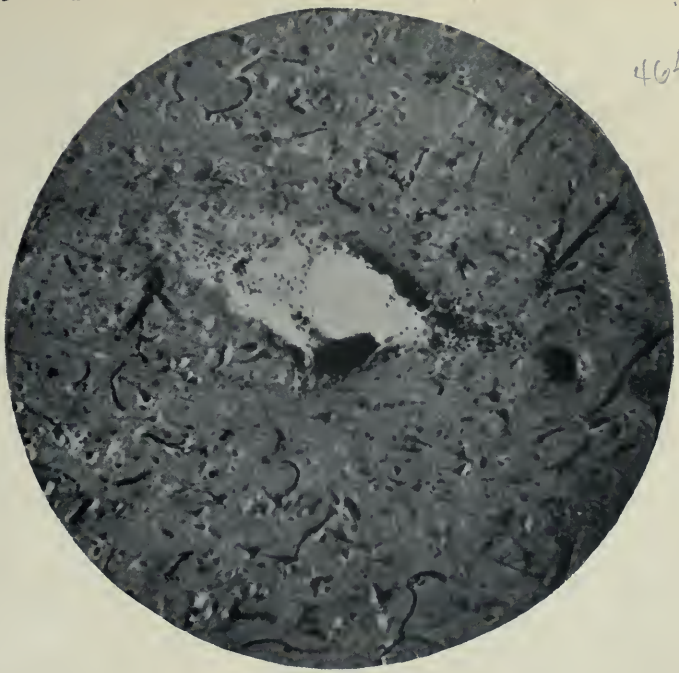

FIG. 5.

Representing a markedly hyperæmic area with a hæmorrhage in the centre.

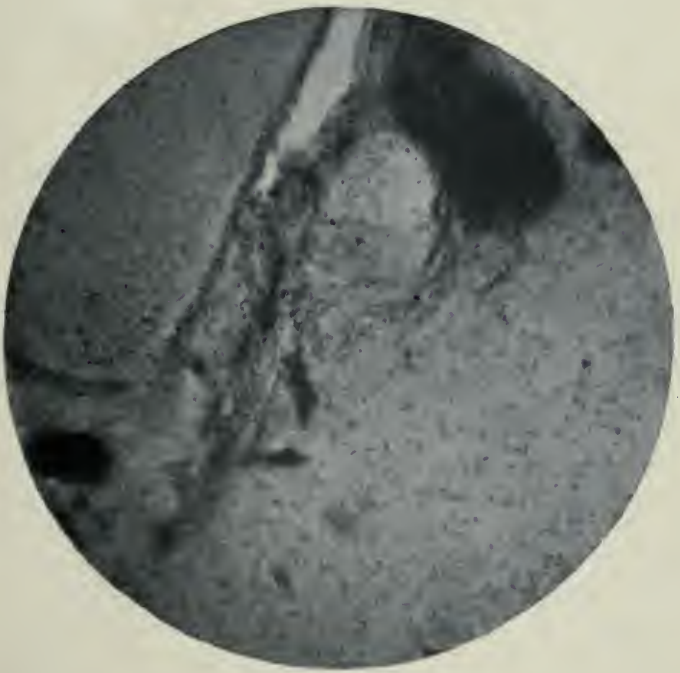

FIG. 6.

Pia on cross section at the bottom of a sulcus. Thickened and closely attached to the cortex. Infiltrated with round cells and containing blood extravasations.

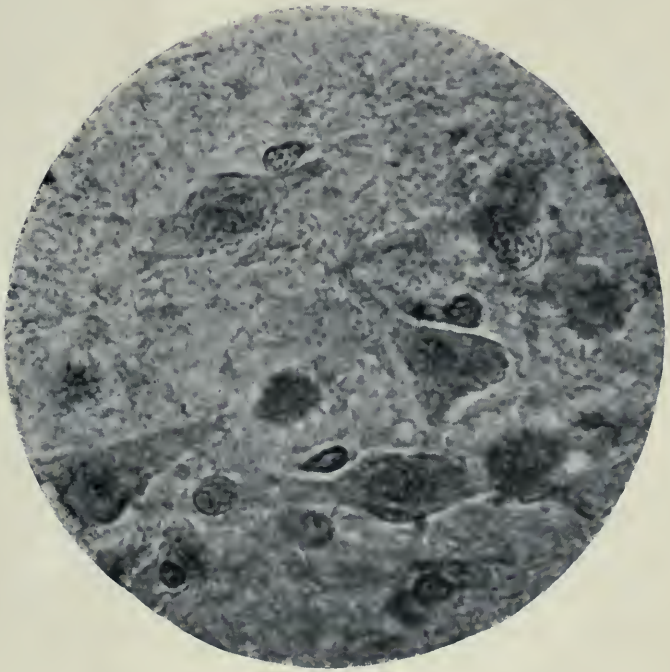

Fig. 7.

Diseased ganglion cells in the cortex, showing the cloudy swelling.

In regard to the pathological condition found, I do not think it necessary to go into details. It thoroughly explains the clinical picture which was present. Before concluding let me direct your attention to one especial feature in this case, viz., the involvement of the pia. This has been found in the cases of Leichtenstern, Eisenlohr, and others. The condition which they found was one of œdema and infiltration of this membrane, whereas in my own case I found a picture which very closely resembles the condition found in fibrinous pleurisy. I am not prepared at present to dilate further upon this pathological fact, and wish therefore to defer further comment.

I wish to express my indebtedness to Dr. B. Sachs for the privilege of reporting my observations upon this case.

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For a complete bibliography of this subject I would refer the reader to the ninth volume of Nothnagel's "Specielle Pathologie und Therapie; Die Encephalitis und Der Hirnabscess," by Prof. Dr. H. Oppenheim, Berlin.

FURTHER NOTES ON GRANULES.

BY ALEX HILL, M.A., M.D.

Master of Downing College, Cambridge.

In two papers published in this journal (Part LXXIII., p. 1, 1896, and Parts LXXVII. and LXXVIII., p. 125, 1897), I described a form of granule which had not hitherto been recognised in the cerebellum. These granules are carrot-shaped, the apical process, usually single, but occasionally bifurcated, tapering, bearing a few lateral branches. The nucleus lies at the thick end of the cell. A nerve fibre of great delicacy is attached to this end. The cells are of the same size as the well-known round granules which were described by Golgi, *i.e.*, 10 μ to 15 μ in diameter.

I have found these cells in the rat, kitten, puppy, and hedgehog. Judging from the closeness with which they appear in certain patches in chrome-silver preparations, they must be extremely numerous. They are not limited to the "granule layer," but are present in all three layers of the cortex of the cerebellum. I find them also in many other parts of the central nervous system.

(A) In the "granule layer" of the cerebellum they are of two kinds, as judged by the course of their axis-cylinder processes. (*a*) Granules of which the apical process is directed towards the fibres of the arbor vitæ and the axis-cylinder process towards the molecular layer. These granules are usually inclined at an acute angle to the fibres; the tapering, apical process enters the layer of fibres; the axis-cylinder process passes vertically outwards, pierces the sheet of cells of Purkinje, and bifurcates in the molecular layer in the same manner as the corresponding process of a round granule. (*b*) Carrot-shaped granules, of which the axis-cylinder process is centripetal and branched.

Fig. A.

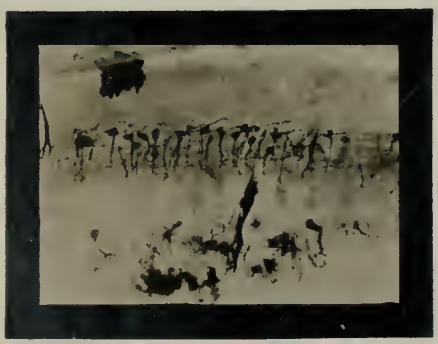

Fig. B.

(B) In the molecular layer also, the granules are of two kinds: (*a*) Carrot-shaped granules, disposed tangentially, with axis-cylinder processes which may be traced for a considerable distance. It is interesting to notice that often the axis-cylinder processes of neighbouring granules run in opposite directions, *i.e.*, the one to the right, the other to the left. Occasionally it is seen to divide into five or six slender branches; in rare cases it loops back upon itself close to the granule. (*b*) Granules placed vertically, with their apical processes directed towards the cells of Purkinje, their axis-cylinder processes centrifugal, or *vice versâ*. In some cases in which it is centrifugal (*a*) the axis-cylinder runs vertically outwards and bifurcates; in other cases (*β*) it inclines to right or left, at an acute angle to the cell.

In an early number of BRAIN I hope to be able to give a detailed description of the various forms of carrot-shaped granule, which I have found, not only in the cerebellum, but in other parts of the central nervous system also; in the meantime the two photographs with which this preliminary notice is illustrated will remove any uncertainty as to the character of the cells, or any doubt as to their dissimilarity from every type of cell hitherto described.

Photograph A (obj. 16 mm. focal length, oc. 4, camera ·8 metre) shows a group of granules lying in the middle of the molecular layer. It will be noticed that the axis-cylinder processes of some of the granules run to the right, of others to the left. The cells of Purkinje were not stained, but they are dimly visible. A cell of Golgi appears at the bottom of the picture. The section was taken from the cerebellum of a puppy 16 days old.

Photograph B is from the same animal (obj. 16 mm., oc. 2, camera ·8 metre). The granules lie in the middle of the molecular layer. Their apical processes are directed towards the cells of Purkinje, the position of which is just recognisable; their axis-cylinder processes run almost tangentially.

STUDIES ON THE NEUROGLIA—II.[1]

BY F. W. EURICH, M.D.

It is a year since we roughly sketched the first results of our examination of the neuroglia by Weigert's method;[2] further investigations have now brought our studies to a stage at which a second publication appears justifiable. A certain amount of repetition of former statements will, however, be unavoidable if clearness of argument is not to suffer.

In pursuing these investigations we have had to turn aside to consider certain questions in comparative anatomy. A brief epitome of the work thus found necessary will, we think, answer the purpose of this Journal better than a detailed account.

The neuroglia, in the widest sense of the term, presents with Golgi's method three great types of cells:—(*a*) The ependyma cell, with its single branched or unbranched process. (*b*) The "astroblast," possessing almost all the characteristics of the former, but that it no longer lines the central canal; and (*c*) The fully formed "astrocyte," or "spider-cell." Two varieties of the astrocyte may easily be distinguished—the one with a small stellate body and numerous long, smooth, unbranched arms (the "fibre-cell" of Andriezen), the other with short, rough, branched processes which give it a peculiar frosted appearance. *Transitional forms between these types and subtypes can be found.* All these types may co-exist in the same animal, either temporarily or permanently; or only two may be found. But the ependyma cell is present in every instance,

[1] Being part of a thesis for the Degree of Doctor of Medicine, Edinburgh University, July, 1897.

[2] In a paper read before the Pathology Section, Annual Meeting of the British Medical Association, Carlisle, 1896.

though it may show signs of atrophy in the adult animal; it may be the sole representative of the neuroglia, as in the amphioxus. The amount of branching displayed by the processes of the ependyma cells and astroblasts depends, *inter alia*, upon the amount of peripheral expansion which a given central nervous organ has undergone in the process of development. The presence of minute hair-like appendages on these processes appears to stand in some relation to the absence of myeline in the areas through which the processes pass; thus, in the chick the "hairs" disappear when the medullary sheaths begin to form; the same can be well seen in the spinal cord of embryonic elasmobranchs, or in the cord of the frog. Where dendritic nerve-cell processes from the grey matter are interspersed in the white columns, as in the lateral tracts of the cord of the spiny dog-fish (*Acanthias vulgaris*) or of the frog, these appendages may be retained. All the characters of the neuroglia are calculated to convey the impression that the functions of this tissue are of a passive, mechanical nature.

The spinal cord, being phylogenetically the oldest part of the central nervous system, always presents the neuroglia in a more advanced condition than do the various parts of the brain, while in the cord itself similar differences in the degree of development of this interstitial tissue may sometimes be discovered. Thus—to take one instance out of many—in the adult frog the cord contains ependyma cells and astroblasts in various stages of evolution, while the cerebral hemispheres of the same animal exhibit ependyma cells only. The higher orders and genera furnish examples to the same effect. In man, too, these differences are shown by the degree of fibrillation to which the neuroglia has attained in various parts of the brain and spinal cord. But we anticipate. For this last statement already touches on a question of histology which has been opened by the publication of Weigert's great monograph on the human neuroglia. We said "opened," but we should more correctly have said "re-opened," for Ranvier[1] had already dissented from the

[1] Ranvier, "De la neuroglie." *Arch. de Phsiol. Norm. et Path.*, February, 1883.

generally accepted view (based on Golgi's silver-impregnation method) that the neuroglia consisted of a feltwork formed by the interlacing branches of innumerable cells. But the method he employed (*maceration, picro-carmine*) was too uncertain in its results. The weight of opposing authority was too great, the simplicity of the conception and the wonderful clearness and elegance of Golgi's silhouettes too convincing for his opinion to gain ground. In 1890 Weigert announced[1] that, by means of a stain which he was still trying to perfect—and which he therefore did not publish at the time—he had come to conclusions similar to Ranvier's. Ranvier's and Weigert's view was this—that in adult man the neuroglia fibres were not an integral part of the glia-cells, but were differentiated from them; that the appearance of the so-called Deiters' cells, in short, was caused by the crossing of fibres on all sides of a small mass of nucleated protoplasm, and that this illusion of a branching cell had been kept up by stains, such as nigrosin, aniline blue-black, and others, which were not sufficiently elective to show this difference between cell-body and fibre. Golgi's method was characterised as particularly liable to mislead, as it displays the cell-elements as silhouettes, and not as transparent objects, thus not permitting an insight into the true relation of fibre to cell. A few years later, Andriezen,[2] apparently without previous knowledge of Weigert's papers, published an article in which he stated that it is sometimes possible, under exceptionally favourable circumstances, to see, even in Golgi preparations, the neuroglia fibres "passing right through the cell-body." At last, in November, 1895, Weigert[3] published his monograph on the Human Neuroglia, with a description of his method of elective staining, embodying the results of many years of labour and patient study. Its appearance aroused renewed opposition, and a series of objections have been put forward, principally by v. Len-

[1] WEIGERT, "Bemerkungen über das Neuroglia-gerüst des menschl. Centralnervsyst." *Anat. Anzeiger*, 1890, p. 543.

[2] ANDRIEZEN, "The Neuroglia Elements of the Human Brain." *Brit. Med. Journal*, July 29, 1893.

[3] WEIGERT, "Beiträge zur Kenntniss der menschlichen Neuroglia," November, 1895.

hossek[1] and Kölliker,[2] which it may be worth our while briefly to examine. It is not a case of "hair-splitting," nor simply a question of the correctness or incorrectness of an unimportant point; for if Weigert's view can be proved tenable, then normal and morbid histology will tend to show that this differentiation of the fibre is, as would be supposed, a further and terminal step in the evolution and development of the neuroglia. But to proceed! Weigert's method stains neuroglia fibres and all nuclei a fine blue, leaving the protaplasm of the cell-body unstained. As for the true fibrous connective tissue, it either remains uncoloured or assumes a violet tint, according as a certain step in the process is omitted or not. The method itself in many respects resembles Weigert's well-known fibrin stain, and upon this fact v. Lenhossek's objection is based. He is of opinion that the staining reaction is due to a difference in the density of the glia-fibres from that of the cell protoplasm, and instances in support of his view, the fact that elastic fibres and other connective tissue are also stained by the process, forgetting to mention, however, that this mesoblastic tissue receives a distinctly different tint, and may even remain unstained if the action of pure chromogen on the sections be omitted. Against the argument itself we would urge the following:—(i.) That the glia-fibres are very sharply defined, so that the transition from the density of the cell-protoplasm to that of the branch (or fibre) must be an exceedingly abrupt one, a condition which, if cell and fibre (or branch) be a perfect corporate whole, would be contrary to all experience. The fact that, in the early stages of certain pathological processes with proliferation of the neuroglia (see later), the blue colour of the glia-fibre is seen to fade gradually into the pallor of the cell-protoplasm cannot militate against us; it is not a permanent feature in these cases, and *must* be noticeable at some time or other during the progress of the differentiation. (ii.) It is very hazardous to draw conclusions

[1] v. Lenhossek, "Der Feinere Bau des Nerven Systems im Lichte neurer Untersuchungen," 2nd edit., 1895, p. 186.

[2] Kölliker, "Handbuch der Gewebelehre, 1896, Bd. ii., pp. 148-150, and 791-793.

as to the chemical or physical similarity of two bodies from similarity in their response to some one chemical or physical test; and such a test—chemical or physical—a staining reaction undoubtedly is. . . . Kölliker enters more deeply into the question. He first falls back upon the researches of Ranvier himself, who was unable to find in the cerebral cortex such differentiated glia-fibres as in the spinal cord; but if we consider that such a differentiation of the fibre from the cell probably represents the most advanced stage in the evolution of the neuroglia, and if we recall that there are phylogenetic differences in the structure of that tissue in the various parts of the central nervous system—that the central cortex, in fact, is "younger" than the cord, then we can easily understand why Ranvier failed to discover such differentiated fibres in that locality. As a matter of fact, they do occur in that region, as Weigert has shown, but they are scanty in health. Kölliker next cites Golgi; the latter had failed to obtain what Ranvier had claimed to have found, though employing the same method, and had remarked that folds in the disc-like body of the glia-cell could closely simulate the passage of a fibre over the surface of the cell body. Weigert has already answered both these objections. The first, it is clear, cannot be urged against Weigert's method, and Golgi's failure was probably due to some difference in the picrocarmine which is known to vary considerably in its staining qualities. As regards the second objection, Wiegert has pointed out that the blue fibres appear as points on transverse section, and that a fold cannot appear as a dot under the like condition. We would add that there is no reason whatever why a fold of protoplasm should assume a deep blue tint, while the rest of the protoplasm remains unstained. As a last objection, Kölliker urges that the short-armed astrocytes, with the frequent attachment of branches to vessel walls, display features which cannot possibly be explained, except by believing the fibres and the cell body to be a corporate whole. Be it so! But Kölliker must have forgotten—or it escaped him altogether—that Weigert does not claim, and never has claimed, for his method that it stains *all* neuroglia fibres; he dis-

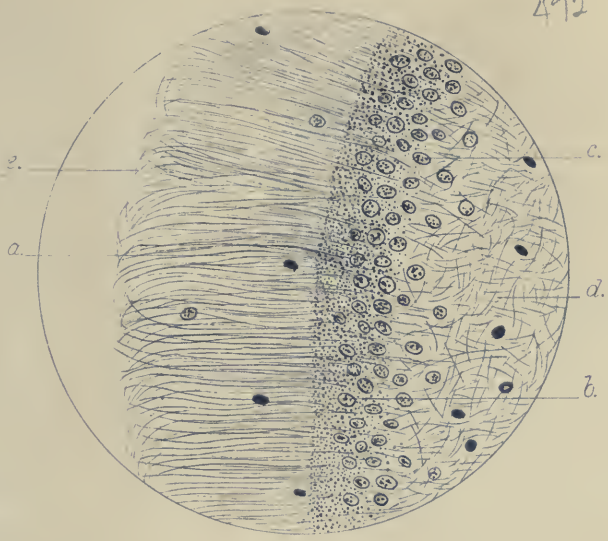

Fig. 1.

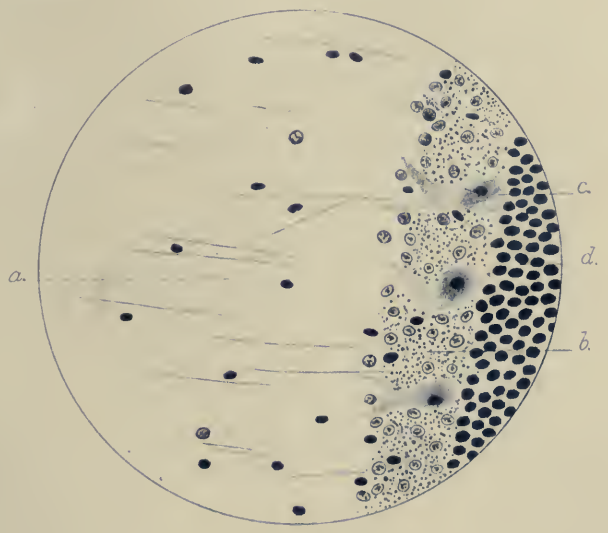

Fig. 2.

Bale & Danielsson, Lith.

tinctly says (pp. 34 and 50, *loc. cit.*):—"there is another possibility, and it is this—that many of these cells are 'astrocytes' in the whole sense of the word, *i.e.*, that they possess not fibres but protoplasmic processes. Such protoplasmic processes, however, are not rendered visible with our method"; and again—"There cannot now remain the slightest doubt that at least the neuroglia corresponding to the so-called 'processes' of Deiters' cells (*i.e.*, the long-armed 'astrocytes') is a true inter-cellular substance, possessing, that is to say, a '*nature connective*,' in the morphological meaning of the term." Having strongly urged all these objections, Kölliker appears to have had misgivings, and propounds a theory which looks very like a compromise, and which in no way helps to simplify matters.[1] He assumes that the glia-fibres spring from a plate or disc which has become differentiated from the cell body, but still lies in contact with it.

We have seen that, from the point of view of function, the branch or fibre is the more important feature of the glia-cell. It must therefore be welcomed as a distinct advance that we now possess in Weigert's method a stain which is not only elective, but which is especially adapted for the study of the *topographical* distribution of the neuroglia.

It is not our present purpose to collect a mass of minute anatomical detail from all parts of the central nervous system. In his monograph Weigert has demonstrated that the distribution of the neuroglia in the various regions follows certain broad principles, and has himself built so extensively on this foundation, that any additions on our part would necessarily lack system. As some of these general principles are of some importance (quite apart from the correctness or incorrectness of Weigert's theory) we deem it advisable to touch upon them here:—(i.) "There is always a thick layer of closely interwoven fibres beneath the epithelium lining the ventricles and the central canal, and this is the densest feltwork to be found in the normal nervous system." (ii.) "With very few exceptions the outer surfaces present a similar concentration of the

[1] KÖLLIKER, *loc. cit.*, p. 150.

neuroglia, but the feltwork is not so dense as in the peri-ependymal layers." (iii.) "These laws hold good not only for the inner and outer surfaces in the adult nervous system, but also for those which are present in the fœtus, and become obliterated in the process of development." (iv.) "Every medullated fibre is separated from its fellow by neuroglia-fibres." "Plexuses of glia-fibres surround the ganglian cells." Thus far Weigert! To these broad statements we should like to add that those parts of the central nervous organs which are phylogenetically the oldest, display these glia-fibres in the greatest profusion, *e.g.*, the substantia gelatinosa centralis. A good instance of this is also to be found in the cerebral cortex. We have had occasion to state that the branches of true astrocytes being protoplasmic and undifferentiated, do not stain by this method. Now, in the cerebral cortex the deeper layer of large pyramidal cells which, according to Betz, is of later development, displays practically no blue-stained fibres, though neuroglia cells can be demonstrated in it by Golgi's method. The inference is obvious; a further argument in favour of this view will be found when discussing the pathology of the neuroglia.

But if the phylogenetically oldest regions display this profusion, it does not by any means follow that every region specially well endowed with these differentiated fibres belongs to that class. All peripheral coverings of neuroglia as defined in Weigert's law iii., are examples of this; but the explanation which we venture to give of these structures will find a better place on a subsequent page. For the minute details of the distribution of the neuroglia we would refer the reader to Weigert's monograph; such details are profitless in the present instance, and we would rather turn to another branch of medical science, and note the behaviour of the neuroglia in various regions under various morbid conditions.

Anatomy and physiology owe many a debt of gratitude to pathology, especially in matters pertaining to the nervous system; for pathological processes are not events out of all harmony with nature, but obey the physiological laws of

the organism. Are there any promises from that quarter in the present instance? We believe that a wide field lies open for investigation in this direction, thanks to Weigert's method; it is a field so rich in virgin soil that we cannot do more than turn a few sods, and perhaps indicate where future workers may hope to reap a harvest. We propose, on this occasion, to confine ourselves to a consideration of *the morbid increase of the neuroglia.*

If a region be examined in which the nervous elements are undergoing a process of destruction, there will be found an increase of cell-elements, which differ in their appearance according to the method employed to demonstrate them; thus, with hæmatoxylin they appear as nuclei with a small amount of protaplasm round them; with van Gieson's stain a few processes become apparent; while, with Bevan Lewis' "fresh method" and aniline blue-black stain, they are seen to be richly branched, spider-like elements, with, may be, one, or sometimes two processes attached to the walls of a vessel. These cells have in times past been termed "neuroglial connective tissue cells," "spider cells" (Jastrowitz), "scavenger cells" (Bevan Lewis), and were considered to be mesoblastic. This conception is still upheld by Hamilton,[1] in the second part of his text-book, published only a few years ago, and finds another advocate in Bevan Lewis.[2] The mesoblastic and the "dualistic" theory will find consideration later. Every part of the central nervous system will, if diseased, present such elements at some stage of the morbid process. They are, however, most readily studied in the cerebral cortex of a general paralytic in the early stage of that disease. If such a piece of cortex be hardened, cut, and stained according to Weigert's instructions, these cells become very distinctly visible; their nuclei are blue, the protoplasm a delicate yellow (from immersion of the section in chromogen), and the processes in various shades of blue, as follows:—The distal parts of the processes are comparatively thin, and deeply stained, but as they approach the body of one of these spider-cells they broaden out, and their

[1] Hamilton, "Text Book of Pathology," vol. ii., part 2 p. 576, *et seq.*

[2] Bevan Lewis, "Text Book of Mental Diseases," 1889.

tint gradually fades, merging gently into that of the cell-protoplasm. In very advanced conditions of the disease, or in old cases of other cortical lesions, such cells are no longer seen. We find nuclei in plenty, and also quantities of blue fibrils, which pass, however, over, under, and on all sides of the nuclei, without any sign of transition into the cell-protoplasm. The fibres appear to have become differentiated from the cell-body. All stages of this process can be seen, from that in which the fibre has taken up the faintest possible blue colouration, to that in which it is difficult to say whether the fibre is still part of the cell or not. A Weigert-preparation, demonstrating the final result of such a morbid process, presents more fibres in the diseased area than are met with there under normal conditions, *but the new fibres differ in no wise from those found in health.* This process, or its final result, constitutes "sclerosis." It is clear that sclerosis is effected by the activity of the same class of elements from which the blue fibres of the normal tissue have originated, viz., the neuroglia cell. Sclerosis—*i.e.*, this formation of fibres—is, however, met with, not only in regions in which such fibres pre-exist in health, but also in areas naturally devoid of them. Such an area is the deepest layer of the cerebral cortex. While not a single blue fibre can be found there in the normal state, yet, under certain morbid conditions, a felt-work of neuroglia becomes visible with Weigert's method. Fresh sections, stained with aniline blue-black, reveal during earlier stages of disease in this layer a comparative richness in spider-cells, when contrasted with the poverty of more superficial strata. Immigration of glia-elements from the upper to the deeper layers is therefore improbable, not only for the reason given, but for another and important one, which will be referred to when discussing cerebellar sclerosis. The spider-cells, and the fibres derived from them, must have developed *in loco* from cells which had not attained to the stage of fibrillation. All these regions, of which we have instanced this cortical layer, contain glia-cells of a type other than the smooth, long-armed "astrocyte," as Golgi's method demonstrates. But if the blue-stained fibres represent the differentiated

branches of the so-called "fibre-cells," or long-armed astrocytes; and if such fibres can, under morbid conditions, be formed in regions in which only the short-armed type prevails, and if immigration can be excluded, then it follows that the short-armed and the long-armed astrocytes belong to the same class of elements, and that Andriezen is wrong when he states that the fibre-cell is true neuroglia (*i.e.*, epiblastic), but that the small bushy variety is of mesoblastic origin. We can therefore affirm that, to whatever stage of development the glia-elements of any region may have advanced, a sclerosis similar in every essential to the process described above may follow the destruction of nerve-elements.

Weigert's stain definitely confirms the glial nature of all sclerotic and cicatricial formations within the central nervous system. We have applied it to the cerebral cortex in epilepsy and general paralysis, to softenings in the basal ganglia, to sclerotic conditions of the cerebellum, to various degenerative diseases of the spinal cord and medulla, to atrophic conditions of the optic nerve—in every instance have we found the identical process, and not a sign of the proliferation of any other tissue.

Thus far it has been shown that sclerosis is in all regions fundamentally the same. We have now to add that certain variations occur, depending upon anatomical differences in the parts affected.

These variations are moreover instructive, for they allow us to recognise that sclerosis is not a confused haphazard process, but that it follows certain laws. In our former paper we instanced the spinal cord in tabes or lateral sclerosis; we showed that, in transverse sections of the white matter, the neuroglia fibres are also cut transversely, appearing as blue dots between the nerve tubules or their remains —in other words, that neuroglia fibre and nerve fibre run parallel to each other. A somewhat less prominent feature is that the septa and trabeculæ are thickened, and that the peri-vascular neuroglia is denser and more plentiful than normal. The sub-pial layer of neuroglia of which these trabeculæ are prolongations, also appears to become more

bulky in cases of long standing—even if a healthy tract intervenes, as Lenhossek had already noted. Thus, in degeneration and sclerosis of the crossed pyramidal tract the corresponding stretch of subpial neuroglia may thicken together with its trabeculæ, while the nerve fibres of the direct cerebellar tract remain apparently healthy. That overgrowth of the neuroglial tissue does not at first seem to destroy the nerve fibres, we find also mentioned by Hamilton (*loc. cit.*) in reference to cases of "hypertrophy" of one cerebral hemisphere due to such overgrowth.

The two directions, the horizontal and the vertical, thus taken by the newly formed fibres, are identical with those followed by the healthy neuroglia; but while the horizontal disposition of the fibres is, perhaps, more favoured in the normal condition, the development of longitudinal fibres distinctly predominates in disease. What has been said of the columns of the spinal cord holds good wherever parallel bundles of nerve fibres degenerate; the optic nerves, where this condition is perhaps found in its simplest form, are an instance in point. How can these facts be accounted for? The best explanation we can think of is the following: The proliferated neuroglia cells upon which the reparative process depends tend at first to conform in shape, and in the direction of their branches, to an earlier type—a type reflecting the character of the "astroblast" in its strong main process stretching to the actual or virtual periphery, and attaching itself there. Other branches develop and increase, and take a longitudinal direction mainly. Why? The duty of the newly formed tissue, is to fill as far as possible the gaps left by the degenerated nerve fibres. This gap extends, in the case of the columns of the cord, both upwards and outwards—roughly in the shape of a cylinder, we will say. Now, any transverse axis of this cylinder is capable of being shortened by shrinking, and by gradual approximation of neighbouring tissues; but the longitudinal axis cannot count upon this help, for the longitudinal axis of any segment of the cord cannot shorten. Here the neuroglia must bear the whole burden and responsibility. That shrinkage of the transverse axis actually occurs and is an important factor,

can be readily seen in the spinal cord from a case of tabes in which the alteration in shape is often very noticeable on transverse section, while distortion in the long axis, *i.e.*, shortening of the posterior columns, is unknown. The course taken by the newly-formed fibres in a sclerosed area appears to us, therefore, to be determined by the following principles :—(i.) A temporary reversion to an earlier type of neuroglia element at first takes place. (ii.) The processes (fibres) of these proliferated glia-elements follow the same directions as the processes of the parent cells. (iii.) The newly-formed fibres will predominate in that direction in which approximation of the neighbouring tissues and shrinkage can give least assistance in filling the gap left by degenerated nervous structures.

We have taken the tracts of the spinal cord to illustrate these principles, and have incidentally mentioned the optic nerves, but any region of sufficiently simple structure could serve as an example. In sclerosis of the cerebral cortex, for example, we find the layer of subpial tangential fibres thickened, similarly the glia-fibres around and along the blood-vessels, while the general direction of the rest of the new fibres is straight inwards towards the deeper layers. A good example is also furnished by the cerebellar cortex.

But the pathology of the neuroglia in the cerebellar cortex is interesting also in other ways. In the outermost, or molecular layer of this cortex, the neuroglia is normally represented by somewhat sparsely scattered blue-stained fibres passing radially outwards to the pia. The cells from which they have sprung lie at the level of Purkinje's cells, as Golgi's method shows. A lesion of this molecular layer gives rise to a great increase in the number of these radial fibres, which are crowded closer together, and appear a little thicker—in perfect accord with principle ii., just laid down. But this is not all. Even if the lesion of the molecular layer be a superficial one, still the whole thickness of the layer is penetrated by the newly-formed fibres—proof evident that the process of repair is undertaken by neuroglia and not by invading mesoblastic elements. And it demonstrates more than this. It shows that an increase of neuroglia over a

certain area does not of necessity imply a primary nerve-lesion of equal extent. The thickening of the trabeculæ passing through the direct cerebellar tract in some cases of lateral sclerosis, described by Lenhossek, and mentioned above, is a further instance of this, though not quite such a clear one, as the thickness of these trabeculæ and of the subpial stratum varies within somewhat wide limits, even in health. The increase in the number of the radial fibres is, of course, accompanied by an increase of the glia-elements. But to whatever depth the molecular layer may have been affected by the lesion, these glia-elements never leave their original site at the level of Purkinje's cells. They never immigrate into the diseased molecular layer. This can be most clearly seen in sections stained with hæmatoxylin; in them a distinct line of nuclei can be discerned at or about the level of Purkinje's cells, and practically none in the molecular layer itself. That these nuclei are neuroglial in nature is evident from their presence in cases of complete destruction of all nerve-elements in that region.

On a previous page we had occasion to refer to what we considered to be a similar behaviour on the part of the neuroglia elements in the deeper layers of the cerebral cortex. If two instances suffice to make a rule—as someone has said—then the following may be formulated:—That sclerosis following a given primary nerve lesion is due to the activity of those neuroglia cells from which the pre-existing normal glia fibres (or branches) of the affected area had originated, and *that such proliferated cells do not migrate* from the site on which they were generated.

In very severe cases of cortical cerebellar sclerosis, with complete destruction of the cortical nerve elements, a few glia-fibres can also be seen, possessing somewhat of a horizontal direction, parallel to the surface, and lying, approximately, midway between the periphery and the row of neuroglia cells mentioned above. The origin of these fibres is not quite clear. We believe that they are, in fact, branches of these very cells, which branches have deviated from their course, as they spring from the cells in a somewhat bush-like fashion, and pass in diverging bundles to the

periphery. But certainly not all have this origin; some probably spring from cells in the molecular layer—cells whose nuclei were mentioned as being visible in *very* small numbers, with the help of hæmatoxylin, &c., embedded in the molecular layer. Ramon y Cajal,[1] in a recent communication, refers to certain cells in this layer which he is inclined to view as glial in nature, though they have not as yet been seen with Golgi's method. Whatever the true explanation may be, their presence does not in any way interfere with the exposition given on the preceding page.

Another form of cerebellar sclerosis is one in which there has occurred a degeneration of the more superficial cells of the *granular* layer among which the bodies of the cells of Purkinje are embedded. When a cerebellar convolution so diseased is cut transversely the neurolgia fibres in the degenerated area appear as fine blue dots, and are strictly limited to the affected part. These fibres are developed from cells lying in the diseased area itself. It will be remembered that the radial fibres of the molecular layer likewise spring from cells situated at about this level; but the two kinds of fibres do not spring from identical cells, as can be seen on careful examination, those for the radial fibres being placed a little further outwards. A consideration of the principle that newly-formed neuroglia elements resemble their progenitors in shape and distribution would also lead us to expect this.

From a study of these varieties of spinal and cerebellar sclerosis, it appears that the neuroglia proliferates and develops along definite lines; and one is even tempted to describe distinct "systems"—(such as the radial system, and another deeper one at right angles to it, in the cerebellar cortex)—were it not that that term has of late been somewhat in disrepute.

Returning to the principle of the non-migration of glia-elements, there may be added the fact that if a lesion be of such a nature as to destroy not only the nerve-elements,

[1] Ramon y Cajal, "Beziehungen der Nervenzellen zu den Neuroglia-zellen," *Monatsschr. für Psych.*, January, 1897. Translated from *Revista trimest. micrograf.*

but the glia-elements also (*e.g.*, a hæmorrhage), then the area so destroyed never undergoes complete cicatrization, but always exhibits a gap or cyst.

But it may be asked—assuming this non-migration of the cells, and granting that the newly-formed cells revert at first to an earlier type—"Have the neuroglia cells of the embryo, then, no power of locomotion, and if so, how do they come to occupy peripheral positions?" We believe that they do not migrate, even in the embryo; that they possess no power of amœboid movement as some authors appear to suppose. One causative factor in the change of position may be their rapid multiplication, by which they are crowded outwards. The chief mechanism, however, appears to us to be the rapid growth of the nervous matter itself. Our theory of the process is this:—The distance between the central canal and the periphery at which the branches of the astroblasts terminate is gradually increased, and unless these branches lengthen the cell body must be drawn outwards. While being drawn outwards they multiply. The cells thus generated being nearer the periphery have a relatively shorter main process, the bifurcation of which takes place nearer the cell body than in the old position. In the further outward passage of succeeding generations this point of bifurcation approaches the cell still more—as Golgi's preparations of suitable material clearly demonstrate. This growth of the nervous centres while increasing the area, has of course also increased and expanded the circumference; the distance between the points of attachment of the divisions of any neuroglia process must, therefore, be increased and their cell of origin drawn up to the periphery; the branching divisions of the process thus gradually apply themselves to the periphery and get to lie parallel to the pia mater. We would give this in explanation, not only of the change in position of the astroblasts and glia-cells in general, but also of the formation of the subpial feltwork, and its prolongations along the vessels. The fact that this subpial layer of neuroglia is present only in the higher classes of animals, and that it is densest in those regions which are, phylogenetically, the oldest, may also in this way be explicable.

Lastly, our theory will—if correct—tend to show that the arrangement and disposition of the neuroglia cells are to a large extent directly brought about by the growth and development of the nerve-elements themselves. In other words, the insulating and supporting structures are procured and arranged mechanically by the growth of those very nervous organs which are to be insulated and supported by them.

One question still remains to be discussed, a question which has already once or twice crossed our path. Is the neuroglia purely epiblastic? Or does it contain elements from the mesoblast also? Or is it altogether mesoblastic in origin? The last can, we think, be summarily dismissed. Epiblastic elements certainly form the neuroglia of the embryo, and the point at issue is this:—Are the cells as found in the adult all derived from these embryonic glia-elements? If we believe that each individual neuroglia cell is directly derived from an ependyma cell, and has to go through every stage of development, from that of the astroblast upwards, then indeed the difficulty is great; for not only does the formation of astroblasts appear to cease long before the nervous organs have attained their full development, but we must also mentally endow the glia-cells with the power of spontaneous movement. Refuge has usually been taken[1] in supposing that undifferentiated spongioblasts have moved outwards in the process of development, and give birth to astrocytes without passing through the astroblast stage. But neuroglia-cells can multiply to form a sclerosis in the adult nervous system, and why cannot they also divide in the embryo? Or are we to assume that a sclerosis, too, is brought about through the agency of such undifferentiated spongioblasts? The whole mechanism of the sclerotic process, as described in these pages, is contradictory to this view. If, however, we adopt the theory with which we have attempted to explain the position of the astroblasts and their derivatives, then these difficulties are to a large extent avoided. We must, on this theory, believe

[1] *E.g.*, v. Lenhossek, "Der Feinere Bau des Nervensyst," 1895.

that every astroblast and astrocyte is capable of multiplying, and that their offspring are characterised by possessing a process standing out from among the rest—thicker, stronger, and passing to the periphery or to a blood-vessel—this process being *the* great family feature. It has been shown in the foregoing that when the branches have become differentiated into fibres they can (in man) be stained according to Weigert's method, and that such fibres can be developed by both the long-armed and the short-armed type of astrocytes. Both these types must therefore belong to the same tissue. Transition forms can, moreover, be found in man, and especially in the lower animals. On all these grounds—both normal and pathological—we feel ourselves justified in believing that the neuroglia is a tissue of epiblastic origin without a trace of the admixture of mesoblastic elements. Positive proof can, however, only be furnished by the discovery in the embryo of mitoses in situations known to be occupied by neuroglia cells only. Investigations on this point are, however, still a desideratum.

In the foregoing pages we have more than once had occasion to express the belief that the function of the neuroglia is a passive one. It is, however, only right to state that other theories ascribing more active and complex properties to that tissue have been put forward by various authorities. Thus Ramon y Cajal, though believing in its insulating properties, is also of opinion that the glia-cells and their branches can expand and contract (like the pigment-cells in the cutis of the frog), and thus prevent or bring about the contact of different nerve-elements, especially of terminal ramifications; in this way he tries to explain associated nerve-action, inhibition of such associations, sleep, &c. The culminating point of his theory is that the will (!) acts on the neuroglia cells and determines the condition of relaxation and contraction. It is self-evident that this theory is absolutely incompatible with Weigert's views as to the histology of the human neuroglia; and it has already been dissected and severely criticised by Kölliker. We can only marvel that a man of

Cajal's mental calibre and scientific accuracy could be tempted to such a theory.

Another function is claimed for the neuroglia by Bevan Lewis—still believed in by some, especially by alienists. According to this, the "Scavenger-cell theory," the glia-cells possess not only the function of removing detritus, but also the power of attacking disease nerve-cells and fibres. What are the facts? Bevan Lewis builds his theory upon the following. Numbers of "spider-cells appear where nervous structures undergo destruction and invade the nervous tissues, which "spider-cells" are attached to vessels by one or more processes and send branches to nerve-cells which they may encircle. The "spider-cells" form fibres and degenerate when the broken-down nerve-tissue is removed. Nerve-detritus can be found in these "scavenger elements." Lastly, domestication is said to have a deteriorating effect on the mental capacities of animals, such as the sheep and the ox; and "scavenger-cells" can be seen in the cerebral cortex of these beasts. To this we would reply: firstly, some twenty years ago Weigert pointed out that any two tissues are preserved in equilibrium by mutual resistance; atrophy of the one lessens such resistance, and proliferation of the other results. Every organ in the body can show examples of this under morbid conditions, yet we would not infer that the proliferated tissue in such cases was playing the part of a scavenger. The neuroglia is no exception; atrophy of nerve-tissue is compensated by growth of neuroglia. In obliteration of the central canal—which furnishes an instructive example—the cubical cells become loosened, and packing the lumen of the canal, are followed by an increase and invasion of neuroglia, fibres from the latter passing between them in all directions. Yet these cubical cells are not attacked by the spider-cells which precede the stage of fibrillation. Nerve-cells, too, may die and yet not be removed; they may calcify instead, as in the hippocampal gyrus in epilepsy; secondly, the attachment to vessels and the feltwork round ganglion cells are normal histological conditions; the latter appearance cannot, there-

fore, be looked upon as evidence that the cell is "attacked" by the "scavenger elements" The increase in this vital activity makes the latter more susceptible to stains, such as aniline blue-black. Further, no proof has ever been furnished that the granules visible in the bodies of spider-cells are composed of nerve-detritus, and are not rather produced by a senile change in the glia-cells themselves; lastly, the glia-elements in the cortex of such animals as the sheep and ox are not in so advanced a condition as those in man; they do not represent a hypertrophied state, having never been smaller; the glia-cells in almost all parts of the nervous system in the lower mammalia are relatively larger than those in man; no sign of degeneration can be found in the cortical nerve-elements themselves. Our interpretation of these "scavenger elements" is, therefore, this, that they can but constitute a form characterising the neuroglia-cell in one period of its life history, and that in any proliferative and reparative process on the part of the neuroglia, this earlier stage must, in the natural course of things, be reverted to before fibrillation, as the final result, can be attained.

Summing-up the results of these "studies" we find that:

(i.) The ultimate stage in the development of the neuroglia cell is its separation or differentiation into free fibre and cell-body; and that this condition is found to a varying extent in different regions.

(ii.) That not all glia-cells reach this stage, but remain as true astrocytes, with protoplasmic branches.

(iii.) Every reparative and sclerotic process is the work of the neuroglia; the fundamental principles of all such processes are the same.

(iv.) These principles consist in:—(*a*) a reversion in type; (*b*) the fibres follow the paths indicated by the pre-existing branches or fibres; (*c*) newly formed glia-cells do not migrate; (*d*) the predominance of fibres in any one direction depends upon the amount of help afforded by shrinking and approximation of surfaces in the other directions; (*e*) both types of astrocytes are capable of developing a sclerosis.

(v.) The neuroglia is in all probability an epiblastic structure, without admixture of mesoblastic elements.

(vi.) The position of the glia-elements in the healthy adult is probably determined by the growth of the nerve-elements themselves.

(vii.) There is no evidence of mesoblastic connective tissue elements assisting in a sclerosis.

(viii.) In sclerosis each glia-fibre has its own area, which area is that "supplied" by the cell from which it has sprung; and each fibre keeps to its own area. In other words, there are "systems"—*sit venia verbo*—of glia-fibres, both in health and disease.

Addendum: Since the above was written, an article on this subject from the pen of Dr. W. F. Robertson has appeared in the *Journal of Mental Science* for October, 1897. This writer differs from us in that he denies a *separation* of fibre from cell, but allows a *differentiation*. To criticise his work would be out of place on the present occasion. We would only point out that, granted but such a differentiation, all the points deduced in this paper still hold good.

DESCRIPTION OF PLATE.

Fig. 1.

Sclerosis of cerebellar cortex; total destruction of nerve-tissue. (From a case of chronic epilepsy.)

a = Sclerosed molecular layer, with its longitudinal fibres.
b = Sclerosed peripheral zone of granular layer, its fibres cut across.
c = Neuroglial nuclei.
d = Sclerosed medulla.

Fig. 2.

Sclerosis of peripheral zone only of granular layer of cerebellar cortex. (From a case of general paralysis.)

a = Molecular layer; no sclerosis.
b = Sclerosed outer zone of granular layer.
c = Cell of Purkinje.
d = Apparently healthy part of granular layer.

Critical Digests.

ON THE TRACTS OF THE SPINAL CORD AND THEIR DEGENERATIONS.

BY ALFRED W. CAMPBELL, M.D.

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Introduction.

It is my object in this digest to confine myself as strictly as possible within anatomico-histological limits. I propose to review at length the observations of those who, as anatomists or histologists, have investigated, experimentally or otherwise, secondary degenerations and scleroses; to consider the publications of workers on the normal anatomy, on the development, and on diseases of the spinal cord, only in so far as they bear upon the spinal nerve tracts; and in conclusion to briefly refer to the changes which occur in the spinal cord after amputations of extremities.

The importance of the contributions which have been added to our literature on secondary degenerations of the spinal cord during the past decade, and the augmentation of our knowledge accruing therefrom, cannot be over-estimated. A reason for the refreshing zeal and industry displayed by workers on this subject is readily found; its mainspring has undoubtedly been the discovery and elaboration by Marchi of his now famous osmium-bichromate method. In the same way as the introduction of the silver method of Golgi has stimulated the neuro-histologist, so the method of Marchi has given a fresh impetus to the neuro-pathologist and proved an especial boon to the experimenter on secondary degenerations; by reason of the delicacy of its working even sparsely disseminated degenerated nerve fibres can be traced with perfect ease and accuracy, and thus many breaches left open by the method of Weigert have now been satisfactorily filled.

To suit purposes of convenience, I shall adopt as my scheme in this periscope the arrangement of the various columns or tracts of the spinal cord into the two main physiological groups,

ascending and descending, and I shall discuss the points concerning the development, the origin, course, connections, and termination of each column individually.

The degenerations which result from spinal hemisections, from sections of nerve roots, &c., and from cerebral and cerebellar lesions, will not be dealt with under special headings, but they will be referred to under the particular spinal tracts which they affect.

Pyramidal or Motor Tracts.

The pyramidal or motor tracts have certainly been more attentively studied of late years by experimental neurologists and neuro-pathologists than any other spinal tract, and consequently there is no other tract concerning the course, the connections, and the functions of which we are more enlightened. Yet, while congratulations are due to the various investigators who have assisted in bringing our knowledge of the tract to this advanced stage, there are, nevertheless, several points, as will be presently indicated, bearing more especially on its connections and its termination, which demand further elucidation and prevent us from declaring that our researches on the subject are yet complete.

Development.—In regard to development, Flechsig's most valuable researches have definitely proved that in the human fœtus there is no trace of myelinisation of the fibres of the pyramidal tract until the end of the normal period of gestation, and yet certain other white columns receive their medullary investment, and can be readily recognised on that account as early as the fifth month of intra-uterine life. Then Bechterew has shown, that in those animals which are endowed at birth with the power of locomotion, the pyramidal tracts are almost completely developed before the termination of the period of gestation. Bechterew has also demonstrated that in animals possessed of coarse movements only, such as the whale, the pyramidal tracts never attain a high stage of development; and it may be accepted as a general rule of comparative anatomy that the degree of development of these tracts is proportional to the multiplicity, complexity, and delicacy of the movements of the animal.

In a research having as its object the determination of the period which elapses before secondary degeneration becomes evident in the various tracts of the spinal cord after an experimental lesion, Schäfer arrived at the conclusion that an analogy exists between the time of onset of secondary degeneration and the period of development of the medullated sheath of the fibres composing these and other tracts. Schäfer employed the cat in

his researches, and after a hemisection of the cord found that degeneration commenced in the column of Goll on the fourth day after the operation, it appeared in the dorso-lateral cerebellar tract on the sixth day, but it was not until the fourteenth day that it became evident in the one of tardiest development, viz., the pyramidal tract. Worotynski has written in confirmation of Schäfer's observations.

Volume.—Recent work confirms the variability in relative volume and representation of the anterior and lateral pyramidal tracts one to another in the spinal cord. According to Flechsig's original doctrine, their relative volume varies with the extent and completeness of the crossing at the point of decussation in the medulla oblongata. In 75 per cent. of cases this decussation is relatively equalised, and well developed anterior and lateral pyramids, which we may regard as normal, are to be found. In the remaining 25 per cent. the crossing is either unequal, incomplete, or excessive; if the decussation be unequal, that is to say, if there be more fibres crossing from one side than from the other, then in that side from which the major number of fibres decussate the pyramidal tracts, both anterior and lateral, will be poorly represented; if, on the other hand, the decussation be incomplete, then the relative size of the direct pyramidal tracts to the lateral ones will be increased above normal; and if, finally, the decussation be complete, then the condition is arrived at which Boyce, Sherrington, Singer, and others have proved to exist in some of the lower animals, viz., the cat, dog, and monkey, in which there is an almost complete absence of the direct pyramidal tracts and relatively large sized lateral tracts.

Length.—In the same way as the volume of these tracts is for the above-mentioned reasons exceedingly variable, so their length is never exactly equal in different individuals, a fact which accounts for a number of discrepancies which appear on reading the observations of various writers. Thus, whereas Bouchard told us that the direct pyramidal tract terminated in and did not descend lower than the mid-dorsal region, Tooth has traced it to the second lumbar segment, and the most recent observers of all, Déjérine and Thomas, assisted by the method of Marchi, have now followed it down to the level of the sixth sacral pair of nerve roots. Then in regard to the lateral pyramidal tract, Loewenthal and Vulpian fixed its termination at a point opposite the origin of the second lumbar pair of nerve roots, Tooth saw it opposite the fourth lumbar roots, and Déjérine and Thomas now find that it actually ends in the conus medullaris, but ceases to exist as a compact bundle at the level of the fourth sacral roots.

Origin and Course.—That the trophic cells for the fibres of the pyramidal tracts are the large pyramidal or multipolar cells situated in the motor region of the cerebral cortex, *i.e.*, the ascending frontal and ascending parietal convolutions and the posterior parts of the first and second frontal gyri, is almost unanimously agreed. But a destructive lesion involving these parts gives rise to secondary degeneration, not only of the recognised motor tracts, but also of other systems of fibres; and though it is not definitely known that the performance of motor acts is dependent upon these other systems, yet it is more than probable that there is some relation, and since the view that a pure motor act is considerably more complex than it was originally supposed to be is every day gaining more supporters, it is possible that these systems aid in the production of that complexity, and it will, therefore, be necessary to mention them.

In the first place Monakow, Redlich and others, after destruction of the motor area of the cat, found marked evidence of degeneration in various parts of the thalamus opticus; the same observers, and also Meynert, likewise found degeneration in the substantia nigra of Sœmmering. Redlich in the human being, as well as in lower animals, has constantly found degeneration of fibres leading to cells in the nuclear matter of the pons, and he suggests that these are collaterals of the pyramidal fibres, and that they are connected by means of a second neuron[1] with the opposite cerebellar hemisphere. Also there have been found degenerated certain fibres of the lemniscus (viz., the lateral pontine bundle of Schlesinger) and internal and external arcuate fibres; and, lastly, fibres have been followed from the pyramidal tract to certain cranial nerve nuclei, viz., the facial and hypoglossal (Hoche).

One has only to contrast the small size of the pyramidal tracts as they lie side by side on the ventral aspect of the medulla oblongata, with the size of the tracts in the crus for instance, to be convinced that an appreciable number of fibres must diverge in the manner above indicated, and Starlinger's experiments on the dog seem to prove that these diverging fibres possess a most important bearing on motion, for he found that section of both pyramidal tracts in the medulla oblongata was attended by no lasting interference with movement, whereas the usual paralysis

[1] Throughout this digest the term "neuron" is employed in its broad and more generally accepted sense, *i.e.*, it includes a cell body along with its dendritic processes or neuro-dendrons and its axis-cylinder process or neuraxon. This note is deemed necessary because some apply the term to the axis-cylinder process only.

followed a lesion of the motor area in the same animal. Redlich obtained like results in the cat, and to account for the opposite consequences in these two operations, they conclude that some fibres other than those embodied in the pyramidal tracts in the medulla oblongata must exist as channels for the conduction of motor impulses, and that some of the diverging fibres above-mentioned must form some of these channels. Doubtless fresh researches will shortly shed new light on this point.

Otherwise there is nothing new to remark concerning the disposition of the motor tract in the internal capsule, the pes, the pons, or the medulla oblongata above the decussation. The exact nature of the decussation of the pyramidal fibres at the lower end of the medulla and elsewhere is, however, still a source of fruitful discussion. This portion of the tract has been dealt with at length by Rothmann; and Boyce, Mott, Déjérine and Thomas, Redlich, and others have made incidental references to it in connection with remarks on the occurrence of bilateral degeneration in the spinal cord in consequence of a unilateral cortical lesion, and since these two subjects are inseparable they will be dealt with together at this stage.

(*a*) *The Pyramidal Decussation;* (*b*) *Bilateral Spinal Degeneration in consequence of a Unilateral Cerebral Lesion.*—Bilateral degeneration in consequence of a unilateral cerebral lesion was first drawn attention to by Westphal as early as 1875. Pitres worked the matter up very thoroughly some years later, Muratoff and others have obtained the degeneration in dogs, Mott and others have found it in the monkey, and Boyce and others have seen it in the cat. Numerous and intricate theories have been promulgated to explain this bilateral degeneration, and though most of these are happily now of historic import only, yet it will be interesting to give a brief outline of the more important ones.

Firstly, Charcot believed that in addition to the usual decussation in the medulla oblongata there occurred an extra crossing in the anterior commissure of the cord, but the method of Marchi overthrows this view by failing to reveal degeneration in this situation. Then Hallopeau, who received the support of Marie and Pitres, thought that at the decussation in the medulla oblongata the fibres from either side come into such close contact with one another that direct extension of the inflammatory process from the diseased to the normal fibres must occur.

Passing over the work of Franck, Moeli, and Schäfer, we next come to the very thorough experimental investigations of Sherrington, which led him to assume that the degenerated fibres,

which are to be seen descending in the lateral pyramidal tract on the same side as the lesion, are derived from fibres of the opposite lateral tract, which, having already decussated at the usual situation, leave it to recross in the grey matter of the spinal cord and regain the side of the lesion without coming into connection with ganglion cells in the grey matter through which they pass. For these fibres he suggested the name "re-crossed pyramidal fibres." Sherrington's observations received support from Unverricht, Kusick, and Vierhuff; but, on the other hand, Loewenthal, Fürstner, and Knoblauch, who practised like experiments, did not get similar results.

Now it must be mentioned that all these observers worked before the method of Marchi, which is so exquisitely adapted for picking out isolated degenerated fibres, was discovered, and as they could only practise the staining methods of Weigert, &c., much of the value of their researches must be discounted.

Since the introduction of Marchi's method the matter has been taken up, amongst others, by Sandmeyer, Muratoff, Boyce, Mott, Hoche, and Rothmann.

Sandmeyer obtained bilateral degeneration nine days after experimental cortical lesions, but offers no explanation as to its occurrence.

Boyce and Mott in the cat and monkey respectively, Déjérine and Thomas in the human being, Muratoff in the dog and in the human subject as well, endeavour to explain the homo-lateral degeneration by a direct passage at or about the level of the decussation of fibres from one cerebral hemisphere to the lateral pyramidal tract on the same side as the lesion.

Marchi, as a result of experimental investigation, attributes the bilateral degeneration to an extra decussation higher up, viz., in the corpus callosum. This idea is supported by Hamilton, but it has been practically nullified by the work of Muratoff and others.

We lastly come to the observations of Rothmann, who examined the spinal cords of dogs operated on in Munk's laboratory and obtained the bilateral degeneration in every instance. His view as to its production is in a sense a revival of that of Hallopeau above mentioned, viz., that it originates at the pyramidal decussation, and that it is due to compression of the bundles of fibres from the healthy side by the swollen bundles of degenerated fibres which are coming from the side on which the lesion was. He urges in support of this that four weeks after the operation the homo-lateral degeneration is no longer recognisable, and publishes convincing drawings to illustrate his views.

From the foregoing it is painfully obvious that the anatomical disposition of the pyramidal fibres at the decussation is by no means clearly understood, and that the source of the bilateral degeneration is not agreed upon.

Topography and Termination of the Lateral Pyramidal Tract.—In regard to the topographical distribution of the lateral pyramidal tract in the spinal cord there is little new, only the method of Marchi has shown that its limits are not so sharp as was formerly supposed, and that scattered aberrant fibres are to be seen in considerable abundance in the columns situated immediately ventral to the old boundary of the tract. These fibres are especially noticeable in the cervical region.

We still require enlightenment as to how and where the pyramidal fibres terminate in the grey matter. Kölliker, Golgi, Ramon y Cajal, Sherrington, and Lenhossek, mainly aided by the method of staining by metallic impregnation of the myelin, have shown that the pyramidal fibres at intervals in their course along the cord issue numbers of collaterals, and Lenhossek claims that these collaterals are not directly connected, as one would suppose, with the ganglial cells of the anterior cornua, but only indirectly so by the intervention of cells of the posterior cornua to which they course and around which they arboresce. This statement, however, requires confirmation. Then, in regard to the termination of the main trunks of these fibres, physiologically proof is not wanting that they are connected with the multipolar cells of the anterior cornua on the same side, but anatomically that link of the fibre, situated between the lateral column and the anterior cornual cell, has not yet been satisfactorily defined. Experimental workers (Starlinger, Monakow, Rothmann, Redlich) employing the method of Marchi, have been unable to trace degenerated fibres from the lateral column to the anterior cornual cells; but it is possible that these observers examined the cords of their animals too soon after the operation, and that they would have detected degeneration if they had allowed them to live longer; because in the case of the spinal cord of a man examined by the writer, in which there was profound descending degeneration in consequence of an extensive cerebral thrombosis six weeks old, the method of Marchi showed distinct darkening of the myelinic plexus in the anterior cornu on the side of the degenerated lateral pyramid, though a connection between any fibre in the lateral column and any anterior cornual cell in its entirety was not traceable. Of course, the difficulty here is that not only is the course of the fibre apparently tortuous, but also it

more or less entirely sheds its myelinic investment as soon as it enters the grey substance, and therefore cannot be clearly followed by a method which has for its object the display of disintegrating myelin.

Monakow discredits the existence of a direct connection between lateral column fibre and anterior cornual cell, and assumes that between the spino-muscular or anterior cornual cell neuron and the cortico-spinal or pyramidal cell neuron, a third neuron is interposed, composed of a cell after the nature of Golgi's second type, and that this cell is situated in the region of the processus reticularis.

Anterior or Direct Pyramidal Tract.—Turning next to the direct pyramidal tract, it has been proved that this tract practically does not exist in the cat or the dog, and likewise in the monkey there appears to be no adequate homologous representation; also, in the light of recent research, the tract is being divested of much of the importance it originally possessed in the human being, and is considered to be less extensive and less voluminous than Ludwig Türck, its original discoverer, taught us to believe. Türck's description of the tract was founded on observations of the changes in the cord resulting from capsular cerebral lesions, and not from cortical lesions. Now the difference between the degree of degeneration in the anterior pyramidal tract after a cortical lesion contrasted with that after a pontine or a capsular lesion is exceedingly marked. In the case of the latter the degeneration in the antero-internal column is profound and complete, but in a case of destruction of the cortical motor sphere the direct pyramidal tract degeneration is never more than partial, that is to say, the degenerated fibres are disseminated throughout it, and are surrounded by an equal or greater number of healthy fibres. These points of difference are well seen in a number of specimens of sections of cords of human beings prepared by the writer. There is no entirely satisfactory explanation of the difference in degree of the degeneration in the anterior pyramid in these two lesions; it can only be assumed that certain fibres which course down in the antero-internal tracts are not derived from the cerebral cortex, but take their origin from structures in the capsular region. For instance, Boyce's cerebral hemi-extirpation experiments in the cat show that the fibres of the posterior longitudinal bundles descend in the antero-internal tract on the same side as the lesion. I am not aware that these fibres have been traced so far downwards in the human being, but it is possible that they can be, and that the source of these fibres, by

Bechterew supposed to be in the optic thalamus, is destroyed in a capsular lesion, whereas it of course escapes in a cortical one.

The anterior pyramidal fibres, like the crossed ones, issue numbers of collaterals in their downward course, and these collaterals, and likewise the main trunks of the fibres, are believed to pass over in the anterior commissure, and to terminate by arborescing round cells in the opposite anterior cornu.

"*Retrograde Degeneration*" *in the Pyramidal Tract.*—The name "retrograde degeneration" has been assigned by Sottas to an ascending change in the pyramidal tracts, which that author observed in four cases of medullary syphilis, in which the lesion was a softening followed by sclerosis, situated in and extending for a variable distance along the dorsal region of the spinal cord. The ascending change was prolonged upwards into the cervical region, it diminished in intensity as it ascended; topographically it corresponded, more or less exactly, to the distribution of the lateral pyramidal tract, and finally it disappeared in the upper part of the cord.

Gombault and Philippe have described similar instances of "retrograde degeneration" in two cases of syringomyelia and two of myelitis; in one of the cases of syringomyelia there was ascending sclerosis of the anterior pyramidal tract, as well as of the lateral.

This retrograde alteration possesses the following characteristics, which distinguish it from Wallerian degeneration. It is of tardy development; it undergoes a gradual diminution in intensity and extent as it ascends, and it has certain quite distinctive histological features. These are, that on transverse section the affected tract seems to be riddled with circular or oval lacunæ of varying dimensions; examined more closely, these lacunæ appear to be altered and enlarged nerve fibres. In the centre of some an axis cylinder is still clearly visible; often normal, sometimes swollen and not susceptible to stains. The spaces frequently contain *débris* of an indefinite nature, and the delicate rim investing them is probably the altered neurilemma or primitive sheath, the myelin of the fibre having disappeared. Throughout the affected tract healthy fibres are disseminated; these increase in number as one ascends, but in the immediate neighbourhood of the lesion the lacunæ greatly preponderate.

The method of Marchi only shows scattered fibres undergoing degeneration. This retrograde change might receive divers interpretations, but, as Gudden, Forel, Monakow, and Darkschewitch have shown, when a system of fibres becomes separated

from its trophic centre, it, as a matter of fact, degenerates in two directions, centripetally as well as centrifugally, but the centripetal degeneration is the least pronounced, is best observed in young animals, and only evidences itself after a more or less prolonged period. The latter condition was fulfilled in all Sottas' and Gombault and Philippe's cases.

It would appear that the changes in the spinal cord in "retrograde degeneration" bear some resemblance in a pathological sense to those seen in the cord in certain cases of amyotrophic lateral sclerosis, viz., those cases in which the disease first definitely proclaims itself in the spino-muscular neuron, and thence spreads upwards along the cortico-spinal neuron, and not those cases such as one described by Mott, in which the degeneration seemed to appear more or less simultaneously along the entire motor neuron. To explain the degeneration of the motor neuron in amyotrophic lateral sclerosis, neuro-pathologists, accepting the neuron theory as their basis, assume that its initial cause is some obscure loss or diminution of function in the trophic cell of the neuron, situated in the motor cerebral cortex or the anterior cornu of the spinal cord. Now, it is readily conceivable that retrograde sclerosis can be explained on the same theory, for, given an initial destructive lesion in the course of any neuron, we, in the first place, have a more or less immediate necrosis of the distal portion of that neuron ; then, secondly, since there is no further call for the service of the central nerve-cell, either in its motor or its trophic function, it can reasonably be assumed that that cell, and likewise the portion of the neuron situated central to the lesion, must naturally undergo a slow process of atrophy, which will ultimately lead to total decay.

If the above view be correct, then it is evident that "retrograde degeneration" cannot possibly be confined to the pyramidal tracts, but must affect other systems of fibres in equal measure, and evidence that this does occur is forthcoming, for on closer examination of sections of the spinal cord from a case of old-standing thrombotic destruction of the cerebellum, which I reported some years ago, in which there existed a degenerative sclerosis of the direct cerebellar tract in its entire length, I find that they present all the histological characteristics of "retrograde degeneration," and I now feel convinced that the case should be ranged in that category. An instance of "retrograde degeneration" occurring in the ascending antero-lateral tract of Gowers' has also been recorded by Gombault and Philippe. Furthermore, the changes in the nervous system to be found after the amputation

of extremities, viz., the spinal hemiatrophy, the disappearance of groups of nerve-cells in the anterior cornua which are known to be related to groups of muscles in the amputated part, must be ascribed to a similar process.

LITERATURE ON THE PYRAMIDAL TRACTS.

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(a) Comma-shaped Tract of Schultze and (b) Oval Centre of Flechsig.

In the *Archiv. für Psychiatrie* for 1883 will be found Schultze's original report of the case of spinal compression in which, in addition to other tracts, this small bundle of fibres in the postero-external columns which now bears that writer's name was found degenerated. Schultze described the bundle as appearing on transverse section in the form of a comma-shaped line in the postero-external column, a line running parallel to the inner edge of the posterior cornu, with its anterior extremity not quite reaching the posterior commissure, and its posterior extremity stopping short some distance off the periphery of the cord. Schultze was unable to follow degeneration in it more than two or three centimetres below the site of the lesion, and supposed that the bundle was composed of descending offshoots of posterior root fibres, a view which we now believe to be incorrect.

It must be mentioned that "kudos" for the discovery of this particular descending degeneration in the posterior columns should not be solely allotted to Schultze, for Bastian, as early as 1867, in a case of injury to the spinal cord in the cervical enlargement mentions such a change, and likewise Kahler and Pick, Strümpell, and Westphal, prior to Schultze, published cases exhibiting analogous degenerations.

More recently detailed descriptions of important cases observed by Gombault and Philippe working conjointly, Barbacci, Tooth, Bruce and Muir, and Hoche, serve to fill up many blanks in the anatomy and connections of this curious tract.

Tooth's case was one of fracture of the spine with consequent almost complete transverse myelitis between the eighth cervical and the first dorsal segments. Degeneration in comma form could be followed down the cord to between the sixth and seventh dorsal segments, lower, therefore, than in Schultze's case, though the method of Marchi does not seem to have been employed.

Gombault and Philippe, in a lengthy paper on systematised lesions in the white columns of the spinal cord, give a detailed description of this bundle of fibres; they review and criticise the work of previous writers, and fully report valuable cases which throw a flood of light on the matter, and force us to considerably modify our views respecting the regional distribution and connections of the tract. A case of complete sclerosis of the fifth and partial sclerosis of the fourth posterior cervical roots, the change being attributable to Saturnism, is adduced for the purpose of disproving the original view promulgated by Schultze, that the comma-shaped tract is formed by descending offshoots of the posterior root fibres. In the case in question, though the root lesion occasioned the familiar ascending changes above the point of entry of the roots, absolutely no degeneration in the posterior columns below the lesion, such as one would expect if the above-mentioned connection actually existed, was discernible. This view of Gombault and Philippe's does not stand unsupported in the literature, for Tooth previously discredited the connection, observing in the spinal cords of animals, in which the posterior nerve roots had been experimentally divided, that no descending degeneration in the comma of Schultze occurred as a result of the operation.

Two cases of spinal compression, one at the eighth cervical, the other between the fourth and sixth dorsal roots, reported by Hoche, furnish a clue to the termination of this tract. The cords were examined by the method of Marchi, and the fibres of the tract were seen to end above the first appearance of the "oval centre of Flechsig," that is, above the twelfth dorsal segment, by passing into the grey substance of the posterior cornu; and as no other observer has traced the tract lower than this level we must for the present accept Hoche's observations as correct.

We might sum up our remarks on this bundle of fibres by saying that it is a small comma-shaped tract, situated in the postero-external column, which degenerates in a descending direction in consequence of a transverse spinal lesion. It arises in the cervical part of the cord, but in what manner and from what cells is unknown, and it terminates in the grey substance of

the posterior cornu in the lower dorsal region. It is independent of the posterior nerve roots, and is probably a system of endogenous internuncial or commissural fibres.

Hitherto one has only mentioned cases of lesions in the upper dorsal or cervical levels of the spinal cord which have given rise to descending degeneration in the posterior columns. Now there are several most interesting cases on record of lesions at lower levels, which have likewise given rise to a descending posterior column degeneration if anything more pronounced, and the question has arisen whether these descending fibres in the posterior columns in the lower levels of the cord are analogous to the fibres which compose the comma-shaped tract of Schultze in the upper levels. The point has been widely discussed, but as yet no satisfactory conclusion has been arrived at. Here is an epitome of the more important cases. In the first instance Gombault and Philippe, in a case of traumatic transverse myelitis situated at the level of the lumbar swelling, found below Schiefferdecker's traumatic zone, *i.e.*, the zone of complete degeneration, which extends through the breadth of the cord for some few millimetres upwards and downwards from the site of the lesion, descending degeneration in the posterior columns, confined to a wedge-shaped triangular area, situated on each side of the posterior median septum, the apex of the triangle pointing to and reaching the commissure, the base being at the periphery. The extent of the degenerated area proportionately diminished in its downward course, and in sections made at a point immediately above the conus medullaris, the apex of the triangle was situated at a point equidistant between the commissure and the posterior margin of the cord.

Bruce and Muir, in a case of fracture dislocation completely destroying the cord in the upper lumbar region, found by the method of Marchi a similar descending degeneration. At the third lumbar root the diseased fibres became collected along the margin of the hinder portion of the postero-median septum and the periphery of the inner half of the posterior columns. At the level of the fourth lumbar root, the tract extended forward as a narrow band along the side of the septum as far as the posterior commissure. At the first and second sacral roots it had the form of a narrow wedge with its base at the periphery and its apex at the posterior commissure. Below the third sacral root the tract diminished in size as far as the coccygeal segment, but retained its triangular form and its relation to the postero-median septum, and the degeneration terminated by passing forwards into the grey matter at the base of the posterior horn of the same side.

In Hoche's two cases of spinal compression in the cervical and dorsal regions respectively, mentioned above as showing degeneration of the comma-shaped tract of Schultze, there also occurred a degeneration in the lumbo-sacral region, which, judging from his drawings, is identical with that found by Bruce and Muir, and similar to that described by Gombault and Philippe. The special point of interest in Hoche's cases is that they show what the upper course of this tract of fibres is. One segment below the lesion he describes them as appearing in the most lateral and external part of the postero-lateral column; they then gradually retreat along the periphery until at the twelfth dorsal level they reach the posterior median septum, and then assume the position described above in Bruce and Muir's case.

In 1891 Barbacci, also using the method of Marchi, reported a case of an intra-medullary tubercular focus situated at the level of the sixth and seventh dorsal nerve roots in which descending degeneration was found very much resembling that described by Hoche, and Kahler and Pick and Daxenberger have narrated cases which might be placed in the same category.

These observations, therefore, conclusively prove that in addition to the comma-shaped tract of Schulze there is another tract which degenerates downwards in the posterior columns, one of greater length (at least 23 spinal segments—Hoche), arising in the cervical region (Hoche), and terminating in the grey matter of the conus medullaris (Bruce and Muir, Hoche and Barbacci); but what its precise origin and connections above are, and also whether it is related to the comma-shaped tract of Schultze, is undetermined. Hoche and Gombault and Philippe regard it as a commissural tract of endogenous origin, and although, as Hoche emphasises, the comma-shaped tract of Schultze absolutely terminates in the lower dorsal region, and takes no part whatever in the continuation of the tract, yet Gombault and Philippe's suggestion that the two tracts are analogous is a reasonable one, because the following resemblances exist:—Both are situated in the posterior column, both degenerate in a descending direction in consequence of a transverse lesion, and both are independent of the posterior roots. Gombault and Philippe proved the latter point in connection with the lower tract by quoting a case of tabes, in which the fourth, fifth, and sixth posterior sacral roots were completely atrophied, and this group of fibres remained unaffected by degenerative processes, while the remainder of the posterior columns was completely sclerosed. There seems, lastly, to be no doubt that the "oval centre" of Flechsig in reality represents the lumbar segment of this tract, and that the "dorso-

median sacral bundle" of Obersteiner represents the sacral segment of the tract, and as both these names are as unnecessary as they are cumbersome and confusing, we are quite in accord with Bruce and Muir's suggestion, that because it bears a relation to the posterior median septum, analogous to that borne by the "sulco-marginal tract" of Marie to the anterior median fissure or sulcus, it should be called "the descending septo-marginal tract."[1]

LITERATURE ON COMMA-SHAPED TRACT OF SCHULTZE AND OVAL CENTRE OF FLECHSIG.

Barbacci. "Contributo anatomico e sperimentale allo studio delle degenerazioni secondarie, &c." *Lo Sperimentale*, iii. and iv., 1891.

Bruce & Muir. "On a descending degeneration in the posterior columns in the lumbo-sacral region of the spinal cord," Brain, 1896.

Hoche. "Ueber Verlauf und Endigungsweise der Fasern des 'ovalen Hinterstrangsfeldes' im Lendenmarke." *Neurol. Centr.*, No. 4, 1896.

Gombault et Philippe. "Contribution à l'étude des lésions systématisées dans les cordon blancs de la moelle épinière." *Arch. de Méd. Expér.*, Tome vi.

Tooth. "The Goulstonian Lectures on Secondary Degenerations of the Spinal Cord." London, 1889.

Schultze. "Beiträge zur Lehre von der secundären Degenerationen im Rückenmarke des Menschen nebst Bemerkungen ueber die Anatomie des Tabes." *Arch. für Psychiatrie*, 1883.

Gombault et Philippe. "Contribution à l'étude des lésions systématisées dans les faisceaux blancs de la moelle épinière." *Arch. de Méd. Expér.*, No. 4, 1894.

Daxenberger. "Uber einen Fall von chronischer Compression des Halsmarkes." *Deutsche Zeitschr. für Nervenheilkunde*, 1893.

Kahler und Pick. *Archiv. für Psychiatrie*, 1880.

Silvester Blum. "Ueber absteigende secundäre Degeneration in den Hinterstränge des Rückenmarkes." *Inaug. Diss.*, Strasburg, 1895.

[1] As the "oval centre," or "medianen Abschnitt der medialen hinteren Wurzelzone," Flechsig described two small longitudinal bundles, running in the posterior columns of the lumbar enlargement, situated one on each side of the posterior median septum, neither reaching the posterior commissure nor touching the periphery of the cord. The shape of each individual bundle is, on transverse section, plano-convex, with the convexity directed outwards, and the two plane surfaces come into opposition at the median fissure, so that the two bundles when viewed together form a bi-convex or oval field, hence the name "oval centre." The integrity of this field in tabes is well recognised, and furthermore, if in a tabetic cord serial sections be made from the lumbar region to the conus medullaris, one can be assured of a reciprocal connection or continuity between this centre and the so-called "dorso-median sacral bundle" of Obersteiner.

Intermediary Bundle of Antero-lateral Column (Loewenthal).

This is a scattered tract, the major part of which occupies the mesial portion of the antero-lateral columns in front of the lateral pyramidal tract; but many aberrant fibres are found towards the periphery in the antero-lateral ascending tract of Gowers, and in the direct cerebellar tract and also in the adjoining crossed pyramidal tract. Loewenthal, in papers published in 1885 and 1886, founded on observations of the degenerations resulting from Schiff's experimental operations on the cerebral hemispheres of dogs, assigned to this tract the position named above, and named it the "Systême intermédiaire ou faisceau intermédiaire du cordon latéral." He regarded it as a special system of long fibres, and noticing that the fibres composing the tract did not degenerate after removal of the motor area, he argued that they could have no connection with the pyramidal tracts and the motor cortex, such as was claimed by Schiefferdecker and Singer, who noticed degeneration in the tract after sections of the dorsal cord in dogs previous to Loewenthal's description of it. The fibres of the tract are of larger calibre than those of the lateral pyramidal tract, they are clearly seen to degenerate after lesions of the medulla oblongata or spinal cord, they are most abundant in the cervical region, diminishing in number as they descend, and their course is long, as they have been traced into the upper lumbar segments.

Loewenthal was uncertain concerning the point of origin of these fibres, and though some recent papers clear the matter to some extent, there is at the present considerable doubt on this issue. Perhaps the most valuable paper dealing with the subject is from the pen of Boyce. This observer, in an extended series of observations on cats, found that after complete removal of one hemisphere, a bundle of degenerated fibres could be seen at the level of the anterior corpora quadrigemina, crossing in Forel's decussation in the raphe and proceeding outwards to occupy a position behind the inner border of the crusta and close to the mesial and dorsal aspect of the lemniscus on the side contralateral to the lesion. This bundle so formed could be traced downwards in the pons and medulla oblongata, always maintaining a lateral position, until ultimately it gained the lateral column of the spinal cord, along which it could be followed as far down as the lumbar region. The position the tract occupies in the lateral column corresponds closely with the area assigned by Loewenthal to the "systême intermédiaire," and Boyce names his

bundle of fibres the "lateral columnar tract." Boyce found that precisely similar degeneration of the tract followed division of the crus cerebri or hemisection of the spinal cord a little below the pyramidal decussation; but there was no degeneration in it subsequent to removal of the motor cortex. On summing up he states that the fibres "are probably the representatives in the medulla, pons, and mesencephalon of the large descending extra-pyramidal fibres found in the lateral columns of the cord, standing in the same relationship to those as do the fibres in the posterior longitudinal bundle and raphe to the anterior and antero-lateral columns. It is possible that in the cat and the dog this higher 'internuncial' or segmental system is more marked than in the monkey or in man, just as in the latter examples the pyramidal system is far more extensive than in the former."

It will be seen that Boyce, though he gives us an admirable description of the tract as it occurs in the cat, offers us nothing more than a clue to its exact origin; and Langley, in telling us that Bouchard in 1866 observed in man that the field of descending degeneration in the lateral column, which resulted from injury to the medulla oblongata, was more extensive than that after injury higher up, and that he located fibres which come from the medulla oblongata in the lateral border of the pyramidal tract, and named them the "fibres commissurales antérieures longues," helps us little, nor does he bring us any nearer the point when in agreement with Sherrington he informs us that the cortical pyramidal tract is of smaller dimensions than that figured by Flechsig, and proposes the name "descending medullary tract" for these extra-pyramidal fibres.

Degenerations consequent on Lesions of the Cerebellum.—Some experimental work completed by Marchi several years back, at first sight adds confusion to our knowledge of the composition and origin of the intermediary system of fibres, but as we now have ample proof that the writer's observations were erroneous, we may fortunately disregard his conclusions. Marchi found that if a hemi-extirpation of the cerebellum of the ape be effected, a secondary descending degeneration ensued in the spinal cord, which corresponded topographically almost precisely to the regional distribution of the "descending sulco-marginal bundle" and the "intermediary system of the lateral column" combined, and the degeneration extended down as far as the lumbar region. He naturally concluded that this system was a descending one, the trophic centre of which was in the cerebellum. Biedl, working in Stricker's laboratory in Vienna, in a paper founded on a

series of observations on cats, of which he had divided the corpus restiforme, writes in confirmation of Marchi's experiments, and so likewise does Thomas. Three English observers, however, Ferrier and Turner, who worked conjointly, and Risien Russell, are entirely opposed to these views originated by Marchi. Having performed many experiments similar to these initiated by Marchi, they are unanimous in denying the occurrence of descending degeneration in the spinal cord as a sequel to an experimental cerebellar lesion in the lower animals, and firmly express the opinion that the degeneration which Marchi obtained was in reality due to an unintentional wounding of Deiter's nucleus and other structures in the lateral region of the medulla, an accident which it would appear it is difficult, nay, almost impossible to avoid during the operation of cerebellar hemi-extirpation, owing to the proximity of the nucleus to the inferior cerebellar peduncle or restiform body, and an injury which is sufficient in itself to produce the exact spinal degeneration figured by Marchi.

This controversy, therefore, has not only been of value in disproving the existence of a direct descending cerebello-spinal tract, but has also led to a closer definition of the origin of Loewenthal's tract, for Risien Russell, who has studied the matter more laboriously than any other observer, in his last paper arrives at the following conclusions :—That the real source of origin of the descending antero-lateral tract, which degenerates in the spinal cord after a lesion of the lateral region of the medulla oblongata, is, as Ferrier and Turner agreed, Deiter's nucleus; that the degenerated fibres, which reach the anterior columns of the upper part of the spinal cord after a lesion of Deiter's nucleus through the posterior longitudinal bundles, are quite distinct from the above tract, and probably belong to some system of internuncial fibres similar to those traced by Boyce to the anterior columns by way of the posterior longitudinal bundles, after hemisection of the mesencephalon ; and lastly, that the direct descending tract of degenerated fibres met with in the spinal cord, in close relationship to the fibres of the crossed pyramidal tract, after a lesion of the lateral region of the medulla, is probably identical with the tract described by Boyce after hemisection of the mesencephalon.

An example of the perplexity occasioned by acceptance without confirmation of Marchi's observations is afforded by a recent paper of Bechterew's. This writer describes a system of fibres equally scattered throughout the lateral pyramidal tracts, which is to be observed in the spinal cord of a fœtus at a time when the ordinary pyramidal fibres are still unmyelinated, and which are untouched in an ordinary secondary descending de-

generation, and on the strength of Marchi and Biedl's observations, he states that they degenerate after idio-lateral cerebellar hemi-extirpation, but it is much more probable that they are aberrant fibres of Loewenthal's intermediary system.

LITERATURE ON INTERMEDIARY BUNDLE OF ANTERO-LATERAL COLUMN (LOEWENTHAL).

Loewenthal. "Dégénération secondaires de la moelle épinière consécutives aux lésions expérimentales médullaires et corticales." *Inaug. Dissertation*, 1885.

Loewenthal. "La région pyramidale de la capsule interne chez le chien et la constitution du cordon antero-lateral de la moelle." *Revue Méd. de la Suisse Romande*, 1886.

Boyce. *Loc. cit.*

Langley. "Recent observations on degeneration of nerve tracts in the spinal cord." Brain, 1886.

Marchi. "Origine e decorso dei peduncoli cerebellari." *Riv. Sper. di Freniatria e Med. leg.*, vol. xvii.

Marchi. "Des dégénérations consécutives à l'extirpation totale et partielle du cervelet." *Arch. ital. de Biologie*, Tome vii., 1886.

Marchi. "Sulle degenerazione consecutive alla estirpazione totale et parziale del cervelletto." *Riv. Sper. di Freniatria*, 1888.

Luciani e Marchi. *Il Cerveletto*, Firenze, 1891.

Biedl. "Absteigende Kleinhirnbahnen." *Neurol. Centr.*, No. 10, 1895.

Risien Russell. "The origin and destination of certain afferent and efferent tracts in the medulla oblongata." *Proc. of Royal Soc.*, London, vol. lxi., 1897.

Ferrier. "Recent work on the cerebellum and its relations, with remarks on the central connections and trophic influence of the fifth nerve." Brain, 1894.

Ferrier and Turner. *Proc. of Royal Soc.*, 1893.

v. Bechterew. "Ueber ein besonderes, intermediäres, in den Pyramidenseitenstrangbahnen befindliches Fasersystem." *Neurol. Centr.*, No. 21, 1895.

Thomas. *Le Cervelet.* Paris (Steinheil), 1897.

Worotynski. *Loc. cit.*

Descending Sulco-marginal Bundle (Marie).

"Système descendant de la zone sulco-marginale" is the name affixed by Marie to a scattered group of fibres, situated in the anterior columns along the margin of the anterior fissure, which undergo degeneration in a downward direction in consequence of a transverse spinal lesion. The length of these fibres

varies; some have been traced from the cervical to the lumbar segments. Though they possess a similar distribution, they are not, as Schiefferdecker thought, aberrant fibres of the direct pyramidal tract, for they do not degenerate after cerebral lesions, and are further to be found in the lower segments of the cord, where the direct pyramids are not represented (Flechsig, Singer). In the dog Singer and Münzer place the fibres of this tract in a small group at the anterior angle of the cord, opposite the mouth of the anterior spinal fissure. In the cat it occupies a similar position, and Schäfer has shown that in that animal this bundle is one of the earliest to undergo secondary degeneration, its fibres exhibiting the characteristic reaction with osmic acid on the fourth day after a spinal hemisection.

Marie thinks it probable that these fibres appertain to the system of longitudinal or internuncial fibres, the existence of which Ramon y Cajal and Golgi have so beautifully demonstrated, and that, originating from an upper cell of the anterior cornu, they cross by the anterior commissure, and course downwards to connect that cell with one situated in the opposite anterior cornu of a lower segment of the cord. We fear that there is insufficient anatomical evidence for the existence of the anterior commissural decussation which Marie alludes to, and since destruction of Deiter's nucleus gives rise to descending degeneration of fibres situated in the sulco-marginal zone, indistinguishable from Marie's fibres (*vide* last paragraph), we would rather assume that the trophic centre for this system of neurons is situated in that nucleus or thereabouts. In man the bundle demands further investigation.

Postero-external Column or Column of Burdach, and Postero-internal Column or Column of Goll.

Since the fibres which compose these columns are derived from a like source, and since Goll's column is really made up of overflow fibres from Burdach's column, it is deemed advisable, as well as convenient, to consider the two together.

Within the past few years these columns have been exhaustively studied by a host of competent observers, some describing the changes in them consequent on division of the posterior nerve roots or spinal hemisections in lower animals (Tooth, Horsley, Mott, Sherrington, Singer and Münzer, Barbacci), others reporting the degenerations attendant upon traumatic lesions or tumours,

&c., of the cord or posterior nerve roots (Schiefferdecker, Gombault and Philippe, Déjérine), and others studying the alterations occurring in them as a result of various diseases (Mayer, Redlich, Obersteiner). In short, our knowledge of these two great centripetal tracts has been increased by leaps and bounds, and at the present time is practically complete, with the exception that the ultimate distribution of the tracts after the fibres quit the nuclei of Goll and Burdach still requires further elucidation.

Development.—Flechsig's development researches show that the fibres of the column of Goll become myelinated at the end of the sixth month of fœtal life, and thus are among the earliest fibres to assume the medullary envelope; and Schäfer in his experiments on cats found that the fibres of the column of Goll degenerated on the fourth day after a hemisection of the cord, and was one of the first tracts to yield evidence of the secondary changes. There is, therefore, a parallel between the time of accession of the secondary degeneration and the period of development of the medullated sheath in this column.

Origin and Destination.—From the degenerations observed after section of the posterior nerve roots, and from diseased conditions affecting these roots, numerous writers have demonstrated that the fibres composing these columns represent the spinal continuations of these posterior roots—in short, they are exclusively exogenous fibres. Although a side issue, it should here be indicated that in certain of the lower animals, such as the frog, chick, &c., a certain number of fibres having an intra-spinal origin issue in the posterior root, and hence do not degenerate in consequence of a root section (v. Lenhossek, Ramon y Cajal, Horton Smith), and the question has arisen whether analogous fibres do not exist in mammals and the human being. But in the case of the cat and the monkey Sherrington, v. Lenhossek and Ramon y Cajal's observations definitely negative this idea, and therefore the original law of Bell, which states that the posterior roots exclusively consist of fibres of extra-spinal origin destined for the conduction of sensory impressions, certainly holds in the case of some mammals, and probably also in the human being.

The fibres of the posterior roots enter the cord in that region which is situated behind the substantia gelatinosa Rolandi, and the most laterally situated fibres of this portion form Lissauer's so-called rootzone. It has been noticed by Obersteiner that just at the point of entry into the cord each fibre dispenses with its medullated sheath, but only for a very brief distance. From personal observations I am convinced of the correctness of this statement, and may mention that this gap in the medulla of the

fibres may be most clearly seen if an acutely degenerated posterior root be stained by the method of Marchi.

Having gained their entry into the spinal cord, the fibres divide into ascending and descending branches, each of which issue collaterals in their course. The descending branches pass downwards for a short distance, and then turn at right angles into the grey matter at the top of the posterior cornu, where they end in arborisations round nerve cells. The ascending branches have varying destinations, and either by means of their terminal branches or their collaterals are said to come into relation with a number of different groups of nerve cells; certainly many fibres go to (*a*) cells in the middle segment of the posterior cornu; others seem to be destined for (*b*) the motor anterior cornual cells, and form the reflex collaterals of Kölliker or postero-anterior collaterals of Ramon y Cajal; others proceed to (*c*) the cells of Clarke's column, and are seen atrophied in tabes dorsalis; a small number of collaterals cross in the grey comissure and go to (*d*) cells in the posterior cornu of the opposite side; and, lastly, the long fibres of the columns reach (*e*) the cells of the nuclei of Goll and Burdach. The researches of Ramon y Cajal, v. Lenhossek, Golgi and Kölliker seem to prove that these fibres, when they approximate these various groups of nerve cells, break up into arborisations which surround the bodies of the cells without visibly coming into contact with them or penetrating their interior; so that if a recently degenerated nerve fibre be followed up to one of the nerve cells, delicate methods may display a disappearance of the investing arborisation, while the cell body and its processes remain intact. But that this cell body or "Endkerne," as His calls it, so shorn of its investing diminutive nerve plexus, undergoes degeneration and disintegration at a later stage is almost certain.

In addition to the above-mentioned destinations of the posterior root fibres, others are supposed by Loewenthal, Oddi and Rossi and Berdez, on the strength of experimental researches, to pass by way of the anterior commissure either into the opposite posterior column or to enter one of the long ascending tracts of the anterior or lateral columns, but the existence of such a decussation is denied by more recent observers. Fajersztajn asserts that the posterior roots have no connection whatever with the anterior commissure, and bases his statement on convincing deductions drawn from experiments on animals; these were the isolation of lengths of the spinal cord by two complete transverse sections made at distances of 6-14 centimetres from one another, and in these cases he found the anterior commissure perfectly

free from degeneration in the neighbourhood of the lower hemisection, where all ascending fibres must necessarily be degenerated. Souques and Marinesco and Mott share this opinion, and the latter mentions the supposition that if degeneration occurs in the crossed afferent tracts of Gowers and Edinger (*vide* antero-lateral ascending tract) it might be attributed, not to the section of the posterior roots, but to unavoidable vascular injuries incident to such an operation affecting nerve cells in the grey matter of the posterior horn, from which it is believed the fibres of the crossed afferent tract of Edinger originates. These latter fibres, it must be noted, are supposed to cross in the anterior commissure.

Lastly, certain physiological experiments point to the probability of a decussation of fibres, either by way of the grey commissure, or directly through the posterior median septum to the opposite side, but no anatomical basis exists for this supposition.

Topography.—Having now indicated the latest views concerning the destinations of the posterior root-fibres, it will next be necessary to refer in some detail to the topographical distribution and arrangement of the individual groups of posterior root-fibres, commencing with those derived from the cauda equina.

Excellent cases, illustrating the course of the degeneration which follows destruction of the cauda equina in the human being, have been recorded by Souques and Marinesco, Darkschewitch, and Déjérine and Sottas; and the following descriptive *résumé* of Souques and Marinesco's case applies more or less to all, and may be accepted as typical. In this instance the cauda equina and conus medullaris, and the three lower sacral segments, were destroyed by the pressure of a group of hydatid cysts. The middle sacral segments exhibited the changes of traumatic degeneration (Schiefferdecker); in the upper sacral segments the entire posterior columns, with the exception of the centrum ovale (Flechsig), the cornu-commissural zone (Marie), and some few fibres coursing along the inner margin of the posterior horns, were degenerated; likewise the delicate myelinic plexus, situate in the grey matter of the posterior and anterior cornua, was at this level markedly atrophied. In the mid-lumbar region similar changes were visible. In the lower dorsal region the degenerated area was separated from the posterior cornua by a definite intervening zone of healthy fibres, and the myelinic plexuses in the anterior cornua were healthy. In the mid-dorsal region the plexus of nerve fibres in the posterior cornua regained a healthy appearance, and the patch of degeneration assumed the form of a funnel or wedge, the point of which reached the posterior commissure, while the base lay

along the periphery. In the upper dorsal region the degeneration became limited to the postero-internal columns, and retreated from the commissure. In the cervical region it affected only the posterior half of the postero-internal columns, and in the medulla degenerated fibres were traceable to the inner side of the nucleus of the funiculus gracilis. Lastly, it should be added that the total volume of the degenerated fibres gradually diminished as the diseased field ascended.

Tooth obtained degeneration resembling this in its distribution, after division of the posterior roots of the cauda equina in the monkey; and Mott, after section of the lumbo-sacral roots in the monkey and cat, got confirmatory results.

The experiments performed by Tooth of division of all the posterior nerve roots between the third dorsal and the third lumbar segments, inclusive, produced like results. The patch of degenerated fibres was situated immediately external in position to that above described, and likewise gradually spread inwards, until in the upper dorsal region it was almost confined to the postero-internal column; then, as it extended along the cervical region, it gradually retired from the posterior commissure, while maintaining its peripheral base. The degenerated fibres terminated mainly in the outer part of the nucleus of the funiculus gracilis.

In the case of the first and second dorsal nerve roots and the two lower cervical roots, some of their fibres pass over to the postero-internal columns, but a portion do not leave the column of Burdach, and in regard to the remaining upper cervical nerve roots, their fibres canton for their entire course in the postero-external column, gradually taking up a more internal position as they ascend, and ultimately gaining the nucleus of the funiculus cuneatus.

A case narrated by Gombault and Philippe, in which there was complete destruction of the fifth right posterior cervical root, and partial destruction of the fourth posterior cervical root on the same side, beautifully illustrates the degeneration as it occurs in the cervical region in the human being. Immediately below the fourth cervical roots, the patch of degeneration is triangular in shape, approximated to the posterior part of the cornu, and separated from the periphery by a thin band of healthy fibres. At the level of the first cervical roots it is seen as a strip, extending from the periphery to the posterior commissure along the inner margin of the postero-internal column. It occurs as a similar strip in the funiculus cuneatus at the first cervical level, and eventually becomes localised in the cuneate nucleus.

From these accounts it may readily be gathered that the long fibres derived from the posterior roots possess one remarkable peculiarity in regard to the course which they pursue, viz., that as they ascend they are gradually being pushed into a more internal position, and having the position which they have just vacated occupied by fibres which enter from the root immediately above. In this manner it comes about that those fibres which occupy the external division of the posterior field in the lower segments of the cord are found in the internal division in the upper segments. This assumption of an eccentric position by the long fibres of the posterior roots is one which is common to a number of other systems, and has led Flatau to formulate the law "that the short fibres of the cord run in close position to the grey matter, while the long fibres select a position next the periphery."

Numerous other reports of the degenerations which have ensued after various forms of transverse lesions in different parts of the spinal cord, and many other experimental researches, might be cited in illustration of the topographical distribution of the posterior root fibres. But this is hardly necessary, and it may be taken for granted that the sum total of these observations is (1) that the fibres which constitute the postero-internal column or column of Goll and eventually enter the nucleus funiculus gracilis, are derived from the posterior roots of the sacral and lumbar segments and of the dorsal segments, with the exception of the first and second, and that all the fibres which enter into the composition of this tract have in one part of their course cantonned in the column of Burdach; (2) that those fibres which constitute the postero-external column or column of Burdach, and terminate in the nucleus funiculus cuneatus, are derived from the posterior roots of the cervical segments, and partly from the two upper dorsal posterior nerve roots.

Distribution of the fibres which emanate from the nuclei of the funiculus gracilis and funiculus cuneatus.

As already stated, considerable doubt exists concerning the ultimate distribution of the fibres issuing from Goll's and Burdach's nuclei, or the fibres of the second sensory neuron as they might be called; the reason, of course, being that lesions which will cause an interruption of these secondary neurons alone, without involving other tracts, are rarely met with. However, the accepted view is, that these fibres are destined for the cortex of the cerebrum and cerebellum respectively, that they are

conveyed to the former in the fillet or lemniscus, to the latter by the corpus restiforme or inferior cerebellar peduncle.

In the first place, in regard to the fillet, articles sufficient to form a considerable volume have been published bearing on its course and connections, but it will be impossible in this review to do more than outline some of the more important observations on the tract.

To begin with, some experimental anatomical papers dealing with the course of these fibres are of interest. Mott, after unilateral separation of the nuclei of the posterior columns from the arciform fibres issuing therefrom in the monkey, obtained degeneration of these fibres and of the opposite inter-olivary layer. In the upper part of the pons varolii the degenerated fibres occupied the median and lateral fillet; some fibres of the lateral fillet apparently went to the corpora quadrigemina, but the majority of the remaining fibres could be traced onwards to the optic thalamus, where they terminated. Evidence of a direct continuity of these degenerated fibres by a "cortical fillet" with the cortex cerebri was not obtainable.

Likewise Singer and Münzer, by destroying the cuneate nuclei, obtained atrophy of the internal arciform fibres of the same side and of the fillet of the opposite side, which was traceable to the optic thalamus.

von Vejas also years ago discovered that destruction of the posterior column nuclei of new-born animals induced atrophy of the same part of the fillet as far as the corpus trapezoides.

Schlesinger, in a most thorough paper on degenerations of the lemniscus well worthy of consultation by anyone specially interested in this tract, traces some fibres of the posterior column nuclei to the anterior corpora quadrigemina and the majority to the optic thalamus. All Schlesinger's observations were made on the human being.

From the foregoing it might be assumed that these fillet fibres issuing from the nuclei of Goll and Burdach's column degenerate only in an ascending direction, but this is not so, for there is an opposite array of evidence to show that they may degenerate or atrophy in a descending direction. Jakob, Mahaim and Bruce have reported such a descending degeneration in cases of central cerebral lesions, and the writer, in a case of tubercular softening of the optic thalamus, examined by the method of Marchi, saw descending degeneration in this tract.

Therefore, though confusing, it seems, nevertheless, to be correct to assume that the fibres of the fillet may degenerate either upwards in consequence of an interruptive lesion near the medul-

lary nuclei, or downwards in consequence of a lesion of the great nuclei at the base of the brain.

Now, since an interruptive lesion of the fillet gives rise to a degeneration which cannot be traced higher than the optic thalamus, the question is, how are sensory impulses conveyed further to the cortex cerebri? And the supposition is that between the thalami and the cortex a third neuron is trajected, which has been named by some the "cortical fillet," and which may under certain conditions undergo degeneration. For instance, Monakow, by destroying the parietal lobe in young animals, produced atrophy of the fillet of the same side and of the internal arciform fibres and nucleus gracilis. Flechsig and Hosel saw the same changes in a case of porencephaly involving the central convolutions; similar cases to this have been recorded, and in two cases of cerebral lesions above the level of the basal nuclei, I have noted corresponding atrophy of the fillet.

We may then sum up by saying that the fibres issuing from the nuclei of Goll and Burdach's columns, which are destined for the cortex cerebri, ascend in the lemniscus or fillet. These fibres decussate in the medulla, continue along the pons and crus cerebri in the mesial and lateral lemniscus, and after giving off collaterals to various nuclear structures, amongst others the anterior corpora quadrigemina and the corpora Luysii, they eventually reach the optic thalamus. At this level it is supposed a third system of sensory neurons begins, the axons of which are first grouped in that part of the posterior limb of the internal capsule which Charcot has named the "carrefour sensitif," and thence stream into the corona radiata to terminate in the cortex of the parieto-occipital and temporal convolutions. But more information on this system of neurons is needed.

Secondly, one must briefly refer to the connection between fibres issuing from the posterior column nuclei and the opposite lobe of the cerebellum. According to Monakow the fibres issuing from the cells of the outer or lateral part of the nucleus of Burdach's column are specially destined for the cerebellum; these fibres are joined by others from the nucleus gracilis, and are directed to the restiform body by the internal arciform fibres, and hence reach the cerebellum, but what part thereof is not exactly known.

According to Bechterew the nucleus gracilis has another crossed connection with the cerebellum; this writer states that some few of the fillet fibres at the decussation of that structure, instead of continuing upwards, pass forwards through the opposite anterior pyramid, and reaching its ventral surface, curve round

the periphery of the medulla oblongata as external arcuate fibres to the corpus restiforme, and thence reach the cerebellum.

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LISSAUER'S ROOTZONE, OR COLUMN.

As Lissauer's rootzone, Waldeyer's "Markbrücke," or Flechsig's "laterale hintere Wurzelzone," is recognised that part of the cord which on transverse section lies immediately dorsal and lateral to the apex of the posterior cornu. All that we know about the area is that it contains fibres of small calibre, supposed to be derived from the posterior roots, and that they proceed in through the substantia gelatinosa to cells of the posterior cornua. Lesions which cause ascending degeneration in the posterior columns also

give rise to degeneration of Lissauer's column, and it is invariably sclerosed in tabes dorsalis. Bechterew has indicated that the fibres of this column are peculiar in becoming myelinated at a later period than any other of the posterior root fibres.

Direct Cerebellar Tract.

The name given to this important afferent tract by Flechsig, its original describer, was "the direct lateral cerebellar tract," but it is now occasionally called the "dorso-lateral, or postero-lateral, ascending cerebellar tract," in contradistinction to the "ventro-lateral or antero-lateral ascending cerebellar tract," *i.e.*, the antero-lateral ascending tract of Gowers.

Development.—The fibres of the tract acquire their medullated sheath not later than the beginning of the sixth month of fœtal life (Bechterew), which is considerably prior to the period of medullation of the fibres of the antero-lateral ascending tract.

Origin.—In regard to the origin and constitution of this tract, we are, as Marie says, even at the present time, in a condition of absolute uncertainty. The two main views in reference to its origin are (1) that the fibres are derived from the cells of the vesicular column of Clarke, a view championed by Mott; (2) that they are derived from the posterior roots, a mode of origin credited by Tooth.

Mott describes how "the cells of the column of Clarke give off large fibres which run upwards, slightly forwards, and then outwards through the lateral column, to reach the direct cerebellar tract of the same side." And while admitting that on account of the obliquity of the course followed by these fibres between their starting-point in the vesicular column and their cantonment in the direct cerebellar tract, it is impossible to cut a section which will include a view of the fibre in the whole of this part of its course, yet he adduces a quantity of evidence—histological, developmental, and experimental—in favour of his claim. Perhaps the most important experiment in support of the view was that of unilateral section of Clarke's column, effected in a monkey at the level of the twelfth dorsal and first lumbar segments, which resulted in ascending degeneration of the direct cerebellar tract on the side of the lesion. Sherrington acquiesces in Mott's views, and they are further supported by Flechsig's developmental researches.

Touching the second theory of origin, Tooth agrees with Mott in thinking that the fibres of the tract do not come by way of the

posterior roots for the lower limbs, but he found that if in monkeys he divided the 6th, 7th, and 8th cervical and the 1st dorsal posterior nerve roots, a complete and absolute degeneration—not of the whole direct cerebellar tract, but of that part of it which lies next the posterior horn—occurs. Like results followed section of the 2nd, 3rd, and 4th dorsal posterior roots. He therefore concludes that certainly in the cervical region, and probably also in the upper dorsal region, fibres of the posterior roots enter largely into the composition of the tract. It is to be further gathered from Tooth's work that he regards the direct cerebellar tract as being entirely made up of fibres of large calibre (Bechterew supports this statement), and that the fibres of small calibre seen in the position of the tract in the lower dorsal region, are descending ones, probably pyramidal fibres, as the pyramidal tract comes to the surface at this level, and that the fibres which Mott and others have seen obliquely crossing the lateral column at this level are fibres of this nature. Tooth's conclusions are rendered insecure by the fact that Singer, Kahler, and others, in their experiments of section of the posterior spinal roots of the dog, did not obtain degeneration of the direct cerebellar tract.

We should also like to ask why it is the exception and not the rule to find sclerosis of the direct cerebellar tracts in cases of tabes dorsalis, in which disease the posterior roots are completely withered up by sclerotic processes.

Unfortunately the recently recorded observations of two Italian experimental neurologists, Pellizzi and Paladino, are not reconcilable to either of the above-mentioned views, and indeed are altogether extraordinary as well as out of accord with the observations of other better known observers, that is, provided the anatomical distribution of the fibres of the spinal cord be similar in the dog to what it is in the monkey, as we may presume it is.

Pellizzi, after unilateral division of the posterior roots of the last lumbar segment and of the first posterior sacral root, and likewise after a hemisection of the spinal cord at this level, obtained, along with other changes, symmetrical ascending degeneration of both direct cerebellar tracts. A like result followed unilaterial section of the four lower posterior cervical roots. He surmises that the direct cerebellar tract receives its fibres partly from the column of Clarke of the same side, partly from the posterior roots of the same side direct, and partly from the posterior roots of the opposite side, the fibres of the latter decussating in the anterior commissure.

Paladino, after unilateral section of the sixth posterior roots of the lumbo-sacral plexus, also obtained bilateral ascending degeneration of the direct cerebellar tracts, and a similar result after unilateral division of the four lower dorsal posterior roots.

Both workers stained their sections by the method of Marchi. Results such as these render our confusion concerning the origin and composition of this tract more profound, but judging from the work of previous experimenters and a quantity of other evidence, we can hardly seriously accept Pellizzi and Paladino's observations as correct, that is to say, as far as the point under consideration, viz., the source of these fibres, is concerned. A difference between the constitution of the cord of the dog and that of the cat or monkey is too unlikely to account for these results, and to my mind they are altogether insufficient to controvert the records of careful experimenters, like Mott and others, who, severing the same roots and employing the same staining method, failed to obtain any degeneration whatever in either direct cerebellar tract, furthermore they are out of harmony with the changes observed in the human cord following lesions of the posterior roots of the lumbo-sacral plexus.

While offering all due respect to Pellizzi and Paladino for their work (Pellizzi's paper is an especially complete one), we therefore feel forced to disregard their conclusions, and hazard the opinion that the anomalous degenerations which their experiments brought forth are to be attributed to some operative mishap, possibly some unnoticed vascular injury, which it is difficult to avoid in these procedures, and which is apt to render any experimental work unreliable.

After sifting all the evidence concerning the origin of the direct cerebellar tract, we are inclined to abide by Mott's decision that it is composed of the neuraxons of the bipolar cells of the vesicular column of Clarke.

Topography.—The level at which this tract commences as a bundle varies with different authors. Tooth concludes from his researches that it begins to show itself somewhere between the 9th and 11th dorsal nerve roots. Mott, and likewise Kahler and Pick, fix it at the 9th dorsal pair, Schultze at the 10th, and more recently Barbacci has observed degeneration in it as low as the point of origin of the 11th and 12th dorsal pair of nerve roots. It is probable that the site at which it appears will vary in different individuals in the same way as the downward distribution of the columns of Clarke is inconstant.

Since it is an established fact that Clarke's column commences to appear at the 2nd or 3rd lumbar segment, and since the direct

cerebellar tract is said to be derived from these cells, the question may be asked: Why is it that after a lesion in the lumbar region where Clarke's column is already formed, degeneration in the direct cerebellar tract does not present itself until the level of origin of the 11th or 12th dorsal nerve roots is reached? This apparent incongruity is explained, as Barbacci and Mott have demonstrated, by the great obliquity of traject of the fibres as they course from Clarke's column to their eventual seat in the tract. This obliquity is such that though Clarke's column may be represented as low down as the 2nd lumbar segment, the axis cylinders of the nerve cells of the columns do not become grouped in their position in the direct cerebellar tract until a point two or three segments higher up is reached.

In form the direct cerebellar tract has been compared to the segment of a ring, occupying about one-sixth of the spinal circumference. Its posterior extremity is thickened and comes into contact with the tip of the posterior cornu. Its anterior extremity is attenuated and at the same time not sharply defined, its fibres mingling with fibres of the adjoining antero-lateral ascending tract. Throughout the whole extent of its course in the cord the tract more or less maintains this shape and position, but at about the level of the 3rd cervical roots it becomes separated from the antero-lateral ascending tract by the interposition of a collection of heterogeneous fibres.

Higher Distribution and Destination.—As the tip of the posterior cornu quits the periphery in the lower part of the medulla the direct cerebellar tract takes its place; it is thrust further back, a little higher up, by the so-called ascending root of the trigeminal nerve, which inserts itself in front of it. Higher still the tract proceeds, first externally to the ascending 5th root, and then comes to lie anteriorly to it. At the level of origin of the 8th nerves the fibres pass obliquely into the corpus restiforme. Thence Loewenthal, in experiments on dogs, traced it to the dentate nucleus of the cerebellum, and inferred that it ultimately terminated in the superior vermiform process. Tooth believes that it is only after section of the posterior roots of the upper segments of the cord that a simple uncomplicated degeneration of the direct cerebellar tract can be satisfactorily traced upwards, because in hemisections of the cord there is a confusing admixture of degeneration of the antero-lateral elements. Bechterew, however, in developmental observations on the fœtus from 25-27 centimetres long, confirms the statement that the direct cerebellar tract passes to the superior

vermis, and Monakow and Mott arrive at the same conclusion experimentally.

Retrograde Sclerosis or Degeneration in the Direct Cerebellar Tract.—In 1894 I reported a case of old standing thrombosis of the posterior inferior cerebellar artery which had left a deep and extensive excavation, involving the parts supplied by that artery. Microscopic examination of the cord in this instance revealed profound sclerosis of the direct cerebellar tract occurring throughout the whole extent of this tract, and this was accompanied by atrophy of the cells of Clarke's column, all on the side of the lesion. Now, the only rational conclusion is that the case was one of retrograde sclerosis in consequence of an obliteration of the terminus to which the fibres of the direct cerebellar tract run. More recent publications show that such a retrograde sclerosis may occur in other tracts and so confirm the view.

Pellizzi, after a lesion at the lower dorsal level, and after unilateral section of the four lower posterior cervical roots, obtained slight descending degeneration in the direct cerebellar tract on the side of the lesion. A result, so far as one can gather, never obtained by other experimenters. He explained the degeneration by supposing that the degenerated fibres are descending branches of the posterior roots.

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Antero-lateral Ascending Tract, Ventro-lateral Ascending Cerebellar Tract, or Tract of Gowers.

This is another important tract which degenerates in an ascending direction in consequence of a transverse spinal lesion, and which has received a lot of attention from those workers who have

studied the direct cerebellar tract, with which it was formerly confused. As will be presently indicated, the cerebellum seems to be its destination, and on that account some writers *e.g.*, Mott, Loewenthal, Schäfer, prefer to name it the "ventro-lateral or antero-lateral ascending cerebellar tract." By Continental writers it is generally called after its original discoverer, "Gowers' Tract."

Origin.—As was the case with the "direct cerebellar tract," so is it with the antero-lateral ascending tract, the exact source of the fibres which compose it is uncertain—perhaps to a greater degree—and the views concerning its origin, whether formulated by experimental or purely anatomical workers, are mainly based on conjecture. That the fibres which constitute the tract enter the spinal cord by the posterior roots would appear to be in a measure negatived by the experiments of division of these roots practised by Mott, Tooth and many others; but Pellizzi, whose extraordinary results have already been referred to in the section on the direct cerebellar tract, obtained symmetrical ascending degeneration in both antero-lateral ascending columns after unilateral section of the last lumbar and the first sacral posterior roots of the dog; he also saw slight degeneration of the same tract, but more marked on the side of the lesion after unilateral section of the four lower posterior cervical nerve roots, and naturally concludes from these experiments that each antero-lateral ascending tract receives some of its fibres from both posterior roots, the anterior commissure serving as the bridge of decussation.

Likewise Loewenthal, Berdez and Langley and Anderson, after similar operations on various animals, obtained degeneration either in the anterior commissure or in both antero-lateral ascending tracts, but always more marked in the latter on the side of the lesion.

Also Mott, in a recent paper, states that in one monkey, of which he had divided the fourth, fifth, sixth, seventh, eighth and ninth sub-thoracic roots on one side, he found some thirty degenerated fibres in the opposite antero-lateral ascending tract, but he firmly expresses the belief that this degeneration was set up by vascular injuries to the grey matter of the posterior horn, accidentally induced by the operation, and in effect totally denies the passage of fibres from the posterior roots directly across the cord to the opposite antero-lateral ascending tract. Mott, in the same paper, further mentions that Loewenthal and Berdez's experiments are on similar grounds not to be relied on, for they certainly did not exclude injury of the tip of the posterior horn and contiguous parts, nor did they take into account the effect of such

injuries. For a like reason Langley and Anderson's experiments cannot be accepted, and it is more than probable that Pellizzi's experiments must be condemned on account of the same flaw.

Unfortunately, we can hardly compare the observations of Kahler, Singer and Münzer and Tooth, or any other experimenter who worked before the method of Marchi was introduced, with the observations of the more recent writers quoted above, all of whom worked with this method, for the simple reason that the method of Weigert and its modifications, which a few years back was the fundamental staining *technique*, fails to reveal degenerated fibres which are disseminated or present only in small groups, whereas the method of Marchi will plainly discover such degeneration.

To support the assumption that no fibres enter the antero-lateral ascending tract directly from the posterior roots, we are therefore practically left with Mott's view only, but this view we prefer to accept until an explanation is forthcoming, showing why it is that in certain cases in which the operation of division of the posterior roots is carefully performed, and the sections of the spinal cord stained by the most recent and approved methods and closely examined, no degeneration whatever is discoverable in either ascending antero-lateral tract. It should also be mentioned that Mott's view is strengthened by the fact that in several cases of injury to the cauda equina in the human being, in which the cord was stained by the method of Marchi, no degeneration whatever was found in either ascending antero-lateral tract (Darkschewitch, Souques and Marinesco, Déjérine and Spiller).

What, then, is the source of these fibres? Tooth, whose opinion deserves consideration in this connection, suggests "that the antero-lateral fibres arise solely from the grey matter, that its fine fibres, which are very numerous in the lower cord, are centripetal from the posterior vesicular column, and that the large fibres are in connection with one or more groups in the anterior horns," but this suggestion, he admits, is quite hypothetical. Hadden and Sherrington's case of tabes, in which the antero-lateral ascending tract was sclerosed, as well as the posterior column, which Tooth quotes in favour of the view that the columns of Clarke give origin to fibres of the antero-lateral ascending tract, does not appear to us to be of much importance, because, though atrophy of the columns of Clarke is almost constant in locomotor ataxia, sclerosis of the lateral columns is rare, and when it does occur involves the direct cerebellar tract as often as, if not more frequently than, the antero-lateral ascending tract. In specimens in which we have seen such changes, we

have attributed them to peripheral vascular alterations concurrent with syphilis and tabes (an idea suggested by Déjérine years ago), rather than to a primary atrophy of Clarke's column.

As far as we can gather, Tooth and Testut appear to be the only authors who maintain that the antero-lateral ascending tract takes any fibres from the columns of Clarke, but there is more or less unanimity of opinion that cells in other parts of the grey matter, particularly at the base of the anterior horn, give origin to it (Bechterew, Mott, Flatau, Gombault and Philippe). Some experiments of Mott's are of considerable importance in this connection; he practised median section of the spinal cord in the lumbar region in two monkeys. In the first experiment the lesion involved the lower end of Clarke's column on one side, and the result was symmetrical ascending degeneration of both ascending antero-lateral tracts, and degeneration of the direct cerebellar tract on the side of the lesion. In the second experiment the incision was successfully made in the middle line below the position of Clarke's column, and while precisely similar bilateral degeneration occurred in the antero-lateral ascending tracts, there was practically none in the direct cerebellar tract. Similar results were obtained by Grünbaum after longitudinal division of the cord. Likewise Ehrlich and Brieger, and Singer and Münzer, finding that compression of the abdominal aorta in rabbits causes destruction of the nerve-cells in the grey matter of the lumbar cord, and also ascending degeneration of the antero-lateral ascending tract, infer therefrom that this tract arises from cells in the grey matter of this region.

These experiments, therefore, in a measure, prove that the fibres of this tract do not originate from the nerve cells of Clarke's columns, but from cells elsewhere in the grey matter whose exact situation is unknown, but supposed to be at the base of the anterior horn, possibly the middle cell column of Waldeyer. The occurrence of bilateral degeneration of the tracts in question after a mesial longitudinal spinal section, warrants the assumption that these cells give off fibres which decussate in the anterior commissure, and so reach the lateral tract, and the existence of such a decussation is also supported by Auerbach's experiment of destruction of a considerable length of one-half of the spinal cord in the cat, which resulted in ascending degeneration of the tract on the side opposite to the lesion.

Crossed Afferent Tract of Edinger.—Some other fibres ascending in the position of the antero-lateral ascending tract must not be confused with those which I have just described; these are afferent fibres which Edinger and Gowers, and likewise Mott,

believe enter from the posterior roots to come into connection with cells of the grey matter, which cells give off axis cylinder processes, also decussating in the anterior commissure, and also ascending in the region of the antero-lateral ascending tract; coursing along the medulla oblongata and pons in or near the fillet, these fibres terminate in the corpora quadrigemina or optic thalami. This tract so formed is called the "crossed afferent tract of Edinger," and differs from the antero-lateral ascending tract probably in its origin, and certainly in its destination. From what Mott says, it is probable that it was degeneration of this tract, and not of the antero-lateral ascending tract, which Loewenthal, Berdez, Langley and Anderson and Pellizzi saw in their posterior root experiments; also that that degeneration occurred not in consequence of the root section, but subsequent to accidental injury of the most dorsal segment of the posterior cornu, and therefore that the seat of origin of these fibres is in that segment of the posterior cornua.

Topography and Termination of Antero-Lateral Ascending Tract.—Having now pointed out the different views concerning the origin of this tract, I will next proceed to deal with its spinal topographical distribution. Firstly, as to the lowest level in the spinal cord at which it has been found. This level is considerably below that at which the direct cerebellar tract first shows itself; as I previously mentioned, degeneration in that tract has not been definitely seen below the 10th dorsal segment, but there are several cases on record of lesions lower than this which caused degeneration of the antero-lateral ascending tract and left the direct cerebellar tract intact; such, for instance, as Schultze's case, in which there was crushing of the lumbar region. Furthermore, Bechterew mentions the existence of the tract even in the lower parts of the lumbar enlargement. If the lesion be below this level, that is to say, if it affect the sacral segments or cauda equina, no degeneration of the tract occurs; this fact is proved by several reported cases of such lesions. In other words, therefore, there is a short stretch of the spinal cord comprising these segments, situated between the 10th dorsal and the last lumbar segment, a lesion of which will cause ascending degeneration of the ascending antero-lateral tract, leaving the direct cerebellar tract intact. And it was from observation of such a case (one of crushing at the 11th dorsal level), that Gowers was enabled to describe the antero-lateral ascending tract as one quite distinct from the direct cerebellar tract, two tracts which had been formerly regarded as one. Bechterew independently confirmed this distinction by adducing developmental proof that

the fibres of this tract become myelinated at the eighth month of fœtal life, that is, later than the fibres of the "direct cerebellar tract."

The tract once formed takes up, according to Tooth, the following position: "it lies in the anterior part of the lateral column, in continuity in many cases with the direct cerebellar tract, behind and close to the anterior rootzone in front. It is not a thin strip like the direct cerebellar tract, but a wedge-shaped area, with its base at the periphery of the section, and its apex towards the grey matter and sometimes bent posteriorly, so as to insinuate itself between the crossed pyramidal and the direct cerebellar tracts. It is situated in an area of the cord called by Flechsig the "mixed zone." This position it more or less maintains throughout the cord.

Until some experiments, performed by Mott with a view to determining the destination of the antero-lateral ascending tracts were published, in 1892, great doubt existed concerning the upper course of this tract, though many, following Bechterew's lead, accepted the nucleus lateralis in the medulla oblongata as its terminus. Mott, by a novel experiment on the monkey, that of section of the fibres of the tract in the upper cervical region, by a specially devised instrument, and by the employment of the method of Marchi, was enabled to trace with the utmost accuracy the line of degeneration marking out the bulbar course of the tract. Tooth, in a subsequent paper, amply confirmed Mott's observations. Schäfer and Loewenthal independently arrived at similar results.

The following are the main anatomical points noted by these observers:—At the level of the nucleus lateralis the tract is seen at the periphery external to that nucleus and to the "so-called" ascending root of the fifth nerve. Dorsally, the tract touches the direct cerebellar tract; also many degenerated fibres are visible in the nucleus lateralis itself, thus a connection between the tract and that nucleus is proved, but it does not end there. A short distance higher up the antero-lateral ascending and direct cerebellar tracts separate from one another, and the divergence becomes wider as one ascends. At the level of the olive the ascending antero-lateral tract lies among the external arciform fibres dorsal to that nucleus. In a transverse section catching the sixth and seventh nerves in their intra-medullary course, the tract is still seen at the periphery between the point of exit of these nerves; but also dorsally in the angle formed by the brachium conjunctivum with the lingula, a small mass of degenerated fibres cut transversely becomes visible, and these represent the descending turn of a

loop formed higher up by the passage of the ventral fibres of the tract over and round the root of the fifth cranial nerve. In sections made higher up the two portions of the loop approximate to one another, and at the level of the lowest point of exit of the fourth nerve the extremity of the loop can be discerned as a strip of fibres lying externally to the lateral lemniscus. Having looped in this manner the tract proceeds to the white centre of the superior vermis by way of the superior cerebellar peduncle and valve of Vieussens.

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A Long Sensory Tract in the Grey Substance of the Cord—(Ciagliński).

In the *Neurologisches Centralblatt* for September, 1896, Adam Ciagliński draws attention to the existence of a long tract situated in and coursing along the grey matter of the cord,

and although his observation still requires confirmation, it is of such an interesting nature as to demand separate consideration.

Ciagliński drew a tight ligature round the cords of dogs in the lumbar region and killed the animals after from 4-24 hours. The cords examined by the method of Marchi showed, in addition to the usual ascending changes in the posterior columns, a diamond-shaped or rhomboidal area of degeneration situated in the grey commissure; between the ventral extremity of the posterior columns and the central canal, the area was composed of an actual column of fibres which, diminishing in volume as it ascended, was traceable upwards as far as the cervical enlargement.

This writer claims to have seen the tract in the normal human cord, but by this he can only mean a few sparsely disseminated fibres to be found in this region. He believes that its fibres are derived from the posterior roots and physiologically belong to that group of sensory fibres intended for the conveyance of impressions of heat and pain. If this be correct, and it is not an unsupported presumption, having been alluded to by Bellingeri in 1823, and Schiff in 1856, degeneration of the tract may explain certain sensory anomalies (loss of sensations of heat and pain) peculiar to syringomyelia.

LITERATURE.

Ciagliński. "Lange sensible Bahnen in der grauen Substanz des Rückenmarkes und ihre experimentelle Degeneration." *Neurolog. Centralb.*, 1896, No. 17.

Lateral Limiting Layer.

The lateral limiting layer, as described by Flechsig, is a part of the lateral columns contiguous or adjacent to the grey substance, which in the upper cervical region bounds practically the whole outer side of both cornua, in the cervical enlargement occupies the angle between the anterior and posterior cornua, and in the dorsal region again reaches forwards outside the anterior cornu, but altogether forms a less voluminous bundle than in the upper cervical region. In the lumbar region the layer requires further study.

Flechsig supposed that the layer was made up of fibres derived from two sources, the posterior roots and the cells of Clarke's columns, but we now know that this is incorrect, for, firstly, the layer does not degenerate in tabes dorsalis, nor does section of the posterior roots affect it, therefore it can have no direct con-

nection with the posterior roots; and, secondly, it is believed that Flechsig mistook for fibres of this layer the neuraxons of the cells of Clarke's column which pass through the lateral limiting layer on their way to the direct cerebellar tract, as described by Mott. It is more likely, as Ramon y Cajal contends, that the layer is composed of fibres which, arising from cells in the anterior or posterior cornua, after a short course either upwards or downwards, again return to the grey matter.

In 1893 Bechterew drew attention to a special bundle of fibres in the posterior portion of Flechsig's lateral limiting layer, which he designated the "mediales Seitenstrangsbündel," he states that this bundle is distinguishable from Flechsig's layer in that its fibres develop their medullated sheath at a later period. It first becomes evident in the dorsal region situated immediately to the outer side of the grey substance between the lateral and posterior cornua, so occupying a portion of the territory between the lateral pyramidal tract and the cornua; further upwards the bundle advances more to the front, but still remains close to the grey substance. Bechterew, though doubtful concerning its constitution, presumes that its fibres stand in relation to cells in the grey substance, and that these fibres must be of very limited length, as they have never been seen degenerated in the human cord.

In a case of amyotrophic lateral sclerosis recently recorded by Bruce, the interesting point was determined that the anterior or ventral part of the lateral limiting layer had undergone partial sclerosis, while the posterior portion was preserved intact, and since atrophy of the anterior cornual cells is a fundamental change in amyotrophic lateral sclerosis, this observation in a measure proves that the anterior portion of the lateral limiting layer is connected with anterior cornual cells, and it suggests a relation between posterior cornual cells and the posterior portion. The layer may, therefore, be said to be composed of two independent systems which merely agree in that the fibres arise from cells in the grey matter and are of short course. Bruce suggests the names antero-lateral and dorso-lateral for these two divisions, but at the time that he wrote seems to have been unaware of the fact that Bechterew had already given a special description of the posterior division.

LITERATURE ON THE LATERAL LIMITING LAYER.

Von Bechterew. "Die Leitungsbahnen im Gehirn und Rückenmark." Leipzig (Besold), 1894.

Von Bechterew. "Ueber das besondere, mediale Bündel der Seitenstränge." *Neurol. Centralb.*, No. 15, 1897.

Bruce. "On a special tract in the lateral limiting layer of the spinal cord." *The Scottish Med. and Surg. Journ.*, vol. i., No. 1, 1897.

Flechsig. "Die Leitungsbahnen im Gehirn und Rückenmark des Menschen auf Grund entwickelungsgeschichtlicher Untersuchungen dargestellt." Leipzig, 1876.

Anterior Ground Bundle or Basis Bundle.

Excluding the fibres of the direct pyramidal tract, the antero-lateral ascending tract, the crossed afferent tract of Edinger (*vide* antero-lateral ascending tract), and the ascending and descending sulco-marginal tracts, the anterior column is composed of the anterior ground fibres, all of which lie in the immediate neighbourhood of the anterior cornu. The volume and the depth of the bundle varies at different levels. From the degenerative point of view it possesses little interest, as secondary changes in its fibres, even in a case of transverse myelitis, cannot be traced beyond the "traumatic zone." Flechsig and Bechterew, however, in their developmental researches, brought out several important points concerning these fibres; in the first place it was discovered that they received their medullary investment at an early date, viz., the fifth month (considerably earlier than the antero-lateral ascending tract, but not long before the direct cerebellar tract), then it was found that all do not become medullated at once, the first to develop being those which lie in immediate contact with the anterior cornu, and those are supposed to be related to fibres of the anterior roots; others which develop later, it is presumed, are short internuncial vertical fibres connecting cells in different levels of the anterior cornu.

Cornuo-Commissural Bundle (Marie).

It would appear from experimental anatomical evidence that a bundle of fibres called by Marie the "faisceau cornu commissural," situated in that part of the column of Burdach which borders the posterior commissure, and also about the ventral half of the posterior cornu, cannot be grouped with the long fibres of the posterior columns, but must be classed among the short internuncial or anastomosing fibres connecting different levels of the grey substance. This bundle escapes degeneration after root lesions, and topographically corresponds with Flechsig's anterior

rootzone or "ventrales Hinterstrangsfeld," with the exception that, according to Marie's representation, it is hardly so extensive. The tract requires further study.[1]

REFERENCES.

MARIE. "Leçons sur les Maladies de la Moelle." Paris, 1892.

CHANGES IN THE SPINAL CORD FOLLOWING AMPUTATION OF EXTREMITIES.

Since the early part of the century, contributions to the literature on this subject have been constantly appearing, and on glancing through the records one finds that while there have been minor differences among the various writers the general results obtained have been tolerably uniform. The predominant alteration is an atrophy of that half of the spinal cord corresponding to the side of the amputation. This has almost invariably been noticed, and is represented by a general reduction in volume of white and grey matter alike, involving those particular segments of the cord which receive and give off the sensory and motor nerves which originally supplied the skin and muscles of the amputated member or part thereof. That the atrophy is approximately confined to these particular segments may be definitely proved, if in a case of amputation these segments be carefully examined and compared with those above and below the suspected level; this was done in two cases coming under my notice, and the results were very definite. That the spinal hemiatrophy has not been definitely localised in this manner by previous writers is obviously owing to the fact that the areas appertaining to the various posterior spinal nerve roots have only been mapped out quite recently; but on analysing records and critically examining published drawings, one finds that the hemiatrophy has almost without exception been noted as being most prominent at the levels which I have emphasised. The cord above and below these levels gradually reassumes its symmetry, and the atrophy is of course not absolutely confined to them.

Studying these changes in greater detail, and in the first place taking the white matter, one finds that though an actual localised

[1] Since the above was written an article by Alexander Bruce "On the Endogenous or Intrinsic Fibres in the Lumbo-sacral Region of the Cord" has appeared in this Journal (part iii., 1897), and therein will be found an excellent description of this set of fibres.

sclerosis may occur, such a change is rare. Marie has reported two such cases, one of amputation of the thigh, another of amputation of the arm, and in both there was ascending bilateral sclerosis in the root area of the posterior columns, most marked on the side of the amputation. Two Italian observers, Guarneri and Bignami, also describe sclerosis of the antero-lateral ascending tract on the opposite side in consequence of an amputation. It appears that for the production of this sclerosis the amputation must be very old standing (in Marie's cases the operation had been performed twenty years previously, and Guarneri and Bignami's bore a similar history). Other changes which are constantly seen in the atrophied white matter are an increase in the nuclei of the neurilemma and an apparent proliferation of the glial elements, but it is worthy of note that though all the white columns of the cord on the side of the amputation may be appreciably reduced in volume, still their staining reaction in all the ordinary methods is practically identical with that of the healthy half.

Next, in regard to the grey substance, it has been mentioned that there is always a concomitant general reduction in its volume, likewise there is a general reduction in number of its contained nerve cells, both large and small, and furthermore there is one special group of cells which, both in the case of amputations of the upper and lower extremities, may be singled out as being particularly prone to atrophy, viz., the postero-lateral group of the anterior cornu. Grigoriew, who is the most recent writer on this subject, is disposed to dissent from this statement, but there is little question concerning its accuracy, for on analysing the literature, one is at once struck with the frequency with which the group is figured or described as having suffered most. Sherrington made a special note of this point in his research on the lumbo-sacral plexus, and uses it in support of his suggestion that in the lumbo-sacral region the postero-lateral group of nerve cells stands related to the intrinsic muscles of the foot. Yet another group of nerve cells which undergoes atrophy after an amputation of the lower extremity is that of the posterior vesicular column of Clarke. Friedländer and Krause first drew attention to this fact, and I noticed it in a case in which I stained my specimens by the method of Nissl.

From the experimental side the subject of the spinal changes after amputations has been vigorously investigated by Homén, and less seriously by Sherrington and others. The former, using the dog as his subject, has succeeded in reproducing most of the changes which have been observed in man; he mentions that

young animals are most suitable for these experiments, and states that in puppies changes commence at the periphery somewhat more than a week after the operation, but in full-grown animals do not appear until one or two months have elapsed.

Friedländer and Krause and Erlitzky maintain that only the sensory neurons atrophy after amputations. Homén's experiments lead him to a similar conclusion, and he publishes apparently convincing coloured plates representing transverse sections of the posterior root ganglia, and showing plainly an intact motor root lying alongside the withered ganglion. But the integrity of the spino-muscular neuron here referred to, or even of the cortico-spinal neuron, can surely only apply to cases of more or less recent amputations, for there is abundant evidence on record to show that not only do the motor roots in course of time atrophy, but also motor anterior cornual cells, and even, likewise, cells in the motor sphere of the cerebral cortex (Charcot and Pitres, Wiglesworth, &c.).

Into the condition of the peripheral nerves in cases of amputation I do not propose to enter at length. The majority of observers state that they are reduced in calibre (Marie has seen them enlarged), the fibro-cellular tissue in them is increased in quantity, the perineurium is thickened, and sometimes myxoid or fatty, and the healthy fibres are reduced in number by about one-half; these changes diminish in severity as one ascends the nerve trunk.

Marie, who in his lectures refers in some detail to the alterations in the peripheral nerves, describes the presence in them of numerous islets, about four times the size of a healthy nerve-fibre, composed of a collection of some five to twenty delicate nerve fibres, possessing an almost imperceptible white sheath. These islets he looks upon as degenerated remnants of an originally healthy fibre, and calls *ilôts de dégénération*.

Some writers have described the posterior root ganglia as intact; others have found them diminished in size, their nerve-cells reduced in number, and their nerve-fibre plexus attenuated. We may take it that in old-standing cases of amputation they are always altered in the same way as the anterior and posterior spinal roots are.

What the precise pathology of the changes in the central nervous system after amputations is, is still *sub judice*. Clearly the change in the sensory nerve distal to the ganglion must be a true secondary degeneration, and we might expect atrophy of the sensory neuron above the ganglion in consequence of loss of function. The atrophy of the system of motor neurons may be

of the nature of a retrograde degeneration or sclerosis, similar to that described elsewhere in the cord. Obviously the spinal changes are not the result of a true secondary degeneration, and the distinguishing term "Gudden's atrophy" has been applied to them.

LITERATURE ON CHANGES IN SPINAL CORD AFTER AMPUTATIONS.

GRIGORIEW. "Zur Kenntniss der Veränderungen des Rückenmarkes beim Menschen nach Extremitätenamputationen." *Zeitschr. für Heilkunde, Prag.*, Band xv., 1894.

HOMÉN. "Veränderungen des Nervensystems nach Amputationen." *Beiträge zur path. Anat.* (Ziegler), Band viii., 1890.

MARIE. "Leçons sur les maladies de la moelle." Paris, 1892.

CHARCOT ET PITRES. "Sur quelques points controversés de la doctrine des localisations cérébrales." *Arch. Cliniques de Bordeaux*, 1894.

ERLITZKY. "Ueber die Veränderungen im Rückenmarke bei amputirten Hunden." *Petersb. Med. Wochenschr.*, 1880.

FRIEDLAENDER UND KRAUSE. "Veränderungen der Nerven und des Rückenmarkes nach Amputationen." *Fortschr. der Medicin.*, Band iv., 1886.

WIGLESWORTH. "A case in which an old amputation of the left upper arm was associated with an atrophied right ascending parietal convolution." *Journ. of Ment. Science*, 1886.

SHERRINGTON. "Notes on the arrangement of some motor fibres in the lumbo-sacral plexus." *Journ. of Physiology*, vol. xiii., 1892.

Full lists of writers on this subject will be found in the papers of Sherrington, Homén and Grigoriew.

HEREDITARY FORM OF PROGRESSIVE MUSCULAR ATROPHY WITH SPINAL LESION IN YOUNG CHILDREN.

(1) "Zwei Frühinfantile Hereditäre Fälle von Progresver Muskelatrophie unter dem Bilde der Dystrophie, aber auf Neurotischer Grundlage." Werdnig. (*Archiv für Psychiatrie*, 1891.)

(2) "Ueber Chronische Spinal Muskelatrophie in Kindesalter auf familliärer Basis." Hoffman. (*Deutsche Zeitschrift für Nervenheilkunde*, 1893.)

(3) "A Case of Progressive Muscular Atrophy in a Child with a Spinal Lesion." Thomson and Bruce. (*Edinburgh Hospital Reports*, 1893.)

(4) "Die Frühinfantile Progressive Spinale Amyotrophie." Werdnig. (*Archiv für Psychiatrie*, 1894.)

(5) "Weiterer Beitrag zur Lehre von der Hereditären Progressiven Spinalen Muskelatrophie in Kindesalter." Hoffmann. (*Deutsche Zeitschrift für Nervenheilkunde*, 1897.)

The above papers deal with a form of muscular atrophy occurring in children which as yet has not obtained general recognition.

The first paper, by Werdnig, deals with two cases, the former of which occurred in a boy, the eldest child of healthy parents. At the tenth month of life weakness of the legs was first noticed (the child having been previously in good health); this was followed by weakness of the muscles of the back, loss of power in the arms so that the child could no longer raise the hand to the mouth, this loss of power being preceded by shaking movements of the arms. The child could no longer support the weight of the head (which, however, was somewhat hydrocephalic). Sensibility was unimpaired; skin reflex was present, but the patella, biceps, and triceps reflexes were lost.

The *post-mortem* revealed a condition of chronic hydrocephalus, some degeneration of the pyramidal tracts, atrophy of the cells of the anterior horns, especially in the cervical and lumbar enlargements, and degeneration of the anterior roots. The gastrocnemius muscle was alone examined and showed simple atrophy.

The second case, brother of the above, was, like him, noticed to be weak when 10 months old; the weakness started in the legs and back; choreiform movements and fibrillary tremors of the muscles were present. The knee-jerks were lost, sensibility was normal. The reaction of degeneration was present in some of the muscles. He died at the age of 6 years. The *post-mortem* and microscopical conditions are described in Werdnig's second paper. The cervical and dorsal cord appear to the naked eye to be normal in appearance; in the lumbar region the distinction between the white and grey matter is not clearly defined; the anterior roots are thin in comparison to the posterior and somewhat darker in colour. The muscles were for the most part extremely atrophied, pale, and many of them fatty.

Microscopically some change was noted in the cells of the nuclei of the motor portion of the 5th and in the 7th. The motor cortex was normal. The pyramids were normal. The cord showed atrophy of the cells of the anterior horn in the cervical and lumbar region; large cells appear but with cloudy contents and indistinct nuclei. The cells of Clarke's column appear to be normal, as well as the cells of the lateral horn. The anterior roots show considerable change; there are fewer bundles and an increase of nuclei and of interstitial tissue; some of the axis cylinders were swollen, and by the Marchi method showed some degeneration. The cauda equina was in parts completely degenerate, while in other parts was normal in appearance. The peripheral nerves, both with Marchi and with Pal, showed considerable degeneration.

In the muscles the most frequent change was simple atrophy, next a homogeneous degeneration, and lastly a ("schollig") degeneration; no fatty degeneration could be found. Large round muscle fibres remained at certain points, and a variable amount of fat and connective tissue; the condition of the muscles is not one of simple atrophy, but is a mixed process.

The first paper by Hoffmann deals with the case of a child (a girl) who, apart from being unusually fat, was well till the ninth month of life; from that time on the child began to lose power, first of standing, then of being able to sit up or turn itself round in bed. The child was intelligent, learnt to speak and never

had any fits. In the place of the fat the extremities and the buttocks became wasted; no atrophy of the face occurred, and the child had no difficulty in swallowing. The child became absolutely unable to lift the head from the pillow, but could roll it from side to side. No fibrillar tremors, no trophic or vaso-motor disturbance were present. Sensation in all the forms remained normal. Pseudo-hypertrophy was never present. Reaction of degeneration was present. The child died when 4 years old. The autopsy showed marked atrophy of the anterior roots of the spinal cord; the muscles were a pale yellow colour, the quadriceps being the most atrophied.

Microscopical examination showed the brain to be normal; extensive atrophy of the cells of the anterior horn was present, but some unaltered cells existed in this region; atrophy of the anterior roots, and also of some of the fibres of the cauda equina and the peripheral nerves.

The muscles had undergone most extreme atrophy, so that in certain specimens not a single normal fibre could be found; hypertrophied fibres were present, and these contained more nuclei than normal; muscle schläuche (spindles) are described.

The intra-muscular nerves contain a fair number of degenerated and degenerating fibres, and rarification of the medullary sheath of the nerve is also described.

No alteration was found in the diaphragm, the masseter, or the cardiac muscle.

The second case, brother of the above, was first seen when $2\frac{3}{4}$ years old. As with his sister he was apparently a normal child till 9 months old, and although he never learnt to stand or walk, he even lost the power of sitting up in the cradle. The loss of power was gradual and not attended with pain. The child learnt to speak and was quite intelligent; he swallowed without difficulty.

When examined the child was fat but without obvious signs of rickets; it was unable to lift up its head and the arms were atrophied, even the small muscles of the hand being affected. The most marked weakness was, however, in the gluteal muscles and the muscles of the thigh; flexion of the hip could not be performed. The knee jerks were absent. This child died when 5 years old, but no autopsy was allowed.

The third case occurred in a boy, first seen when 9 months old, having loss of power in the gluteal, thigh and back muscles. Reaction of degeneration was present. The knee jerks were absent. The small muscles of the hand were affected, but there

was no fibrillary tremor. The child died when 14 months old, but no autopsy was obtained.

The report of a fourth case is also given without autopsy.

In the second paper by Hoffmann he reports a case, the sister of the child described in case No. 3 of his first paper.

This child, born of healthy parents and in natural labour, was apparently healthy till the seventh month of life, when it began without known cause, or sign of acute or chronic disease, to have weakness in the hip and thigh muscles of both sides; this weakness gradually extended to the muscles of the back, the neck and shoulder; later in the course of the disease the upper arm, the lower arm, and the muscles of the hand on one side and the leg muscles of the other side became affected. There were no fibrillary tremors and the sensation and the sphincters were normal. The knee jerks were absent. No electrical examination of the muscles was made.

The mental development was not impaired, and the face, tongue, and deglutition muscles were normal. Death took place at $2\frac{1}{2}$ years from secondary lung affection.

Microscopical Examination.—The brain, cerebellum, the cranial nuclei and nerves were normal. From the medulla downwards the nerve cells of the anterior horn are atrophied or have entirely disappeared; the nervous network in the anterior horn is less than normal, and the neuroglia is thicker than natural.

The direct pyramidal and the lateral columns are by the Weigert method somewhat paler than normal, and Goll's column is rather paler than Burdach.

The anterior roots are reduced in volume and degenerated; in cross section there appear small islands of structureless tissue; these are thought to be nerve bundles which have become converted into connective tissue; they are found only at the exit of the anterior roots, and not in the posterior roots or in the cauda equina. Whole bundles of the nerves in the cauda equina are found to be degenerated.

Changes were also found in the sciatic and peroneal nerve, and to a lesser degree in the ulnar and radial.

The muscle fibres are reduced in size, measuring about 5-10 μ; they are round in form and the striation is well preserved even in the very smallest. There is also a diminution in the number of nuclei in the muscle fibre; normally there are about $1\frac{2}{3}$ of a nucleus to a fibre, while in the present case there is only about $\frac{2}{5}$ to $\frac{3}{5}$ of a nucleus to a fibre. There is no fatty degeneration or vacuolation; the intermuscular nerves are somewhat degenerated. Muscle spindles are noted as being present.

The paper by Bruce and Thomson deals with the case of a child, the daughter of healthy parents, there being two other children in the family : (1) a boy who has congenital talipes varus ; and (2) a girl who is strong and healthy.

At birth and during the first twelve months of life the child was quite healthy. Teeth began to appear at the sixth month, and at the twelfth month she could walk round the room holding on to a chair. Soon after the beginning of the second year of life she became unable to walk, and at eighteen months she could not get on to her feet. At the twenty-eighth month she could still stand with support, and was able to sit up and feed herself ; she could sing and talk loudly ; her arms remained fairly strong. When 3 years old she began to have pains in the legs, generally referred to the knee ; the pain recurred at intervals for four months, but the attacks did not last long. On examining the child when 3 years old the appearance of limpness was very noticeable ; the child could sit on a chair when balanced, but was very easily upset. She was bright and intelligent ; there was some twitching of the angles of the mouth, and sometimes of the corrugator supercilii, also of the orbicularis oris and the orbicularis palpebrarum. Sensation to touch, to heat and cold were well preserved, but to the faradic current the child seemed quite insensitive. Deglutition, respiration, micturition, defæcation, perfectly normal. Knee jerks were absent ; there was no A.C. The weakness was general ; there was no special weakness of any group of muscles. The paresis was least marked in the shoulders and arms, more in the neck, back and abdomen, and most in the loins, buttocks and lower limbs. No hypertrophy or enlargement of any muscle. Co-ordination perfect.

Electrical examination showed diminished irritability to the faradic current, more marked in the legs than in the arms. The galvanic irritability was almost minimal. Distinct R.D. was never found. The sensibility to the faradic current was entirely abolished ; the coil could be pushed up to its utmost limit without the child wincing.

The child died when 6 years old.

Post-mortem the brain and cord appeared normal ; the muscles were pale and some were fatty.

Microscopical examination of the muscles showed all degrees of atrophy ; hypertrophied fibres were present, and an increase of the nuclei of the sarcolemma ; the striation was well preserved in all except the smallest fibres. The occurrence of minute muscular fibres in the centre of a nucleated fibrous ring is noted ;

sometimes these rings are divided into compartments, one of which contains a nerve fibre. These are muscle spindles.

The sciatic and nerves of the brachial plexus were examined by Weigert's method, and atrophy of some fibres and attenuation of the myelin sheath of other was found. The spinal cord showed no tract degeneration; the posterior roots were normal, but the anterior showed slight atrophy. The cells of the anterior horn had to a great extent disappeared, and those remaining had undergone considerable atrophy. In the lumbar region the process had proceeded so far that only a few highly atrophic cells could be seen. None of them presented any abnormal pigmentation.

There remains an uncertainty as to which was the primary lesion in this case. The authors, however, think that it might be classed under the "simple atrophic" form of Erb.

The characteristics of the disease, as illustrated by the above cases, are, then, as follows: an apparently healthy and intelligent child who has made normal progress to the age of 10 months, begins without any sudden onset or known cause to lose power, the weakness being first noticed in the muscles about the hips and in the muscles of the back. The disease pursues a progressive course, the shoulders, the thighs, the upper arm, the forearm and leg being successively involved, and finally the muscles of the hands and feet become affected, the parts being involved in the order above-mentioned. No special group of muscles are affected. Fibrillary twitchings of the muscles are present in some cases. Bulbar symptoms may supervene, and contractions of the limbs may be present in some cases (Werdnig). The limbs are absolutely flaccid, reaction of degeneration is often present, the deep and sometimes the superficial reflexes are abolished. There is as a rule no tenderness or pain, though the latter may be present (see Thomson and Bruce's case). There is no disturbance of sensation, but again in the case just referred to there was an extraordinary absence of pain to the stimulus by the faradic current. The sphincters are normal. The mental condition continues unimpaired throughout the whole course of the disease. The atrophy of the muscles becomes extreme, and the disease runs its course in at most a few years. Heredity plays an important part in the etiology of the disease; in one family of six, two brothers were affected; in another of fifteen, four boys and two girls were affected; in a third family of nine, two boys and a girl were affected; and in a fourth family of twelve, eight were affected in one generation, and two girls and a boy in the

second generation, through the mother. The evidence on which some of the above is based is open to doubt, as out of the above twenty-two cases only four were examined pathologically.

In Thomson and Bruce's case there was no hereditary or family history. The pathological condition which has been found in these cases is atrophy of the cells of the anterior horn, together with degenerative changes in the anterior nerve roots and in the peripheral nerves. In the muscles sometimes simple atrophy is found, sometimes the condition usually found in cases of primary muscular atrophy.

Whether the atrophy in the cells of the anterior horns is the primary lesion or is secondary to the condition of muscular atrophy is a point that is difficult to decide; certain evidence in the above cases would seem to point to the probable spinal origin of the disease.

F. E. Batten.

Reviews and Abstracts.

Eye-Strain in Health and Disease; with special reference to the amelioration or cure of Chronic Nervous Derangement without the aid of Drugs. By Ambrose L. Ranney, A.M., M.D., 1 vol., 8vo., p. 321. Davis Co., Philadelphia.

In this volume the author has brought together the substance of several monographs, published in past years in various medical journals, and has added considerable new matter.

It evinces signs of much careful and laborious work, and deserves, therefore, patient and impartial consideration on the part of the reader. The tone of the book as a whole is, perhaps, somewhat excessive and extravagant, but this may possibly be explained by the desire of a man, with an enthusiastic belief in his subject, to impress strongly his views of the case upon the world. Some fifty or sixty pages are given up to a very concise and remarkably clear account of the steps to be taken in examining the vision and the ocular movements. This, a little enlarged and improved, would make a useful text-book if published in a separate volume, but seems, to a certain extent, out of place in its present environment.

The so-called "Heterophoria" occupies a very important position in this work, one equal, if not superior, to that held by errors of refraction, with regard to the causation and treatment of nervous diseases. In this country we do not recognise it as being of very frequent occurrence, nor as producing such enormously injurious results to the nervous system. We acknowledge that lack of equilibrium of the ocular muscles exists, and have done so for many, many years, but we think that "heterophoria" and its treatment by operation (graduated tenotomy), as at present practised, is carried too far by some of our brethren in the United States. Possibly the citizens of the United States suffer from this trouble more than we do.

The rest of the book consists, in a large part, of cases of many and various kinds, showing the results of treatment by glasses and operation, interspersed with remarks and arguments. The great

principle which Dr. Ranney advances strongly is the removal of peripheral irritation, and he desires to show that the eyes in a large number of cases are undoubted sources of trouble. He deprecates the indiscriminate use of drugs, especially the bromides, and earnestly begs for the careful examination of the eyes and other organs of the body before medicines are employed. All this is just and proper, and cannot be too frequently set forth before the profession at large. Dr. Ranney recognises neurasthenia, &c., as a frequent result of eye-strain, but, curiously enough, says little about cases of eye-strain as a consequence of nervous trouble, which latter form a large portion of the patients who have to be treated for eye trouble.

The book is worth reading. In doing so, however, it is necessary to guard oneself against an impression produced, viz.: that eye-treatment cures or relieves everything.

H. Work Dodd, F.R.C.S.

The New Psychology. By E. W. Scripture, Ph.D. (Contemporary Science Series. Walter Scott, London.)

This is distinctly a good book. Although laying no claim to to be an exhaustive compendium like those of Wundt, Ladd, and Külpe, it is in reality a great deal more compendious, as well as more readable, than most other books of equal size. To anyone —whether physiologist or neurologist—who has had a passing acquaintance with the literature of experimental psychology of recent date, it is a valuable *résumé* and digest of some of the best work that has appeared. To the professing laboratory psychologist it is valuable by reason of the numerous experimental hints and the considerable amount of original matter embodied in its pages.

Part I., consisting of four chapters on Observation, Statistics, Measurement, and Experimenting, are couched in a tone that should go far to allay the suspicions with which most scientific readers are apt to view an exponent of the "new psychology." The figure on p. 65, from Hansen and Lehmann, serves as a rather startling illustration of the way in which one successfully makes out points of resemblance in apparent "proof" of thought-transference. The similarity in this case is at first sight close enough to arouse the incredulity of an ordinary observer, or to

satisfy a psychical researcher that thought has been "transferred," yet it is clear enough on closer investigation that the apparent similarities are mainly invented by the observer, *i.e.*, oneself. The contrast between this investigation and that of an untrained scientist (a well-known psychical "researcher") is very effectively brought out by the brief summary of the "experiments" on thought-transference by Ochorowicz.

Part II. consists of an account of measurements of various kinds of reaction-times, and of the time-factor in memory, and of fatigue-phenomena. Several useful pieces of apparatus are figured and described; and the spark-dot method of recording long series of reaction-times strikes one as bringing out results in a very elegant form. Exner's sensation-curves, and the plotted data upon which the logarithmic law of declining memory is based, catch the eye as the pages are turned over, and are welcome. But the internal mechanism of the kinetoscope is not particularly welcome—in this connection at least.

Part III. deals with the manifestations of energy, and with the sensational effects of stimulation. Dynamometry, fatigue, the influence of mental states, and of various kinds of sensificatory stimuli upon volitional power are considered; the plotted curves exhibiting the effects of cerebral fatigue upon the movements of the eyes and upon the speed of accommodation are of special interest; these, as well as the "tapping-frequency" experiments mentioned in an earlier chapter, are the results of work done by the author or under his supervision. This is also the case in the measurements of suggestibility described at pp. 272-282. The tricolour lantern figured on p. 349, appears to be an excellent means of demonstrating colour mixtures on the screen.

Part IV. is concerned with the psychological dimensions of space.

Part V., of which one chapter is devoted to a biography of Fechner, Helmholtz and Wundt, takes stock of the present state and immediate future of the new or quantitative psychology. The author takes a sober and temperate view of its scientific gravity in the present, but a very hopeful view indeed of its development and ultimate fruitfulness.

Several useful tables are contained in the Appendix, and the following note (p. 484) on the measurement of illusions in it strikes one as capital. One does not feel the title to be hopeful, nor yet the opening paragraph, but the two concluding ones are quite satisfactory and eminently convincing of the soundness and ingenuity of their author. The paragraph runs thus:

"*On the Measurement of Imagination.*—In the course of the investigations on measuring hallucinations it occurred to me that it might be possible to measure the intensity of an imagination also. The experiment was successful. The method is not difficult and is readily intelligible. In order to explain the method it will be sufficient to describe the first simple experiment made.

"The apparatus consists of a screen of fine tissue paper. The tissue paper is illuminated by daylight in front and by a gas flame at the back. When the gas flame is turned down, the eye looking through a telescope tube sees a plain white circle illuminated by daylight.

"The first experiment was made on a student acccustomed to using the telescope. He was told to imagine hair lines on the white surface, like the hair lines seen in an astronomical telescope. This was successfully done. He was asked to describe them and compare their blackness. There is, he said, a horizontal line, which is the blackest of them, and three vertical lines of about equal blackness. He was told that the field of view was to be made gradually lighter by turning on a flame behind, and he was to tell how the lines behaved. As the gas was slowly turned on, he described various changes in the lines. Finally he said he saw a slant line that he had not imagined before. It appeared just about as black as the horizontal line and blacker than any of the others. Thereupon the experiment was ended.

"The slant line was a real line. This he did not and still today does not know. On the back of the tissue paper a slant line had been drawn, and as the gas was turned up of course it showed through. Thus we had a direct, unsuspecting comparison of intensity between a real line and an imaginary one.

"The photometric determination of the intensity of the real line is not a difficult matter. A phantasimeter has been devised in which the graduation is done beforehand, but the simple arrangement just described serves to indicate the method of experiment."

A. D. W.

Anæsthesia of the Trunk in Locomotor Ataxia. By Hugh T. Patrick, M.D., *New York Medical Journal*, February 6, 1897.

Sensory Disturbances in Locomotor Ataxia; a Study of the Localisation of Anæsthetic Areas as an Early Symptom. By Allan Blair Bonar, M.D., *Medical Record*, New York, May 22, 1897.

Hitzig and Laehr first drew attention to the sensory affections of locomotor ataxy. Patrick and Bonar have carefully investigated twenty and twenty-one cases of tabes respectively, with special reference to the areas of sensory disturbance on the trunk.

Seventeen of Patrick's cases and eighteen of Bonar's exhibited such areas. Laehr had found them in fifty-five out of sixty cases.

The sensory disturbances in question are mostly in the form of tactile anæsthesia, but there may be analgesia, and in a few cases loss of sensibility to heat and cold. They occur as more or less broad bands in the neighbourhood of the nipple. They may be unilateral or bilateral, with corresponding areas on the back. They may extend to the mid line, or fall short of it. Sometimes there are intervening areas of normal sensation between plaques of tactile anæsthesia. When bilateral the areas may be at different levels. Commonly above the anæsthetic areas there is a zone of hyperæsthesia, mostly (according to Hitzig) to cold.

When the anæsthesia reaches the level of the third rib it also involves a tongue-shaped portion of the axillary side of the corresponding arm, and when it extends above this level in the chest the anæsthesia spreads throughout the distribution of the ulnar nerve.

The anæsthesia does not correspond to the distribution of the intercostal nerves, but answers to the segments of the spinal cord, as differentiated by Kocher.

This is, however, not usually the case, Bonar says, when the sensory disturbance affects the lower extremities.

Laehr states that anæsthesia of the trunk only extends downwards, but both Patrick and Bonar have found numerous exceptions to this rule.

All agree that the anæsthesia is extremely variable, and that its boundaries are more easily defined when approached from within their limits than from outside.

Anæsthetic areas may increase or decrease, and even disappear, or hyperæsthesia may give place to anæsthesia. The conditions do not advance progressively with the course of the

disease. They may be found in cases of 3 to 5 years' duration, and may be hardly marked in those which have lasted 25 to 28 years.

They are usually least apparent in cases where optic atrophy is prominent. The superficial reflexes are increased over hyperæsthetic zones, and diminished or absent in those of anæsthesia.

As regards the causation of tactile anæsthesia of the trunk, Patrick points out that any process which involves the posterior nerve roots, such as tumour, meningitis or spinal caries, may produce it; it may be present in syringomyelia and hysteria. In tabes he regards it as evidence of intra-medullary affection of the long fibres, which pass directly upwards in the posterior columns, leaving uninjured those that pass by various ways into the posterior grey horns and columns of Clarke.

As regards the diagnostic value of trunk anæsthesia, Patrick thinks it is probably not very great.

It seemed of value only in one case, and it was by no means absolutely certain that this was a case of tabes.

Laehr found it early, but except in one case not so early as analgesia of the legs.

Bonar regards paræsthesiæ in general as valuable evidence of early tabes.

He found them in a number of consecutive cases, and says that they often exist unknown to the patient unless involving the hands, feet, or buttocks.

The present writer has usually found some amount of tactile anæsthesia of the trunk in well-marked cases of tabes, but not in doubtful cases. One patient for upwards of a year has had progressive atrophy of both discs—Argyll Robertson phenomenon—and extremely sluggish knee jerks; but his sensation is perfect from head to foot. This, however, perhaps bears out Laehr's statement that sensory disturbances are commonly absent when optic atrophy is prominent.

LEONARD G. GUTHRIE.

PROCEEDINGS OF THE SOCIETY.

At a Meeting of the Society held on December 8th, Dr. Hughlings Jackson delivered the first Hughlings Jackson Lecture, the subject of which was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1897,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1896.

LONDON
JOHN BALE & SONS
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1897

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Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 14, 1897.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The Members now number 171.

The Inaugural Address was delivered at the Annual General Meeting on January 16th, by Dr. ALEXANDER HILL, Master of Downing, on "The Chrome Silver Method: a study of the Conditions under which the reaction occurs and a Criticism of its Results."

The Council has much pleasure in announcing that it has, by request, inaugurated a lectureship in honour of the discoverer of Cortical Epilepsy and its relation to Cerebral Localization, to be termed the "Hughlings Jackson Lectureship." The Council anticipate that the establishment of this lectureship during the lifetime of Dr. JACKSON will be productive of the best results for Neurological science; moreover, it is gratifying to know that Dr. JACKSON has kindly consented to give the first Lecture this year, the date of which will be duly announced.

The Index of *Brain* is almost completed, and the Council desires to express its obligations to Sir WILLIAM BROADBENT, Bart., for having generously defrayed the expenses thereof; without this assistance it would have been impossible, as the appended Balance Sheet shows a large expenditure in illustrating the Journal. The increased cost of *Brain* has been, and still is, the subject of consideration by the Council.

The Council regrets the loss sustained by the Society by the death of an illustrious Corresponding Member, Professor SCHIFF, of Geneva; also of Dr. LANGDON DOWN, and of Dr. BLANDFORD, of Croydon. The proceedings appended hereto show the character and value of the work of the Society during the past year.

Proceedings of the Society during the year 1896.

January 16th.—Dr. Alexander Hill, Inaugural Address, "The Chrome-Silver Method: a study of the Conditions under which the reaction occurs and a Criticism of its Results," *Brain*, vol. xix., p. 1.

March 5th.—A Discussion on "The Visual Areas of the Cerebral Cortex" was opened by Dr. Sharkey, Professor Schaefer, Drs. Ferrier, Beevor, Lauder Brunton, Goodall, and Risien Russell took part in the Discussion, and Dr. Sharkey replied.

May 14th.—Dr. Colman, "Meningocele with Extreme Exophthalmos, and an obscure case resembling Friedreich's Disease." Professor Victor Horsley, "Case of Removal of a large Cerebral Tumour, 23 months after the operation." Dr. Ferrier, "Bilateral Paralysis of the Muscles of Expression and Mastication." Dr. Beevor, "Two Cases of Bilateral Paralysis of the Soft Palate." Dr. Ormerod, "A Case with Ataxia, Paraplegia, and Affection of Speech, and Family Paralysis commencing in Muscles of the Thigh." Dr. Savill, "Hysterical Contracture of nine years' standing treated by Hypnotism."

June 25th.—Dr. Waller, "The Action upon Isolated Nerve of Anæsthetics, Sedatives, and Narcotics." (Experimental and Lantern Demonstration.)

November 5th.—Dr. Wiglesworth, "A series of Idiots' Brains, illustrative of Maldevelopment, Porencephaly, and other Defects." Dr. Fletcher Beach, "Microcephalic Brains." Dr. Aldren Turner, "Tumour of the Gyrus Hippocampi, Two Cases of Tumour of the Corpora Quadrigemina, and Two Cases of Tumour of the Middle Cerebellar Peduncle." Dr. Mott, "Amyotrophic Lateral Sclerosis, and Combined Sclerosis associated with grave Anæmia." Dr. Beevor, "Progressive Muscular Atrophy." Dr. Bryan, "Tumour of the Spinal Accessory Nerve, with Histological Examination by Dr. Mott." Dr. Risien Russell, "Combined Sclerosis resembling that associated with Anæmia." Dr. Alexander Bruce, "Descending Degeneration in the Posterior Columns in the Lumbo-Sacral region of the Spinal Cord." (The Papers were all illustrated by Lantern Demonstrations, with the exception of that by Dr. Beach, and some also by macroscopic and microscopic specimens and casts.)

BALANCE SHEET, 1896.

Dr. **NEUROLOGICAL SOCIETY.** Cr.

RECEIPTS.				£	s.	d.
To Balance in hand	General Balance,	£82 19 2		120	15	2
	Sir W. Broadbent	£37 16 0				
To Subscriptions for current year				167	8	10
Do. in advance				3	0	0
Do. in arrears				5	0	0
Share of profit on "Brain"				36	5	1
				£331	10	1

EXPENDITURE.					£	s.	d.
By Stationery and Printing	Bale & Sons	£3	18	11			
	Pulman & Sons	11	14	2	15	13	1
By Illustrations—Danielsson		17	0	0			
Thévoz & Co.		13	2	0			
W. S. Griffith		8	8	0			
Briginshaw		20	10	0			
Bale & Sons		19	1	6			
Pearson		49	2	6	127	4	0
Microscopes—Baker					1	6	6
Hire of Rooms—Royal Medico-Chirurgical Society					3	3	0
Messrs. Macmillan for publishing "Brain"					97	12	6
Reporting—Mr. Creasy					3	7	8
Petty cash					3	16	8
Bank charges					0	3	9
Indexing "Brain"					29	8	0
Balance	Indexing "Brain" Fund	£8	8	0	49	14	11
	General Account	41	6	11			
					£331	10	1

CHARLES E. BEEVOR,
Hon. Treasurer.

Audited and found correct, Dec. 10th, 1896,

HOWARD H. TOOTH.
W. A. TURNER.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Correspouding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. Herbert Spencer, London.
Professor Wundt, Leipzig.

CORRESPONDING MEMBERS.

Professor Erb, Leipzig.
,, Goltz, Strasburg.
,, Grasset, Montpellier.
,, Hitzig, Halle.
,, Jolly, Berlin.
,, Luciani, Rome.
,, Munk, Berlin.
,, Obersteiner, Vienna.
,, Ribot, Paris.
,, Golgi, Pavia.
Dr. Weir Mitchell, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. Hughlings Jackson, M.D., F.R.S.
1887 Samuel Wilks, M.D., F.R.S.
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 Henry C. Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D.
1896 Alexander Hill, M.A., M.D.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

ORDINARY MEMBERS.

Elected.

1892 Alexander, Robert Reid, M.D., The Asylum, Hanwell, W.

1894 Andriezen, W. Lloyd, M.D., West Riding Asylum, Wakefield.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897.)

O.M. Barlow, Thomas, M.D., F.R.C.P., 10, Wimpole Street, W.

,, Bastian, H. Charlton, M.D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5.)

1897 Batten, Frederick E., M.D., M.R.C.P., 124, Harley Street.

,, Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, Beevor, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)

1888 Birt, Ernest, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1896 Bond, C. Hubert, M.D., B.Sc.Edin., London County Asylum, Banstead.

1892 Bowlby, Anthony A., F.R.C.S., 24, Manchester Square, W.

1889 Bradford, J. R., M.D., D.Sc., M.R.C.P., F.R.S., 52, Upper Berkeley Street, W.

O.M. Bramwell, Byrom, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 Bramwell, John Milne, M.B., C.M., 2, Henrietta Street, W.

1892 Bristowe, Hubert Carpenter, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. Broadbent, Sir Wm. H., Bart., M.D., L.R.C.P., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1895.)

1894 Brodie, T. Gregor, M. ., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 Brown, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 Brown, Sanger, M.D., 34, Washington Street, Chicago.

O.M. Bruce, Alex., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C. 1895.)

O.M. Bruce, J. Mitchell, M.D., F.R.C.P., 23, Harley Street, W.

,, Brunton, T. Lauder, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C. 1896.)

1895 Bryan, Frederick, M.B. Dunelm, M.R.C.P., The Asylum, New Southgate, N.

1895 Bryant, John Henry, M.D. Lond., L.R.C.P., Guy's Hospital, S.E.

1890 Bury, Judson Sykes, M.D., M.R.C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-5.)

1891 Cagney, James, M.D., M.Ch., M.R.C.P., 93, Wimpole Street, W.

1889 Carter, Robert Brudenell, F.R.C.S., 31, Harley Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Mitchell, M.D., M.R.C.P., 28, Pembroke Road, Clifton, Bristol.

O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 Colman, W. S., M.D., M.R.C.P., 22, Wimpole Street, W.

,, Corner, Harry, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

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1895 Davidson, Andrew, M.B., C.M., The County Asylum, Dorchester, Dorset.

1894 Dean, H. Percy, M.D., F.R.C.S., 84, Wimpole Street, W.

O.M. Donkin, H. B., M.D., F.R.C.P., 108, Harley Street, W. (C., 1897.)

,, Dreschfeld, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester.

,, Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Simons, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. Edmunds, Walter, M.B., F.R.C.S., 75, Lambeth Palace Road, S.E.

,, Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

,, Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

1897 Fricke, E., M.R.C.S., L.R.C.P., County Asylum, Prestwich.

,, Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (C., 1896; V.P., 1897.)

,, Gee, Samuel J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1890 Goodall, E. W., M.D., Eastern Hospitals, Homerton, E.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 Gotch, Francis, M.A., F.R.S., 11, Prince's Park, Liverpool.

1893 Grant, Dundas, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.

1895 Guthrie, Leonard, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 109, Harley Street, W.

O.M. Haycraft, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 Head, Henry, M.D., C.M., L.R.C.P., 6, Clarence Terrace, Regent's Park, N.W.

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, Horsley, Victor, F.R.C.S., F.R.S, 25, Cavendish Square, W. (V.P., 1896; C., 1892-3.)

1889 Huggard, William R., M.D., M.R.C.P., Davos Platz, Switzerland.

1896 Hulme, George Frederick, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 Humphry, Laurence, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlehem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; C., 1887-92.)

,, Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.B., M.R.C.P., 2, Brunswick Place, Brighton.

1892 Jones, Henry Lewis, M.D., F.R.C.P., 9, Upper Wimpole Street, W.

1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W.

1896 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

O.M. Langley, J. N., M.A., F.R.S., Trinity College, Cambridge.

1888 Lawford, J. B., M.D., F.R.C.S., 55, Queen Anne Street, W.

Lowenthal, Max, M.D., M.R.C.P., 123, Harley Street.

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896.)

1896 Mackintosh, Ashley W., Wellington House, Alford Place, Aberdeen.

1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.

1894 Martin, Sidney, M.D., F.R.C.P., 10, Mansfield Street, W.

1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 May, W. Page, M.D., M.R.C.S., 49, Welbeck Street, W.

1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. Mercier, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.

,, Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895.)

1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.

1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 84, Wimpole Street, Cavendish Square, W. (Sec., 1894.)

1894 Murray, George, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.

1889 Murray, H. Montague, M.D., F.R.C.P., 27, Savile Row, W.

O.M. Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.

,, Nicolson, David, M.D., M.R.C.P.Ed., Broadmoor, Wokingham, Berks.

1888 Niermeyer, J. H. H., M.D., Amsterdam.

,, Norman, Conolly, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 Ogilvie, Leslie, M.B., M.R.C.P., 46, Welbeck Street, W.

,, Oliver, Thomas, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. Orange, Wm., C.B., M.D., F.R.C.P., 12, Lexham Gardens, Kensington, W.

,, Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895.)

,, Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 Parkinson, John Porter, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.

1896 Paterson, Donald Rose, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.

1895 Pinder, George, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 Pitt, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.

O.M. Poore, G. Vivian, M.D., F.R.C.P., 30, Wimpole Street, W.

1887 Pringle, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.

O.M. Purves, W. Laidlaw, M.D., 20, Stratford Place, W.

1894 Ransom, William B., M.D., M.R.C.P., The Pavement, Nottingham.

1890 Rayner, Henry, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. Reid, E. W., M.B., University College, Dundee.

1893 Rennie, George E., M.D., 16, College Street, Sydney, N. S. Wales.

1895 Reynolds, Ernest S., M.D., M.R.C.P., 23, St. John Street, Manchester.

1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 Rivers, W. H. R., M.D., M.R.C.S., St. John's College, Cambridge. (C. 1897.)

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1892 Ruffer, Marc Armand, M.D., Cairo.

1889 Russell, J. S. R., M.D., C.M., M.R.C.P., 4, Queen Anne Street, W. (Sec., 1896.)

O.M. Sanderson, J. Burdon, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (V.P., 1895; Pres., 1897; C., 1886 90.)

1888 Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. Schaefer, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)

,, Semon, Felix, M.D., F.R.C.P., 39, Wimpole Street, W.

,, Sharkey, S. J., M.D., F.R.C.P., 2, Portland Place, W. (C., 1889-92.)

O.M. Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1895 Smith, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 Smith, R. Percy, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.

1895 Smith, Telford, M.D., B.S., Royal Albert Asylum, Lancaster.

1895 Spicer, William J. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.

1895 Stansfield, Thos. E. K., M.B., Claybury Asylum, Woodford, Essex.

1895 Stanley, Douglas, M.D.Edin., 9, Easy Row, Birmingham.

1892 Starling, Earnest Henry, M.D., M.R.C.P., B.S., 107, Clifton Hill, N.W.

O.M. Stewart, Sir T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, Stirling, Wm., M.D., D.Sc., Owens College, Manchester.

1897 Stoddart, Wm. H., M.B., B.S., National Hospital, Queen Square.

1895 Stout, G. F., M.A., St. John's College, Cambridge.

1887 Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

O.M. Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)

1889 Sutherland, Henry, M.D., M.R.C.P., 6, Richmond Terrace, Whitehall.

1888 Syers, Henry W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.

1891 Taylor, James, M.D., M.R.C.P., 49, Welbeck Street, W.

1889 Thorburn, Wm., F.R.C.S., Rusholme, Manchester.

1892 Titchener, E. B., B. A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896.)

1892 Trevelyan, E. F., M.D., B.Sc., 40, Park Square, Leeds.

,, Tuckey, Charles Lloyd, M.D., C.M., 14, Green Street, Grosvenor Square, W.

1892 Tuke, J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Square, Edinburgh.

1891 Tuke, Thomas Seymour, M.B., M.R.C.S., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.D., F.R.C.P., 13, Queen Anne Street, W.

O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.

1888 Voorthuis, J. A., M.D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)

1895 Wade, Arthur L., M.D., Somerset and Bath Asylum, Wells.

O.M. Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (C. 1894.)

1894 Walker, A. Stodart, M.B., 30, Walker Street, Edinburgh.

O.M. Ward, J., D.Sc., Trinity College, Cambridge

,, Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 Washbourn, J. W., M.D., F.R.C.P., Guy's Hospital, S.E.

O.M. Watteville, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95; Sec. 1886-9; Editor of *Brain*.)

O.M. White, W. Hale, M.D., F.R.C.P., 65, Harley Street, W. (C. 1897.)

1894 Whiting, Arthur J., M.D., National Hospital, Queen Square, W.C.

1889 Wigglesworth, Joseph, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.

1894 Williamson, Richard T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

O.M. Wilks, Samuel, M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)

1896 Wilson, Albert, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone,

,, Wood, Guy M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 Wood, T. Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1898,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1897.

LONDON:
JOHN BALE, SONS & DANIELSSON, LTD.,
85-87-89, GREAT TITCHFIELD STREET, OXFORD STREET, W.
1898.

Office Bearers for the Year 1898.

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VICTOR HORSLEY, F.R.C.S., F.R.S.

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W. H. GASKELL, M.D., F.R.S.
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C. A. BALLANCE, M.S., F.R.C.S.
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W. HALE WHITE, M.D., F.R.C.P.

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CHARLES EDWARD BEEVOR, M.D., F.R.C.P.

Editor (ex-officio).

A. de WATTEVILLE, M.A., M.D., B.Sc.

Secretaries.

F. W. MOTT, M.D., F.R.C.P., F.R.S.
J. S. RISIEN RUSSELL, M.D., F.R.C.P.

Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 13, 1898.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The Members now number 187.

The Inaugural Address was delivered at the Annual General Meeting on January 14th, 1897, by Dr. G. H. Savage, on "Heredity in the Neuroses."

A special feature in the year's programme has been the delivery of the first Hughlings Jackson Lecture on December 8th, 1897, by Dr. Hughlings Jackson himself, in whose honour the Lectureship was founded by the Society last year. The subject of the Lecture was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

A satisfactory arrangement has been made with Messrs. Macmillan & Co. in view of the increased cost of publishing *Brain*, as they have undertaken to hand over to the Society one-third of their share of the net profits from the sale of the Journal, as a contribution towards the cost of illustrations.

The Council regrets the loss sustained by the Society by the death of an illustrious Honorary Member, Professor Du Bois Reymond, of Berlin; also of Dr. James Cagney and Dr. E. Fricke.

The Proceedings have been published at greater length in *Brain* than in former years, and an abstract appended to this Report shows the character and value of the work of the Society during the past year.

Proceedings of the Society during the year 1897.

January 14th.—Dr. G. H. Savage, Inaugural Address, "Heredity in the Neuroses," *Brain*, vol. xx., p. 1.

March 4th.—Dr. F. E. Batten made a communication on "The Muscle Spindle under Pathological Conditions," and illustrated his paper by lantern slides and by specimens under the microscope. Professor Sherrington and Mr. Victor Horsley also made communications on the Muscle Spindle, and illustrated their remarks in the same way.

April 22nd.—Dr. Ferrier, "Two cases of Myoclonus Epilepticus." Dr. Donkin (1) "Probably Subacute Poliomyelitis in a man aged 45 years"; (2) "Tabes Dorsalis with a great Preponderance of Sensory Disturbance"; (3) "Pseudo-hypertrophic Paralysis in an Advanced Stage." Dr. Gossage, "Progressive Muscular Atrophy Commencing at the Early Age of 10½ years." Dr. Lunn, Dr. Beevor, and Mr. Ballance, "Case of Removal of a Cerebellar Tumour, two years and five months after the operation." Dr. James Taylor, "Syringomyelia with Bulbar Symptoms." Dr. Harris, "Syringomyelia with Supposed Hæmorrhage into the Cavity."

June 3rd.—Professor Schäfer, "Is there a Direct Relationship between the Motor Paralysis produced by Lesions of the Cortex and Loss of Sensibility, Muscular or other?" The paper was illustrated by Lantern Slides, three living monkeys and the brain of a fourth that had been killed. Professor Boyce and Dr. Warrington, "Contributions to the Anatomy of some of the Ascending and Descending Nerve Tracts in the Fowl." (Lantern Demonstration.)

October 28th.—Dr. J. A. Ormerod on behalf of Drs. Sanger Brown and Adolf Meyer, "Morbid Anatomy in a case of Hereditary Ataxy of the type described by Sanger Brown." (Illustrated by specimens under the microscope.) Drs. Samuel Gee and H. H. Tooth, "Case of Hæmorrhage in the Region of the Pons Varolii, almost entirely on the Right Side." (Lantern Demonstration.) Dr. W. A. Turner, "Symmetrical Softening of the Pyramids and Inter-olivary Strata." (Lantern Demonstration.) Dr. James Taylor and Mr. C. A. Ballance, "Case of Tumour of the Right Lateral Lobe of the Cerebellum." Dr. Risien Russell, "Degenerations in the Spinal Cord after Removal of a Large Tumour from one Cerebral Hemisphere, and in the case of a Tumour of the Lumbo-Sacral Cord."

December 8th.—Dr. Hughlings Jackson delivered the first Hughlings Jackson Lecture, the subject of which was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

BALANCE SHEET, 1897.

NEUROLOGICAL SOCIETY.

RECEIPTS.

	£	s.	d.	£	s.	d.
To Balance—						
General Balance ...	41	6	11			
Sir W. Broadbent ...	8	8	0			
				49	14	11
Subscriptions for current year				171	4	0
Do. in advance				1	0	0
Do. in arrears				12	0	0
Share of profit on "Brain"				46	10	4
Contribution by Messrs. Macmillan towards cost of illustrations in "Brain"				15	0	0
				£295	9	3

CHARLES E. BEEVOR, *Hon. Treasurer.*

EXPENDITURE.

	£	s.	d.	£	s.	d.
By Stationery and Printing—						
Pulman & Sons	7	15	3			
Bale & Danielsson ...	9	9	9			
				17	5	0
Illustrations—						
Bale & Danielsson ...	75	11	6			
Pearson	2	0	0			
				77	11	6
Microscopes—Baker				6	16	0
Hire of Rooms and Refreshments—						
Royal Medico-Chirurgical Society				12	12	0
Medical Society	21	7	0			
Fielder (Univ. Coll.) ...	1	7	6			
				22	14	6
Messrs. Macmillan for publishing "Brain"				102	14	3
Petty Cash				0	18	0
Bank Charges				0	4	6
Indexing "Brain"				8	8	0
Balance				46	5	6
				£295	9	3

Audited and found correct, December 17th, 1897. { J. B. LAWFORD. W. S. COLMAN.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. Herbert Spencer, London.
Professor Wundt, Leipzig.
„ Hitzig, Halle.

CORRESPONDING MEMBERS.

Professor Erb, Heidelberg.
„ Golgi, Pavia.
„ Goltz, Strasburg.
„ Grasset, Montpelier.
„ Jolly, Berlin.
„ Luciani, Rome.
„ Munk, Berlin.
„ Obersteiner, Vienna.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia,

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. Hughlings Jackson, M.D., F.R.S.
1887 Sir Samuel Wilks, Bart., M.D., F.R.S
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 Henry C. Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D., F.R.S
1896 Alexander Hill, M.A., M.D.
1897 George H. Savage, M.D.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected.

1892 Alexander, Robert Reid, M.D., The Asylum, Hanwell, W.

1897 Anderson, H. Kerr, M.B., B.Sc., 9, Park Terrace, Cambridge.

1894 Andriezen, W. Lloyd, M.D., Darenth Asylum.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897.)

O.M. Barlow, Thomas, M.D., F.R.C.P., 10, Wimpole Street, W.

,, Bastian, H. Charlton, M.D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5.)

1897 Batten, Frederick E., M.D., M.R.C.P., 124, Harley Street.

,, Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, Beevor, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)

1888 Birt, Ernest, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1898 Bolton, Joseph Shaw, M.D., B.S., B.Sc., Mason College, Birmingham.

1896 Bond, C. Hubert, M.D., B.Sc.Edin., London County Asylum, Banstead.

1892 Bowlby, Anthony A., F.R.C.S., 24, Manchester Square, W.

1889 Bradford, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 60, Wimpole St., W.

O.M. Bramwell, Byrom, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 Bramwell, John Milne, M.B., C.M., 2, Henrietta Street, W.

1892 Bristowe, Hubert Carpenter, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. Broadbent, Sir Wm. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1895.)

1894 Brodie, T. Gregor, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 Brown, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 Brown, Sanger, M.D., 34, Washington Street, Chicago.

O.M. Bruce, Alex., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C., 1895.)

O.M. Bruce, J. Mitchell, M.D., F.R.C.P., 23, Harley Street, W.

,, Brunton, T. Lauder, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896.)

1895 Bryan, Frederick, M.B.Dunelm, M.R.C.P., The Asylum, New Southgate, N.

1895 Bryant, John Henry, M.D.Lond., L.R.C.P., Guy's Hospital, S.E.

1890 Bury, Judson Sykes, M.D., M.R.C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-5.)

1889 Carter, Robert Brudenell, F.R.C.S., 31, Harley Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 Colman, W. S., M.D., F.R.C.P., 22, Wimpole Street, W.

,, Corner, Harry, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1894 Cuming, James, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.

1895 Davidson, Andrew, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 Dawson, William R., M.D., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

1894 Dean, H. Percy, M.D., F.R.C.S., 69, Harley Street, W.

O.M. Donkin, H. B., M.D., F.R.C.P., 108, Harley Street, W. (C., 1897.)

,, Dreschfeld, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P., 1898.)

,, Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Simons, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. Edmunds, Walter, M.B., F.R.C.S., 75, Lambeth Palace Road, S.E.

,, Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

,, Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

,, Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (C., 1896; V.P., 1897.)

,, Gee, Samuel, J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1890 Goodall, E. W., M.D., Eastern Hospitals, Homerton, E.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, Dundas, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.

1895 Guthrie, Leonard, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1897 Harris, Wilfred J., M.D., M.R.C.P., 7, Craven Mansions, Craven Terrace, W.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 Head, Henry, M.D., C.M., M.R.C.P., 61, Wimpole Street, W.

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, Hopkins, John, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896; C., 1892-3.)

1889 Huggard, William R., M.D., M.R.C.P., Davos Platz, Switzerland.

1896 Hulme, George Frederick, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 Humphry, Laurence, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlehem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.D., M.R.C.P., 6, Manchester Square, W.

1892 Jones, Henry Lewis, M.D., F.R.C.P., 9, Upper Wimpole Street, W.

1894 Jones, Robert, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W.

1896 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

O.M. Langley, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (C., 1898.)

1888 Lawford, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

,, Lewis, H. Wolseley, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1897 Lowenthal, Max, M.D., M.R.C.P., 84, Princes' Road, Liverpool.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, Mackenzie, Hector William Gavin, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. Mackenzie, Stephen, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896.)

1896 Mackintosh, Ashley W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1889 Macphail, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.

1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1893 Maude, Arthur, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 May, W. Page, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. Mercier, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.

Meyer, Adolf, M.D., Clark University, Worcester, Mass., U.S.A.

,, Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895-97.)

1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.

1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894, 1897.)

1894 Murray, George, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.

1889 Murray, H. Montague, M.D., F.R.C.P., 27, Savile Row, W.

O.M. Nettleship, Edward, F.R.C.S., 5, Wimpole Street, W.

,, Nicolson, David, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.

1888 Niermeyer, J. H. H., M.D., Amsterdam.

,, Norman, Conolly, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

,, Ogilvie, Leslie, M.B., M.R.C.P., 46, Welbeck Street, W.

,, Oliver, Thomas, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. Orange, Wm., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, Ord, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

,, Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 Parkinson, John Porter, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.

1896 Paterson, Donald Rose, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.

1895 Pinder, George, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 Pitt, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.

O.M. Poore, G. Vivian, M.D., F.R.C.P., 32, Wimpole Street, W. (C., 1898.)

1887 Pringle, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.

O.M. Purves, W. Laidlaw, M.D., 20, Stratford Place, W.

1894 Ransom, William B., M.D., M.R.C.P., The Pavement, Nottingham.

1890 Rayner, Henry, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. Reid, E. W., M.B., University College, Dundee.

1897 Reissmann, Charles H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.

1893 Rennie, George E., M.D., 16, College Street, Sydney, N. S. Wales.

1895 Reynolds, Ernest S., M.D., F.R.C.P., 23, St. John Street, Manchester.

1892 Richards, Joseph Peeke, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 Rivers, W. H. R., M.A., M.D., M.R.C.P., St. John's College, Cambridge. (C. 1897.)

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1892 Ruffer, Marc Armand, M.D., Cairo.

1889 Russell, J. S. Risien, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1897.)

O.M. Sanderson, J. Burdon, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres. 1897; V. P., 1895; C., 1886-90, 1898.)

1888 Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. Schaefer, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)

,, Semon, Sir Felix, M.D., F.R.C.P., 39, Wimpole Street, W.

,, Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1895 Smith, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 Smith, R. Percy, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.

1895 Smith, Telford, M.D., B.S., Royal Albert Asylum, Lancaster.

1895 Spicer, William T. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.

1895 Stansfield, Thos. E. K., M.B., Claybury Asylum, Woodford, Essex.

1895 Stanley, Douglas, M.D.Edin., 9, Easy Row, Birmingham.

1892 Starling, Ernest Henry, M.D., F.R.C.P., B.S., 8, Park Square, N.W.

1897 Stewart, Purves, M.A., M.D., M.R.C.P., National Hospital, Queen Square, W.C.

O.M. Stewart, Sir T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, Stirling, Wm., M.D., D.Sc., Owens College, Manchester.

1897 Stoddart, Wm. H., M.B., B.S., M.R.C.P., County Asylum, Prestwich, Manchester.

1895 Stout, G. F., M.A., St. John's College, Cambridge.

1887 Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

O.M. Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)

1889 Sutherland, Henry, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 Syers, Henry W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.

1891 Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W.

1898 Thomson, H. Campbell, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 Thorburn, Wm., F.R.C.S., The Limes, Nelson Street, Manchester.

1892 Titchener, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896.)

1892 Trevelyan, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

1892 Tuckey, Charles Lloyd, M.D., C.M., 88, Park Street, Grosvenor Square, W.

1892 Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

1891 Tuke, Thomas Seymour, M.B., M.R.C.S., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.D., F.R.C.P., 13, Queen Anne Street, W.

O.M. Tweedy, John, F.R.C.S., 100, Harley Street, W.

1888 Voorthuis, J. A., M.D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)

1895 Wade, Arthur L., M.D., Somerset and Bath Asylum, Wells.

1894 Walker, A. Stodart, M.B., 30, Walker Street, Edinburgh.

O.M. Waller, Augustus, M.D., F.R.S., 16, Grove End Road, N.W. (C. 1894-97.)

O.M. Ward, J., D.Sc., Trinity College, Cambridge.

,, Warner, Francis, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 Washbourn, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. Watteville, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896; Sec., 1886-9; Editor of *Brain.*)

1897 Weber, F. Parkes, M.D., M.R.C.P., 19, Harley Street, W.

1897 West, Samuel, M.D., F.R.C.P., 43, Wimpole Street, W.

1897 White, Ernest W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. White, W. Hale, M.D., F.R.C.P., 65, Harley Street, W. (C. 1897.)

1894 Whiting, Arthur J., M.D., M.R.C.P., 142, Harley Street, W.

1889 Wigglesworth, Joseph, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.

1894 Williamson, Richard T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

O.M. Wilks, Sir Samuel, Bart., M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)

1896 Wilson, Albert, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, Wood, Guy M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 Wood, T. Outterson, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 Woodhead, G. Sims, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

1898 Young, Robert Arthur, M.D., B.Sc., M.R.C.P., 37, Trevor Sq., S.W.