AN ADDRESS ON THE ORIGIN AND SEAT OF EPILEPTIC DISTURBANCE.

Delivered before the Cardiff Medical Society.

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THE question before us this afternoon is really a double one. We have to consider first the seat in the nervous system of the disturbance which we call an epileptic fit, and secondly the place of the origin of that disturbance. At the outset we must define clearly what class of fits or convulsions are referred to, for, as a few words would suffice to show, while the onset and progress of an attack of Jacksonian epilepsy are easy to trace and easy to follow, it is extremely difficult with our present knowledge to analyse the phenomena of a general or idiopathic epileptic fit. It is with the view of pointing out what has been done in this latter and obscurer division of the subject that I venture to address you to-day.

If we are to group epileptic convulsions into classes, I think it is possible to divide them into two great divisions:

1. Idiopathic or general epilepsy (*grand mal, petit mal*), convulsions of children, reflex epilepsy, etc.
2. Jacksonian epilepsy and hystero-epilepsy.

With the latter of these two classes we have nothing to do save more than by a passing reference. A glance at a tumour which invades the excitable area of the cortex cerebri, the position it takes among the various centres therein represented, and its precise limitations all show most clearly that both the seat and origin of the epileptic disturbance in such a case are sufficiently obvious, namely, the cortex cerebri. So, too, in hystero-epilepsy it is clear, I think, to anyone who studies closely that subject, that the phenomena in all their stages demonstrate their origin and seat to be the whole cortex of the brain. We may pass on therefore to the consideration of the first division. From this class we may separate those convulsions and fits which clinical observation has shown to be primarily caused by peripheral irritation, either in the alimentary tract or on any of the afferent channels of the nervous system, whether among the peripheral nerves or in the spinal cord. The mode of origin of these conditions is sufficiently obvious, but it must be admitted that it still remains for investigators a very fruitful field to discover the seat of the encephalic disturbance in these varieties of convulsions. Certain subordinate questions which have a special interest in this class and which are not completely answered in such a well-known condition as Jacksonian epilepsy, form also some of the most important problems in relation to the so-called idiopathic or general epilepsy, which, as I have stated before, is, in its pathology, by far the most obscure of all kinds of convulsions.

Turning now to the question as it presents itself to us for consideration, we must briefly review the phenomena which we observe in a patient who is so attacked. In the first place these cases are divisible into two chief varieties: (1) Those with an aura; (2) those without an aura. We will put aside for the moment those with an aura, because whereas in some cases the aura is very clearly of cortical origin—and that fact would tend to show that in such instances we have a hint as to what the origin of the disturbance may be—it is with the other class that we ought rather to deal, inasmuch as the attacks without an aura are the most difficult to solve (since we have in them no such suggestive symptoms) are as frequent, are as severe or more severe, and similarly lead to mental degradation. It is perfectly clear therefore that, humanly speaking, these are the worst forms of epilepsy, and as such constitute a very great evil to mankind, which it should be our business to try to remove.

The phenomena which mark an attack of this kind may be enumerated in the following order: (1) Semivoluntary move-

ment, for example, rising into a standing posture; (2) change in respiration, inspiratory spasm with cry and commencing asphyxia; (3) in the worst cases simultaneous with (2), loss of consciousness (instantaneous); (4) muscular spasms, tonic stage; (5) muscular spasms, clonic stage; (6) exhaustion.

To this enumeration must be added the facts that the asphyxial condition is very prolonged from its commencement, and that coupled with the early phenomena are also changes in the condition of the blood vessels, to which a great deal of importance used at one time to be attached, but, as this was based upon clinical observation, the deductions made involve such vast assumptions as to be worse than useless. Now the first point among the phenomena which attracts attention is that of the loss of consciousness. It is this item in the condition which beyond all others compels us to admit the depth of our ignorance in this most common clinical malady. I venture to approach it in the following way: First, what is the part of the nervous system upon the functional activity of which the state we call consciousness depends? Secondly, what agencies are capable of producing such an unconsciousness as is seen in the kind of epilepsy under discussion?

The first question may be answered fairly readily, for the clinical observations of Strümpell, and the still more wonderful extirpation experiments of Goltz, show that the cerebral hemispheres (that is, the cortical perceptive centres of the same) are the essential regions which contribute to the conscious state. It is, therefore, a warrantable assumption that the phenomenon of loss of consciousness must be brought about by some agency which abrogates the functional activity of the cortex cerebri.

This much agreed upon, the next point to consider naturally is—what agencies are there which can thus suddenly annihilate the activity of the cortex cerebri? These agencies may be divided into intrinsic and extrinsic. By an extrinsic agency we mean that if a narcotic substance be absorbed into the circulation and distributed in large doses to the cortex, by this means the function of the latter would fail in proportion to the dose of the agent; or if indeed the circulation were suddenly reduced in the brain below a certain point, the function of the latter would be interrupted (Vulpian and others). Those, in short, would be extrinsic agencies which, acting outside the nerve corpuscles that make up the nerve centre, would paralyse the activity of such elements. But it is conceivable that what we may call intrinsic and not extrinsic agencies may be at work. Thus by suitable excitation of a sensory nerve—for example, optic, auditory, etc., the functional activity of the cortex cerebri may be very profoundly depressed, as, for example, in the hypnotic state. It might well be, therefore, that the loss of consciousness in the general epileptic convulsion may be due not to circumstances operating outside the nerve corpuscles and channels, but to a sudden perversion of the activity of the nerve protoplasm.¹ At present the only thing we know that would produce such a condition is, as we have just stated, a strong afferent excitation; but, considering our profound ignorance on the subject, it is plain that we must keep our minds open for the reception of any information, however startling, as to the agency which produces this terrible phenomenon. To emphasise my point, I would remark that it has been the fashion to attribute the loss of consciousness to extrinsic agencies, and the most remarkable assertions have been made as to the ability of the circulation to produce the effect in question; but when we come to examine the grounds on which these assertions are based, we are astonished to find how feeble they are.

The experiments of Kussmaul and Tenner, upon which so much stress has been laid, and from which I believe many erroneous deductions have been drawn, consisted in the production of sudden anæmia by ligature of all the vessels leading to the encephalon, but as a matter of fact their experiments involved not merely anæmia as the cause of the convulsions but also asphyxia, since the effect of the ligature in

¹ I merely suggest this to indicate that, as the extrinsic anæmia which has for a long time been supposed to be the *vera causa* will have to be given up (*vide infra*) and ought never to have been brought forward, we must look further afield for the agent which evokes unconsciousness; but in making the above suggestion, I fervently hope it will not be regarded as a "theory" or hypothesis of the same value as an experimental fact.

question was to cut off the nutrition of the respiratory centre. A yet more fundamental criticism which should be applied to these experiments is that although they are very interesting and important, nevertheless the convulsions evoked in this manner are of quite a different type to those of the general or so-called idiopathic epilepsy. It is extraordinary how freely these observations have been quoted, and the most elaborate hypotheses built up by clinicians as to the action of the vasomotor centre in constricting the vessels of the brain, etc., without the simple fact that we are entirely ignorant of such action having been considered. For these two great reasons, therefore, it seems to me that though Kussmaul and Tenner's experiments probably throw some light on those extraordinary cases of sudden and brief convulsions which are from time to time recorded, yet as regards general epilepsy I do not think we are justified in basing any conclusions drawn from them. The chief argument which has been employed for the purpose of showing that epileptic fits are due to vascular changes in the brain is the ordinary clinical observation that the face is noticed to turn pale at the commencement of an attack. It is hardly credible that a theory of the causation of epilepsy should have been built upon such an observation, and that that theory should have been accepted in a very universal manner without a proper experimental examination, or, as it would seem, without a moment's consideration. Yet this nevertheless is the case, and I fancy that the idea is pretty prevalent that there is at the commencement of an epileptic fit anemia of the brain, and that this anemia is the cause of the convulsions. Yet if you were to ask a clinical observer what ground he had for assuming that, because he saw the face turn pale, the brain was also anæmic, he would have to admit that he had no such ground; nay more, we shall see directly that there is strong evidence against such a notion. Let me dismiss this subject by saying that it always appears to me that the construction of seductive hypotheses like this one is hardly less than a crime committed against science and medical knowledge, inasmuch as men's minds so readily travel on easy lines, that they will rest satisfied for a long time with an hypothesis which so readily explains everything, however baseless and even false it may be, and the result is a stagnation of scientific investigation and consequently of progress in that direction. You may say, Well, if this be so we cannot expect that many new facts have been ascertained till lately, directly concerning the origin of an epileptic fit; and that unfortunately is the case. What has been done I will now attempt to briefly lay before you.

We may best group the facts according as to whether they have been acquired by experimental investigation into the modes of artificially inducing convulsions or by investigations in which the conditions of the nerve centres has been directly examined during a convulsion. Taking the first category to begin with, the earliest and most far-reaching experiments were made by Nothnagel. This investigator, you will remember, came to the conclusion that the establishment of minute lesions in the medulla oblongata or pons evoked generalised convulsions, often tonic movement, sometimes clonic. But a consideration of the protocols of his experiments shows that the fits were not of the same character as those of general epilepsy, and although his experimental observations throw a great deal of light on convulsive seizures which start from mischief in the base of the encephalon, yet nevertheless they must be put aside as not directly bearing upon the general or epileptic fit. Moreover, of recent years these experiments have been repeated on a wonderfully extensive scale, and with the effect of greatly increasing our knowledge of this kind of convulsion by Professor Binswanger, who came to the conclusion that the movements in question were evoked by excitation, not of the central apparatus or of a convulsive centre such as had been theoretically supposed to exist either in the pons or in the bulb, but owed their existence to irritation of either ingoing or outgoing nerve channels.

A large series of experiments have been made by a number of workers by means of electrical excitation of the cortex cerebri since the great discovery of Hitzig and Fritsch and the observations of Ferrier. It is not necessary for me to do more than mention these researches, since the facts are so familiar to everyone. The conclusions to which they invariably lead are given further on.

The next category of observations are those in which the nerve centres are the subject of direct examination during a convulsive attack. There are very few ways in which a genuine epileptic fit can be artificially induced, but unquestionably by far the best is one which, after the earlier observations of Marcé, was introduced and investigated by Professor Magnan. This consists in injecting into a vein 1 minim of a solution of essence of absinthe. This substance has a most wonderful toxic effect on the central nervous system, and, as I shall show directly, only the encephalic portion of the neural axis. After the injection, a brief interval of from fourteen to twenty-five seconds elapses, that is, just enough time for the circulation to carry it round from the venous system up to the encephalon, and then there commences what every one would recognise as a typical general or so-called idiopathic epileptic fit. If the injection be made, for example, in a monkey, the animal goes through all the stages described above as constituting the phenomena of a fit in man. It is plain, therefore, that in this reagent we have a most valuable means at our disposal for carrying out a series of investigations which solve the great problem of the seat of the epileptic disturbance, its mode of origin, and certainly the condition of the nerve centres during an epileptic fit. As the latter point is one upon which the whole, and as I think I have shown untrue, hypothesis of the vascular causation of the epileptic convulsion was built, I may simply dismiss the point by stating that during the last seven years I have directly observed, in a large number of experiments in which typical fits were started by absinthe, the conditions of the vascular supply of the nerve centres, and it was then obvious that so far from being anæmic, they are actually hyperæmic. My observations on this point have recently been confirmed and extended by the valuable researches of Todorsky under the direction of Bechterew², who, not only by inspection of the cortex, but also by manometric measurement of the blood pressure in both the distal and central ends of the carotid, found that there was active hyperæmia of the cortical centres at the commencement and development of fits excited by absinthe or electricity. Fifteen months ago I had the opportunity of witnessing an epileptic fit in a patient at the time of an operation in which the "motor" region, so-called, was exposed and electrically stimulated. In that case, the cortex was by no means anæmic, but distinctly hyperæmic during the attack. Further, in some of the experiments with absinthe just alluded to, observation of the optic discs showed no notable pallor, and this fact was determined by Mr. Brudenell Carter, who very kindly examined the eyes during the fit. Of course, this is not an absolute proof of what occurs in the clinical condition of general epilepsy in man, but at any rate it shows that there is no ground whatever for the gratuitous clinical assumption that the brain is anæmic, and suggests as strongly as possible that it is, on the contrary, hyperæmic and turgid.

To proceed now to the attempt to discover by this means what is the position of the starting point of the epileptic convulsion. With whatever method we employ, it is plain that we must see how far it demonstrates to us the causation of the earliest phenomena or symptoms of the convulsion. Now I suppose that the earliest phenomenon after the semi-voluntary rising into a standing posture has occurred is the change in respiration; in other words, therefore, we should look to the respiratory centre as the indicating symptom, and that would lead us to think that the medulla is the point of the initial disturbance. This, in fact, was the view taken by Van der Kolk and others. But with the progress of experimental physiology we know that it is not legitimate to regard the fourth ventricle as the only seat of co-ordination of respiratory impulses, that as a matter of fact not only is there a direct representation of respiration in the cortex cerebri, but also in the spinal cord. The first question, therefore, which has to be decided is the following: Does the agency which produces the epileptic fit strike the whole of the neural axis at once, or only one part of it? To answer this question is no easy matter, although, as you see, it is the very basis of our knowledge; neither, curiously enough, has it been made the object of investigation, so far as I am aware, of more than very few observers—in fact, by the absinthe method, only two, Magnan and myself. It would be obvious to you that the

² *Neurolog. Centralblatt.*, 1891, 684

first thing which we should settle would be the participation of the spinal cord, or, in other words, whether the spinal nerve centres are capable of initiating the discharge of nerve energy, and so producing a convulsion of those parts of the body with which they are in relation. Magnan made an experiment of, first, division of the bulb, then induction of artificial respiration, and, finally, injecting absinthe; and he stated that he obtained a convulsion from the nerve centres below as well as above the point of section.

I have, however, repeated these experiments in various ways a large number of times during the last five or six years, and have always employed them in my class lectures with the absolutely constant result that if the sections of the spinal cord be complete, no fit can be evoked from the centres below the section, thus proving that although, in the complete attack, the spinal nerve centres are being used at least as conductors if not participators, they nevertheless have no power, when irritated by the absinthe, of initiating the convulsion, and it becomes therefore a definite conclusion that somewhere in the encephalon must be the starting point of the epileptic convulsion. But the encephalon includes the cerebral hemispheres, the cerebellum, the mid-brain, and the bulb or medulla oblongata—in which of these is the starting point we are in search of? I am not aware that this question can at the present time be answered at all positively; we need numbers of investigators to take up so obscure a point, and one that involves such considerable difficulties in the way of experimentation. I will not, therefore, waste your time by spinning theories on the subject, but draw your attention to the question of next importance—namely, What part of the encephalon is involved in the production of the full convulsion of the fit from its commencement, because that also partly includes the first and more difficult problem? The phenomena, examination of which has revealed within the last few years the facts we are in search of in a very striking manner, are (1) the loss of consciousness, (2) the change in respiration, (3) the cry, (4) the muscular spasms. I have already alluded to the now well-known fact that consciousness is dependent on the integrity of the cortex and the cerebral hemispheres, and does not appear to be the concomitant of the functional activity of the remainder of the encephalon. We are justified, therefore, in concluding that even if they be not the starting point of the convulsions, the hemispheres—that is, their cortical mantle, are affected very early in the convulsion, and in many cases where loss of consciousness, and, consequently, the fall, is the initial symptom, it is clear that the epileptogenous agent, if it does not commence its action therein, at least attacks the cortex at the same time that it does other parts.

Next, the change in respiration may of course be due to involvement of the respiratory centre in the medulla, but we must not lose sight of the fact that François-Franck, Lépine, Bochefontaine, Danilewsky, Goldstein, Sihler, Munk, Unverricht, Preolraschensky, Semon, and myself have found extensive representation of respiration in the cortex cerebri. Further, the cry is not to be regarded, as it formerly was, as an evidence of disturbance of the medulla oblongata, because although of course it can be evoked therefrom, as Vulpian and others have shown, by simple reflex stimulation of the bulbar nucleus of the fifth nerve, several authors, especially Ferrier, Duret, Krause, Semon, and myself have shown that phonation is also very extensively represented in the cortex cerebri. The disturbance, therefore, of these two functions of respiration and phonation may be due to changes in the cortex just as well as to changes in the medulla oblongata. Future investigation alone can decide the relative involvement of these parts. So far, therefore, it is clear that the cortex is probably the chief seat of the disturbance.

I come now to the consideration of the last fundamental symptom, namely, the muscular spasm; and had I not believed that the general discussion of the subject at this epoch of our knowledge was a very essential matter to lay before a society of this nature, I would have devoted the whole of the time at my disposal to discussing the origin and nature, the mechanism and kinds of the muscular spasms which form such a very prominent and distressing feature in an epileptic convulsion. For many ages clinicians have recognised that the muscular spasms, or even the general epileptic fit, are divisible into two stages, the tonic stage and

the clonic stage, the muscular contractions in the first of these being of a tetanic or continuous character, in the latter intermittent, or, according to the popular term, spasmodic. The order of events, therefore, as far as the muscles are concerned, is that of tonus followed by clonus. The question comes before us, What nerve centres in the neural axis are capable, when excited by any kind of agency, of producing this definite effect of tonus followed by clonus in the muscles they supply?

Supposing we isolate a portion of the spinal cord and stimulate that, for instance, with the induced interrupted current, we shall find that in the vast majority of instances the nerve centres apparently deliver a stream of energy which, although it may be intermitting at a fairly rapid rate, nevertheless appears in the main as a tetanic contraction or clonus, the component elements of which can sometimes be made out, and at any rate in the vast majority of cases it is not followed when the excitation is left off by any clonic stage. I say in the vast majority of instances, but reservation has to be made that this rule is not absolute; that in some animals, even of different species and under circumstances which at the present moment are not accurately understood, the spinal nerve centres continue to discharge their energy in an intermittent fashion after the original excitation has ceased; in other words, they exhibit what is known as a clonic after-effect.

In leaving the consideration of these lower centres, the lowest level of Dr. Jackson, I must insist once more on the fact that it is so difficult to obtain this after-effect from the centres of the bulb or spinal cord, that it is quite evident that the spinal apparatus is not the place whence come the invariably characteristic tonus and clonus of the epileptic convulsion. We are driven, therefore, to seek for it higher up, above the bulb, and on the authority of all observers we may exclude the nerve centres in the basal ganglia, the mid-brain and the cerebellum, and although I consider that these parts ought to be thoroughly re-investigated, yet I think you will agree with me that upon the evidence which I shall now lay before you, we are justified in concluding that the cortex cerebri is the seat of production of the characteristic muscular spasm in the general or idiopathic epileptic fit. Simple electrical excitation of the cortex in the so-called "motor" region has in the hands of most observers evoked this characteristic tonus followed by clonus in the muscles, the centre of which has been excited.

I am unable to discuss here the points raised by Ziehen, who considers that not only is the tonus produced by the lower centres and the clonus by the cortex, but also that minimal excitation produces clonus from the cortex. The latter contention, of course, is very possible, but in the hands of all observers an adequate excitation always evokes the two stages of tonus followed by clonus. Next, if we cut off the cortex, as was done by Franck and Pitres (confirmed by Schäfer, myself, Rosenbach, and others), and if we then excite the subjacent fibres of the corona radiata, we find that we no longer obtain this tonus and clonus but only the tonus. Moreover, when an epileptic fit has thus been evoked by electrical excitation and is in full progress, Munk found that instantaneous slicing off of the cortical grey matter immediately arrested the fit.

This evidence, which has been confirmed by all subsequent investigators including myself, we might be inclined to regard as conclusive proof that the cerebral hemispheres are the seat of the motor disturbance, but you must not forget that by this method of experimentation, that is observation and graphic record, we are dealing with the whole intact nervous system, not merely the cortex of the brain which we can excite and cause to discharge but also the nerve centres which are situated in the bulb and spinal cord, and which latter inasmuch as they have all the same general structure and likeness of function we may group together and speak of them collectively as the bulbo-spinal system or the "lowest level" (Jackson). We know that issuing from the spinal cord, the epileptic nerve impulses come to the muscles in this characteristic manner of first tonus and then clonus, but we do not know from the older methods of observation whether that character is really due to the encephalic centres, for example, the cortex; or whether the spinal centres are not exerting their influence in the production of the phenomenon. It

therefore has become absolutely necessary to obtain some evidence as to what kind of impulses come down the spinal cord during an epileptic fit, in order to pass out to the muscles. Unfortunately we have no means of getting hold of nerve energy, we have no physical instrument by means of which we can measure or record nerve energy in the same way that we can electrical energy, but most fortunately we have at our disposal, thanks to the genius of du Bois-Reymond, a method by means of which we know precisely when a nerve impulse is passing along a nerve fibre. As you know well, he discovered that when a nerve impulse passes along a nerve fibre an electrical change is produced in the electrical state of that nerve fibre. When nerve fibres are cut across or injured at one point the seat of injury becomes electro-negative to the rest of the nerve fibre, and thus an electrical difference is established between the parts of the nerve thus treated. Du Bois-Reymond found that if a single nerve impulse passed along that nerve, that that electrical difference would be diminished, and the little current which was set up by the passage of the impulse, and which by being of an opposite direction diminished the original difference was called by him the *negative variation*, but is usually known to other observers as the *action current*. If therefore we have in our hands an index of this kind it is reasonable to assume that if we could only tap as it were the fibres coming down the spinal cord from the brain, and connect them to a delicate galvanometer or electrometer that such a recording instrument would reveal the little action currents if such were produced by the epileptic nerve impulses streaming down the cord. Professor Gotch and myself have for several years employed this electrical method, as it may be termed, to investigate the passage of such and other nerve impulses in the spinal cord; we have succeeded in showing that the impulses which the cortex delivers and sends down the cord are of the characteristic form of tonus followed by clonus, and consequently we have now the proof that the "motor" part of the epileptic disturbance is seated in the cortex of the hemispheres.

Granted that this is so, we would naturally next inquire whether both hemispheres are involved in the epileptic disturbance. Observation of many clinical cases would seem to show that one hemisphere may start discharging a few seconds before the other; but in the majority of cases both are involved simultaneously, to judge by the incidence of muscular spasm in the limbs of both sides of the body.

This introduces us to the very large question of bilateral representation. The amount of attention that has been paid to this matter is very great, and a large number of observers (*vide infra*) have stated that if excitation be applied to one hemisphere and bilateral epileptic convulsions are the consequence, we are to deduce therefrom the view that one half of the brain is capable of throwing the muscles of both sides of the body into the characteristic condition of tonus followed by clonus. I have repeatedly shown that, whether we divide the commissures or whether we perform the converse experiment—namely, remove the excitable region of one hemisphere and then administer absinthe—the only effect is to produce the characteristic spasms in those muscles which are on the opposite side of the body in relation with uninjured cortex. Gotch and I have also approached the matter by the electrical method, and have shown² that in the cat, as well as in the monkey, unilaterality of representation prevails. Therefore we must conclude that, under ordinary circumstances, when the limbs of both sides of the body are in active convulsion both hemispheres are involved in the fit.

To sum up: Whatever be the point which the epileptogenous agency first attacks, we must conclude that the principal seat of the disturbance of a general or idiopathic fit must be the cerebral hemispheres, and especially their cortical mantle. Further that the condition of the cortex during the attack is one of congestion, and not anæmia; and finally that in all probability this portion of the encephalon is actually the place of origin of the disturbance.

² We have also, in the *Philosophical Transactions*, 1891 (Croonian Lecture), given the literature of this subject so far as it is known to us.

DR PAUL AUBRY has been deputed by the French Government to attend the International Congress of Criminal Anthropology, which, as already announced, is to be held at Brussels in August, 1892.

GOULSTONIAN LECTURES ON THE CHEMICAL PATHOLOGY OF DIPHTHERIA, COMPARED WITH THAT OF ANTHRAX, INFECTIVE ENDOCARDITIS, AND TETANUS.

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LECTURE II.

Examination of the Nervous System.—All parts of the nervous system were examined. The motor nerves were cut out with a piece of muscle attached, so as to admit of the examination of the intra-muscular branches. The only method which is of value in the study of peripheral nerve degeneration is staining the fresh nerve with osmic acid, so that the white substance of Schwann is stained black, and counterstaining with carmine to bring out the nuclei and axis cylinder. In all the animals the same changes were found in the nerves, and the following description of the pathological condition refers to specimens taken from the animals as illustrating the points to be referred to.

Motor Nerves.—After the motor nerve with the piece of muscle attached has been stained in osmic acid, it is separated from the muscle with its intramuscular branches as far as possible. This is a photograph of one of the intramus-

Fig. 1.—Nerve to triceps.

cular branches to the triceps muscle. In this small intramuscular nerve mounted whole as a specimen, it is seen that the nerve is stained black with osmic acid, except at two points just before it divides into two branches. At these points the nerve is attenuated and degenerated. This serves as a type of the degeneration in the nerves. All the parts near the origin of the nerve were quite normal. When the nerve is teased with needles from the normal part to the end of the small branches, it is found that at the upper part the fibres are normal; a little lower down they show a breaking up of the white substance of Schwann, which is becoming thin and is but little stained by the osmic acid, while lower down still the white substance disappears altogether. This is at the portion of the nerve unstained by osmic acid. Beyond this the white substance again appears, but it is very pale and granular, and broken up; lower down still it regains its dark colour. The primitive sheath of the nerve is intact throughout; the axis cylinder