THE LUMLEIAN LECTURES
ON
CONVULSIVE SEIZURES.

Delivered before the Royal College of Physicians of London.

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Lecture II.

Epileptiform (Middle Level) Fits.—Varieties.—Diluted Convulsions.—Crude Sensations.—Locality and Order of Convulsive Movements.—Degrees and Ranges of Fits.—Hyper- and Hypophysiological States.—A Discharging Lesion.—The "Buttoning Centre."—Physiological Fulminate.—The March of the Convulsion.—Compound Order.—Cells of a Discharging Lesion Quasi-parasitical.—Nutrition of Cells.—Summary.

I take for most particular consideration the second kind of fits, epileptiform seizures (middle level fits); they were first described by Bravais in 1824. There are many reasons why we should study them first. (1) Their investigation is comparatively easy. We can, if present at a paroxysm, ascertain the place of onset and trace the March of the convulsion; and if we witness no attacks, since consciousness is not lost or is lost late in the seizures, the patient can tell us the place of onset and much more about his fits. (2) They have permanent paralytic counterparts in monoplegias and hemiplegias from destructive lesions of the "motor region." (3) There is after them, at least in many cases, temporary paralysis of the parts first and most convulsed. (4) There is not rarely permanent paralysis of the parts which are occasionally convulsed, this showing—a very important thing with regard to doctrines of localisation—that in the same case there is coexistence of a destructive lesion and a discharging lesion of different elements of the same centre. (5) Because there is after some seizures temporary defect of speech ("partial aphasia"). (6) Because, if we can find the condition of centres answering to paralysis and aphasia after these seizures, we shall be greatly helped in our investigation of the state of the highest centres in insanities after epileptic fits. (7) Because epileptiform seizures often depend on gross organic disease, such as tumour (syphilitic or non-syphilitic); hence, we may at *post-mortem* examinations obtain proof or disproof of any notions on localisation and gross pathology we had from a study of the patient's symptomatology during his life. (8) It is in epileptiform seizures that operations have been done by Macewen, Godlee, Horsley, Barker, and others. Hence, very precise study of fits of this kind is necessary. In this regard, as well as in many others, the minute investigations of the "motor region" by Beevor and Horsley are of great value.

It would be very unmethodical to begin the scientific study of the more complex (epileptic) kind of fits before the study of the epileptiform kind.

There are Varieties of epileptiform seizures. The convulsion begins in some part of one side of the body. Varieties are distinguished by the particular place of onset ("signal symptom" of Seguin) of the convulsion. The fit may begin in some part of either right or left side of the body; I call that on which it does begin the "first side," and the corresponding (opposite) half of the brain in which the discharge begins the "first half." If the fit begins on the left side, that is the first side, and the right half of the brain is the first half; suppose that the convulsion becomes universal, then the right side of the body is the second side, and the left half of the brain (if it discharges after the right) is the second half. Whether the onset be right or left sided is a very important matter, because there is often defect of speech after right-side-beginning fits; moreover, on account of the "speech centre," operations on the left half of the brain are more serious than operations on the right.

The three commonest Varieties of epileptiform seizures are—(1) Fits starting in the hand (most often in the thumb or index finger or in both). (2) Fits starting in one side of the face (most often near the mouth) or in the tongue, or in both these parts. (3) Fits starting in the foot (nearly always in the great toe). (Of course one always means convulsive development of movements of muscles belonging to the several parts mentioned.)

Of necessity the three Varieties depend on the fact that the local discharging lesion is of cells of different parts of the "motor region"—of hand, face, and foot centre respectively. The starting point is almost invariably the same in each patient, but not always. A patient whose fits commonly begin in the hand may sometimes have the face of the same side slightly and solely affected; or a patient may tell us that his fits "fly about"—that is, leaving the face for the hand, or *vice versâ*.

We sometimes meet with paroxysms of one-sided tremor—what looks superficially like tremor, really a "diluted convulsion"—dependent, I think, on discharges beginning in motor elements of the so-called sensory centres behind the motor region.[1] I do not call these epileptiform seizures.

Often enough there is "tingling" or some other crude sensation in the place of onset before convulsion starts; this is part of the proof that the so-called motor region, although mainly motor,[2] is not purely motor. Sometimes there is at or near to the onset of an otherwise ordinary epileptiform seizure excessive development of colour, or of sound; these also are Crude Sensations. Epileptiform seizures with crude sensations deserve very careful attention, but time will not allow me to consider these complications.[3]

It is worth mentioning that some patients have a feeling as if a part were convulsed when it does not really move; one of my patients subject to veritable convulsions beginning in his left thumb had sometimes what he called "convulsions not to be seen" of that part. Another patient had the feeling of convulsion of one side of his face, but looking in a glass he saw that it did not move. He died of brain tumour (necropsy), but its exact position I did not learn. A patient who has fits starting in the hand will say that he feels "it" in the face, when, however, the face does not move; this "it" may be owing to very slight discharges of sensory or motor elements, or of both.[4]

Epileptiform fits begin in the Animal parts of the body, and commonly in those of them which are the most animal. Thus the majority begin in the arm, and nearly all these in the most animal part of that most animal part, the hand, and most of them in the thumb or index finger or both—the most animal parts of the whole organism. Perhaps the term animal is awkward in this connection; it is convenient in contrast to organic. We can, however, use other terms, and say that epileptiform seizures most often begin in those parts which, speaking popularly, have the most "voluntary" uses; in those parts which have great independence of movement; in those parts which have the greater number of different and more special (definite) movements, at the greater number of different intervals. The foot in man has, however, few *different* movements; the hallux in him is much less specialised than in the monkey. It is, then, a very striking thing that epileptiform seizures beginning in the leg almost invariably start by spasm of the great toe, in the most, and yet but little, differentiated part of the whole lower limb.

It may be objected to the principle stated that, to take an example, convulsion in an epileptiform seizure may begin in the shoulder, the least "voluntary" part of the arm, and spread down the limb.[5] I have considered this objection in the Journal of August

[1] I have never believed that any part of the cortex is purely motor or purely sensory. If the elaborate visual projections in some cases of migraine occur during discharge beginning in a "sensory centre," then the elements of that so-called sensory centre are only chiefly sensory; the fortification outline and the vibrations of parts of the migrainous visual spectre imply discharge of motor elements as certainly as the colours imply discharge of sensory elements.

[2] See remarks (Lecture I) on France's *Researches on Degeneration of Fibres of the Lateral Column of the Cord, consequent on Lesions of the Gyrus Fornicatus.*

[3] Of course crude sensations (psychical) and convulsion (physical) are in no way comparable: the comparisons and contrasts are of excessive discharges of sensory elements *during which* crude sensations arise, with such discharges of motor elements *from which* convulsion arises.

[4] When a healthy person thinks of doing something ("has an idea of a movement"), I consider that what occurs physically is slight discharge not only of sensory but also of motor nervous arrangements of his highest centres; there is "nascent movement"—slight discharge of the very same motor nervous arrangements of the highest centres which, if more strongly discharged, would, by intermediation of middle and lowest motor centres, produce the actual movement. It is probable that a similar explanation (the middle motor centres being here primarily concerned) applies to the "ideas of convulsion" spoken of in the text. No doubt if a man subject to fits beginning in his right thumb were to lose the right arm by amputation his fits would still *seem* to him to begin in his thumb—to begin in his spectral thumb, for some time at least.

[5] I have recorded a case of this kind, *Medical Times and Gazette*, June 5th, 1875.

17th, 1889. We have, as I continually insist, in strictness to speak not of representation of parts of the body (muscular regions), but of representation of movements. Movement of any part of the body done with intention is a "voluntary" movement. Evidently when a card-sharper shrugs his shoulder as a sign to a confederate to play a trump, that is his then most voluntary movement. In so far as any part of the body has a movement independent of the rest, in so far that movement has, I suggest, a degree of localness of representation in the middle and highest motor centres. Horsley and Beevor assign a point of the "motor region" for the primary movement of each segment of the upper limb. It may, however, be taken that epileptiform seizures most often begin in the most "voluntary" parts, and that admitting many varieties the three commonest are selected for comment.

Epileptiform seizures illustrate Dissolution—dissolution in process of being effected—in which the order is from "voluntary" towards automatic. *In normal development of movements the order is the opposite—is that of Evolution; it is from automatic to "voluntary."*[6]

The "motor region" (motor province of the middle level) presumably represents at least motorily the whole of the body, demonstrably nearly all parts organic as well as animal. Hence, as the discharging lesion may theoretically be of any part of the "motor region," it is a legitimate hypothesis that there are fits starting by excessive discharges of cells of nervous arrangements of the "motor region" which especially represent parts in the organic field, and that some fits called *epileptic* are such seizures. I do not hold that hypothesis. There are parts—for example, the vocal cords—which we may speak of as organico-animal.

Munk and Krause[7] in the dog, and Horsley and Semon in the monkey, have investigated the cortical representation of the movements of the vocal cords. Krause in the dog, Horsley and Semon in the monkey,[8] produced convulsive seizures by excitation of the cortical "laryngeal centre." Semon thinks that laryngismus stridulus in man (the infant human being) is a cortical fit; but if so there is not an exception to the statement that epileptiform fits begin in the animal parts, if we adopt Semon's views of, and inferences from, the kind of representation of the vocal cords in the motor cortex ("purposive or volitional, since it is adduction, that is phonation").

Although epileptiform seizures most often begin in the three most animal parts of the animal parts mentioned, yet as the fits go on the organic field becomes involved. In a dog poisoned by curara (respiration being artificially kept up) the animal parts are excluded, and then we see that by excessive discharges experimentally induced in its "motor region," effects are produced in, and are then limited to, the organic field.

There are Degrees of each variety corresponding to degrees of the "severity" of the excessive discharge. Degrees are to be considered with respect to two factors, (1) amount of convulsion, and (2) range of convulsion. I illustrate here quite arbitrarily by Range only, and take the case of an epileptiform fit starting in the thumb. Admitting that there are many ranges, it is allowable to make arbitrary divisions (they must not be taken for real distinctions) into four Ranges: (1) Terminal[9] fit—the spasm involves, say the hand, or some part of it only; (2) monospasm—the arm becomes involved; (3) hemispasm; (4) bilateral convulsion—the second side being gained, the fit becomes universal. Observe the use of the word "become." In the range (4) there is a March of spasm from the part first seized all over the body, answering to increased spreading of the central excessive discharge beginning in some particular part of the motor region of the first half, and may be extending to the second half, of the brain. The second side of the body (in 4) is affected later, and commonly less than, the first side.

From (1) to (4) there is but a single variety of epileptiform seizure in four different degrees. Otherwise stated, a man who has had convulsion of the range (1) or (2) does not become subject to another variety of epileptiform seizure when he has also fits which, beginning in the same terminal part (hand, we are supposing), have a more extensive range however much more extensive it is; so to speak, that patient is subject to *a* fit, and has it in different degrees on different occasions. However limited or however wide the range of convulsion, the corresponding discharge begins in the very same centre of the motor region of but one, the opposite half of the brain. This means that it is the place of onset ("the signal symptom" which localises)[10]; it points to the particular part of the "motor region" of the cells of which part the discharging lesion is made up. When a man's epileptiform fits "get worse"—when we are supposing they become of greater range—there is the same discharging lesion, but it has become of more cells, or the cells of it have become still more highly unstable, or perhaps there are both kinds of change; in consequence of this purely local ingravescence, the induced discharges (compelled discharges) of normal stable cells are more numerous.

Were A—and B—to be universally convulsed, they would have different varieties of epileptiform seizures if in A—the onset was by spasm of the hand, and in B—by spasm of the foot, however much alike the fits looked at the acme of each; the discharging lesion in A—would be of some cells of his "arm centre," in B—of his "leg centre." To note the "signal symptom"—to use Seguin's term again, and therefrom to infer the seat of the discharging lesion—is more important, obviously for "brain surgery," than anything else about the paroxysms.

It has been implied, but it may be well to say explicitly, that an epileptiform seizure does not, when become universal, turn into the other *kind* of fit—the epileptic. I mention this because I think a rapidly universal epileptiform seizure superficially resembles an epileptic one, and a slowly developed epileptic seizure superficially resembles an epileptiform seizure.

I have often urged that the Clinical Problem in every nervous malady is of three elements: (1) Anatomical, (2) Physiological, (3) Pathological. In other words, we have in every case to seek: (1) the seat of the lesion, the structure damaged, (2) to infer the kind of functional change of structure, and (3) to discover the nature of the abnormal nutritive process of which the alteration of composition of nervous matter producing the abnormal functional state of structure is effected (*vide infra*).

It may be properly said that structure, function, and nutrition never exist separately. It is quite obvious, taking for comment normal conditions, that there would be no persistence of function without continuous nutrition; that function is not conceivable apart from some structure, and that structure without function is nothing for an organism. It is, however, convenient, if not necessary, for clear exposition of nervous maladies, to consider for a time each of the three separately. It will, however, be impossible to consider one without frequent explicit and always implicit reference to the others. Perhaps I use the term Anatomy regarding the nervous system in an unusual sense; I do not use it convertibly with Morphology. A knowledge of the Anatomy of any Centre is *a knowledge of the parts of the body which that centre represents, and of the ways in which it represents them.*

Anatomy.—(Localisation.) I shall say very little under this head. The patient A. is subject to fits, every one of which begins in his right thumb; there is a lesion of the thumb centre of the left middle level ("motor region")—this is the anatomical diagnosis. (According to Beevor and Horsley, "the middle third of the ascending parietal convolution is the focus of representation of the movements of the thumb.")

Physiology.—Physiology deals with the dynamics of the organism—that is, with its function. I use the term "function" with regard to nervous diseases in a strict sense, and never in the way it or its adjective is used when applied to the symptoms of a hysterical woman, or to minute or transitory changes of structure.[11] I must now define the term. The function of organic matter and *par excellence* of nervous matter, to which I confine further remarks, is to store up energy,[12] to liberate that energy (nervous discharge) at different rates against resistances of different amounts; the energy liberated is dissipated or does work, or

[6] Properly from most automatic to least automatic in evolution; the opposite in dissolution. "Most voluntary movement" is objectionable because it is a mixture of psychological and anatomico-physiological language; it is a popular expression, equivalent to what is scientifically "least automatic movement."

[7] *Sitzungsberichte der Königlichen Academie der Wissenschaften und Archiv für Anatomie und Physiologie*, 1883.

[8] JOURNAL, December 21st, 1889.

[9] It may be well said that a fit beginning by spasm of the cheek does not begin in a *terminal* part; but the meaning of the word terminal may, for my present purpose, be used to include that onset. Peripheral would have too wide a meaning. I would suggest acro-epilepsy as a name for epileptiform seizures were it not that it might mislead some to think that there are epileptiform seizures which begin in the nose.

[10] The term aura is, I suppose, never used now in its original sense. When a medical man speaks of an aura in an epileptic fit starting from the epigastric region, he is understood to mean that some sensation is referred by the patient to that part of the body, and to believe that the sensation arises during the incipience of a discharge of some central sensory elements.

[11] See an able paper by Dr. Allchin, *Westminster Hospital Reports*, vol. ii.

[12] Assimilation of material having potential energy (anabolism); decomposition of material (katabolism) with liberation of energy.

there are both consequences according as the resistances encountered are or are not overcome, or are only partially overcome. We have here to deal with abnormalities of this function—that is, with abnormal physiological states (I need not always add the adjective abnormal).

There are two kinds of physiological or functional states in cases of disease of the nervous system. (1) Function may be exalted, and is, sometimes, as that of cells of a discharging lesion, very greatly exalted; these are superpositive functional changes—that is, hyper-physiological states. (2) Function may be diminished or lost; these are negative physiological or functional states—they are hypo-physiological states. Destructive lesions come in this category, although there is in them loss, not merely of function, but of functionable material also.

The two kinds of functional changes—the hyper- and the hypophysiological—are opposites; there are no degrees from one to the other; so to say, they depart from normal function, the one upwards, the other downwards. We shall have to speak later of a negative functional change in temporary post-epileptiform paralysis; that negative change (there is temporary exhaustion of nervous elements) is a direct result of the excessive discharge in the prior paroxysm; the two opposite functional states of the same nervous elements occur in immediate sequence.

Now I consider the inclusion of negative functional states in cases of mere temporary exhaustion of nervous elements in the same category with destructive lesions when nervous elements and function are gone together. I dare say the former would be called functional, because the alteration in the composition of the material of nervous elements, whatever its nature, is no doubt slight and is certainly temporary. But I call it a negative functional change, *because function is lost:* the slight and temporary alteration of composition is a pathological change. For the time being fibres exhausted are not in effect nerve fibres at all; with a morphological plenum there is a functional vacuum as certainly as there is when they are destroyed. Whether nervous elements are functionless or gone altogether, the situation *for the time being* is the same. I shall speak of both as "negative lesions." And saying that the exhausted fibres recover soon is recognising fully the vast difference in their condition from that of fibres destroyed —properly, absence of fibres.

There are degrees of negative function from defect to loss. Of hese I will say nothing. There are two greatly different degrees, if indeed we may not say two kinds, of superpositive (hyperphysiological) function. We are concerned with both in this inquiry; it is imperative to distinguish them clearly.

There is (*a*) the vast exaltation of function of cells of a discharging lesion; this degree is always produced by some pathological process—that is, by morbid nutrition, a nutrition which alters the composition of nervous matter.[13] This, the first degree of functional change, is of a highly explosive character; it issues in paroxysmal discharges.

There is (*b*) a comparatively slight exaltation of function with which we shall be particularly concerned when we consider post-epileptiform conditions. In these conditions, besides paralysis answering to a negative functional state of fibres of the second segment of the kinetic route, there are increased tendon reactions answering to a hyperphysiological state of this degree (*b*) of cells of anterior horns (lowest motor centres). I believe that the second degree of superpositive functional state is not—certainly not in the case just alluded to—the direct result of a pathological process, but that it is the *indirect* result of a negative functional state of *other*, related nervous elements. (Here is, essentially, an application of a principle long ago put forward by Anstie and Thompson Dickson). This, the second degree of functional change, does not issue in paroxysmal discharges, but in continuous discharges; or discharge can at any time be evoked by appropriate slight excitations. Returning to post-epileptiform conditions, the negative functional state answering to the paralysis is alone produced by a pathological process; the abnormal condition of anterior horns implied by increase of the tendon reactions is one of over-activity of perfectly healthy nervous elements. There is simply the proper activity of certain lowest motor centres, which is manifested at its maximum when an obstacle has been removed, when control has been taken off. I think, too, that cerebellar influx is no longer antagonised (see Lecture I); and if so, the cerebellum is simply unhindered in its activity, and is doing, now that an obstacle is removed, what it was always "trying to do." Here, again, we come across the principle that parts of the symptomatologies of nervous maladies are owing to activities or over-activities of perfectly healthy nervous arrangements. If this principle be valid, it is evidently important to distinguish between the physiological and the pathological factor in nervous maladies. Of course increased activity of any nervous elements implies increased nutrition, but there is not, I submit, in the case of elements answering to the second kind of functional change, morbid nutrition. There is not, then, what I call a pathological process; the composition of the matter of the cells concerned is not altered. For the present I shall speak only of the first degree (*a*) of superpositive functional states—that which is of a highly explosive degree—and is produced by a pathological process.

The hyperphysiological state (degree *a*) in the case of A—(*vide supra*) is what I call a "discharging lesion." It is a crude hyperphysiological state just as its diametrical opposite, loss of function, is a crude hypophysiological state. I continue to speak of healthy cells as stable; they are naturally unstable, of course, but I mean by using the term stable that they are so *in comparison with* those of discharging lesions, which I speak of as being *highly* unstable. The discharging lesion is of a few cells which have got far above the rest of the cortical cells in degree of tension and instability of equilibrium. That lesion is made up of cells of nervous arrangements which represent some most special movements of a particular muscular region; the sudden and excessive development of these movements from discharge of those cells is the convulsion incipient ("signal symptom" of Seguin), or, if there be no spreading of the spasm, it is the convulsion total.

The hyperphysiology of epileptiform seizures is the element of the threefold clinical problem of which we are sure. We are quite certain that normal movements are the results of liberation of energy attending katabolism; thus the inference is irresistible that that sudden and excessive development of many movements at once, which we call convulsion, must be a result of an excess of the same physiological process—of a sudden and excessive energy-liberation attending great and rapid katabolism. I will give illustrations of the different effects of discharges of stable cells (cells of comparatively low tension and of comparatively stable equilibrium) in normal operations, and of those of cells of a discharging lesion (cells of very high tension and very unstable equilibrium) in a convulsion.

It would be most absurd to say that there is a Buttoning Centre, but for convenience of illustration we may imagine such a centre. I illustrate by motor elements only, although, of course, sensory elements are concerned in all operations. In the operation of buttoning there are slight and slow discharges of normal stable cells (fibres are understood) of different nervous arrangements of our supposed centre (lower centres and muscles being understood) simultaneously and successively; in consequence there is a harmony and a melody of the different movements of the hand and arm by which the button is got into its hole. There is Harmony by Contemperation[14] of different movements, and there is Melody in that the compound contemperated movements follow one another at proper time intervals; there is what is commonly called co-ordination, although the time element (melody) is not much considered in most accounts of the process of co-ordination. So much for the discharges of stable cells in the operations of health.

Now let us suppose that the cells of the buttoning centre become by some, any, pathological process, so highly unstable as to constitute a discharging lesion. (For the present we shall speak as if the excessive discharges of the highly unstable cells did not provoke similar discharges of stable cells of collateral centres.) The discharges are sudden and rapid,[15] and of all the cells nearly

[13] This is the definition I would suggest of a pathological process (morbid nutrition) or of its result. I suppose that such poisons as strychnine alter the composition of nervous elements, in consequence of which morbid nutrition (a substitution nutrition) there is a functional change of the degree (*a*). It seems more likely that strychnine enters into the very composition of matter of nerve cells than that it stands outside the cells and "irritates" them. When a rabbit's respiratory centres discharge excessively on withdrawal of blood, one must suppose that before that discharge there is an abnormal metabolism (pathological process) whereby matter of the cells of the centre becomes more explosive. Certain "pathological processes," such as cerebral hæmorrhage, are really injuries; hemiplegia resulting from clot is, so far as destruction of fibres goes, like that caused by the prong of a pitchfork; there is no real pathological process in either of these modes of destruction.

[14] Contemperation is, says the *Imperial Dictionary*, an obsolete word. One of its meanings given *op. cit.* is, "The act of reducing a quality by admixture of the contrary."

[15] I do not mean by using the word rapid, here or anywhere, that nerve impulses travel more rapidly in the case of excessive discharges, but that more occur in a given time than is normal—that there is a greater quantity of motion in a given time.

at once; they do not cause a speedy and vigorous act of buttoning, but a short, severe, and rapid contention of all the movements of that operation; this contention is convulsion of the hand and arm. There is no harmony and melody; on the contrary, in the tonic stage, all contemplation is lost and all time-intervals are merged, so that there is but one rigid state of the musculature of the hand and arm, and, in the clonic stage, there is but a succession of such rigid states. There are no movements properly so-called in this convulsion; but, if I may use the word for once, there is first but a single big useless movement, and next a series of such so-called movements which do nothing but "mark time."

The illustration, although the supposition of a buttoning centre is grotesque, may render clear what presumably occurs when A— is "attacked by his convulsion." The convulsion is a brutal development of that man's own movements. I make this odd remark because I do not think that it is always vividly realised that an ordinary severe epileptic "attack" (to take that kind of fit for a moment's illustration) is nothing more than a sudden excessive and temporary contention of very many of the patient's familiar normal movements—those of smiling, masticating, articulating, singing, manipulating, etc. A convulsion is not something altogether *sui generis*. Speaking figuratively and more generally, and still of an epileptic paroxysm, there is the mad endeavour of the highest centres to develop the maximum of function of every part of the body, animal and organic, and of all parts at once; the phenomena of a very severe epileptic fit show that this endeavour is nearly successful; the patient is almost killed by the paroxysm, and is nearly dead (deeply comatose) after it.

The cells of a discharging lesion are not to be thought of only as occasionally discharging excessively; we have also to consider their aspect of too easy dischargeability, otherwise their highly unstable equilibrium. In health the cells of the "buttoning centre" are made to discharge slightly on special excitations of definite force, so that their discharges are in particular relations, and in some degree of community with those of cells of other centres (I will call them "collateral stable cells") with which they are connected. The cells of the "buttoning centre," when they have become highly explosive, are still integral parts of the nervous system, and have the very same connections they had before some pathological process so altered their nutrition that they became highly explosive (and then, metaphorically speaking, "mad parts"). Their equilibrium can be upset, they can be made to discharge, and to discharge excessively, by less special excitations of less definite force coming to them from collateral stable cells—that is, their discharge depends on their own easy dischargeability rather than on the particularity of the excitation reaching them. Possibly their discharge is spontaneous when tension is very great, and equilibrium very highly unstable.

I repeat that the cells which have become very highly "explosive" (those of the discharging lesion) continue to be elements of the same nervous arrangements, and that these nervous arrangements are still connected in the very same ways with the remainder of the nervous system[16] as when their cells were stable. Hence I do not speak of the highly explosive cells as being *in* any motor centre, but as being *of* that centre. I submit that the highly "explosive" cells of a discharging lesion will on their fulminating discharge overcome the resistance of, and thus produce excessive discharge of, collateral stable nervous elements. The epileptiform fit (excepting, perhaps, some slight terminal fits) is not the result only of the discharge of the cells of the discharging lesion, as was supposed for a limited illustration, when speaking of the imaginary "buttoning centre." Making a purely arbitrary limitation, we may imagine that the primary excessive discharge, that of the fulminate of A.'s thumb-centre, only produces directly the initial spasm, that of his thumb (the "signal symptom"); all the rest of his convulsion will be indirectly produced by it, by *compelled* excessive discharges of stable cells. Of these compelled discharges I shall speak again later.

It will now be seen why the term physiological fulminate is occasionally used. I use it in almost a literal sense; the discharging lesion is supposed to be a detonator of collateral stable cells, just as a fulminate (in the artillerist's use of the term) is of the comparatively stable gunpowder in a cannon. I believe that the only thing persistently physiologically abnormal in A. is that some few cells of his thumb-centre have become fulminant. To speak figuratively, this "mad part" compels many collateral "sane" cells and cells of middle and lowest motor centres, and ultimately the muscles, to co-operate in its occasional and sudden excesses—makes them "act madly" for a time. If the few highly explosive cells, those of the discharging lesion, could be destroyed, the patient would be rid of his fits; he would lose nervous elements which are doubtless never of value for co-operation with the collateral stable cells in normal operations; he would lose cells of negative value and of positive injury—cells like those of an animal poisoned by strychnine, which, on their discharge, "run up" movements into useless contentions. The cells of A.'s fulminate, when called on to co-operate with normally stable cells in any operation, function excessively, and so as, after the manner of a detonator, to cause wide excessive discharges of many stable cells. It is a pity that A. cannot be rid of these worse than useless cells; but I know of no way of effecting this riddance. There is the surgical question of cutting out part of the cortex.

I have, in the foregoing, used, regarding hyper-physiological discharges, the term excessive, and, regarding the resulting convulsion, the term severe; the more excessive the discharge the severer the fit. I now use more precise terms, and consider this part of the subject as it was in effect, so it seems to me, long ago considered by Herpin. I regret greatly that my ignorance of physics renders me unable to deal with it adequately.

With regard to nervous discharges, or, as I shall here say, liberations of energy by nervous elements, we have to consider two aspects—quantity of energy liberated, and the rate of its liberation; the two varying factors both in normal and in "excessive" nervous discharges. With regard to the convulsion, we have to consider its degree, and the rate with which it is produced.[17] We have to study the amount of convulsion, the range of convulsion, and particularly the "deliberate" or "sudden" rate of onset. In two liberations of equal quantities of energy at different rates there is the same momentum or quantity of motion. But the force of the more rapid but shorter liberation of energy will be greater than that of the slower and longer liberation. Force only exists while it lasts; there is no doctrine of conservation of force. The more rapid the liberation of energy by a discharging lesion the greater resistances will be overcome, the more numerous collateral stable elements will be compelled to discharge, and thus the more the amount of convulsion and the greater it range.

I suppose that there are degrees of instability of the cells of the fulminates in different cases of epileptiform seizures and at different periods of the same case. When A.'s fits "get worse" (greater amount of convulsion and greater range of convulsion), more cells may have then become highly unstable, or those already highly unstable may have become still more so; his fulminate becomes more fulminant, but that fulminate is made up of cells of the same part of the "motor region," if not of the very same cells. I now consider it must be taken hypothetically, the differences of fulmination of discharging lesions. Let me suppose two cases, in each of which there is a discharging lesion constituted by cells of the thumb centre. In the patient A. the cells are (I am only able to distinguish vaguely) of lower degree of high instability (his fulminate is much less of a fulminate) than in the case of A. A. In both the first spasm is of the thumb. I shall mention the cortical discharges only; the sequent discharges of the lowest motor centres and of the muscles are to be taken for granted.

In A—the primary discharge, that of the fulminate, is deliberate, produces (secundo-primary) discharges of few collateral stable cells, and produces them slowly one after another; the corresponding convulsion sets in deliberately and spreads slowly; is of little range (very local; for example, of the arm only), is of long duration, and there is an easily traceable distinct sequence over the range (all this, of course, comparatively with what occurs in the next case). In A. A. the primary discharge is sudden, produces (secundo-primary) discharges of many collateral stable cells, and produces them rapidly and more nearly at the same time; the corresponding convulsion sets in suddenly and spreads rapidly; it is of great range, is of short duration, and there is a less easily traceable distinct sequence over the range.

The more rapid, though the shorter, of the two primary liberations by the discharging lesions—supposing an equal quantity of energy to be liberated by each—will overcome greater resistances, and will thus compel discharges of a greater number of collateral

[16] There is the obvious qualification that some nervous elements may have been destroyed by the same pathological process which caused high instability of other elements, and thus that some connections of the latter are cut.

[17] Herpin, in his valuable work on *Epilepsy*, sums up several propositions as follows (italics in original):—"En résumé: *plus le début est long, moins la crise est violente, plus il est instantané, plus l'accès est intense.*"

stable elements; the convulsion produced will be both of greater amount and of greater range. Lines of many different degrees of resistance will be overcome by such a primary liberation more nearly at the same time; the convulsion will more quickly attain its maximum at every part affected, and will be more nearly of the same degree in all parts affected.

We have not only to note how much of the body is ultimately involved, but also the order in which the several parts involved are affected—the March of the convulsion. There is not a simple, but a compound sequence of spasm; the convulsion does not cease in one part when another is involved. To observe, to give a simple example, how much of the arm has been involved when convulsion appears in the face will, I think, help us to clearer notions of localisation of movements of those two separate parts of the body in the centre discharging (anatomical localisation); or if not, at any rate as to the time relations of different elements of different centres (physiological localisation). From increasing discharge of a motor centre there is a double effect; there is not simply "more convulsion," there is (1) greater amount of convulsion of the part first seized, and there is (2) extension of convulsion to the next part of the same muscular region, or to some other part represented in the centre discharging (or in another centre connected with the one primarily discharging by particular time relations). Most generally, the progress of the two dissolutions—convulsion from increasing discharge of a motor centre, and paralysis from increasing destruction of a motor centre—may be rudely (and without the least pretence at exact quantification) symbolised as (1) x, (2) x^2y, (3) x^3y^2z; the order x, y, z, implies the representation in the centre supposed of the time relations, and of the degree of speciality of movements of the three parts. Horsley and Beevor find Compound Order in their development of "simple movements" by very slight excitations of small parts of a monkey's "motor region." After developing what they call the Primary Movement of a part, say one of the shoulder, there follow, on increasing the excitation, secondary and tertiary movements—that is, there is produced a sequence of movements of segments of the arm. But when the secondary movement comes the primary has not ceased; on the contrary, it is intensified: the development may be roughly symbolised as p and then p^2s.

Returning to epileptiform seizures, Compound Order is observed on a small scale in fits involving limited regions of the body (very well seen in the face), and on a large scale when such seizures become universal. There is a very intricate compound sequence from the beginning of the fit to its universalisation. I shall, however, make artificial separations, and say a fit which becomes universal that excessive discharge beginning in some cells of a part of the right middle motor centres produces convulsion (1) starting in the left hand and spreading up the arm and down the leg (first side), (2) involving both sides of the trunk, and (3) finally gaining the limbs of the right (second) side when all parts of the body are in convulsion together. That the left limbs are convulsed from discharge of the right middle motor centres, by intermediation of the first set of fibres of the second segment, is not doubted. I used to suggest that convulsion of both sides of the trunk and of the right limbs is also produced by discharge of that half by the second and (or rather as I should now say) third sets of fibres. This was rash, for supposing that some convulsion of the second (right) side *is producible* by discharge of that one half (right), it does not follow that the convulsion which actually occurs *is produced* by it alone. For my hypothesis of representation of both sides of the body in each half of the brain, it would suffice if there were any degree of tonic or clonic convulsion of the right (second) side, for I only suppose that the right side is represented less than and subordinately to the left side, and also second in time in the right half. The subject is a difficult one. Horsley finds that when the corpus callosum is divided, excitation of the right (I continue to say "right" for convenience) middle motor centres produces epileptic convulsion of the left limbs only.[18] In the case of dogs, Franck and Pitres found that artificially induced discharges of the right "motor region" caused universal convulsion when the left motor region had been extirpated and the corpus callosum divided. I quoted them to this effect in my third Croonian Lecture.[19] They, however, as I then said, attribute the universalisation of the convulsion to the pons, medulla, and cord (which together I call the lowest level).

Considering, then, the opinions of those who hold that the second and third sets of fibres, as well as the first set (all the fibres of the second segment of the kinetic route interconnecting the right middle motor centres with the lowest motor centres), end in the left lowest motor centres, the researches of Franck and Pitres and those of Horsley, and also the great complexity of the subject, I ought not to be dogmatic as to the process by which universalisation of an epileptiform convulsion is produced. Most likely in severe seizures the left middle motor centres are discharged, as Horsley supposes, after the right by intermediation of callosal fibres, although possibly some slight convulsion of the right limbs is produced from discharge of the right half alone by intermediation of the second and third sets of fibres. And most likely, too, the anterior commissure of the pons, medulla, and cord (intrinsic fibres of the lowest level interconnecting left and right lowest motor centres) are concerned. Probably the loss of consciousness answers to excessive discharges of the highest centres caused by the medium of sensory (upward) fibres. The process of universalisation of epileptiform seizures is a very intricate one, and deserves more precise analysis than I am capable of making.

I stay here to say that, taking the case of A. in illustration, when we have located his lesion we have done anatomical work only; when we have noted all we can about his convulsion (therefrom inferring that the lesion is a "discharging lesion" and the effects of its discharge direct and consecutive), we have done physiological work only. The experimenters on animals do the anatomico-physiological work thoroughly well.

Taking up again the case of A., we have now the third element, pathology, to consider. Here is the great difficulty. Being sure that A. has a discharging lesion of this thumb centre, there may be no evidence to show whether that lesion is produced (indirectly) by tumour or not. Here I urge again the necessity of distinguishing between the physiology and pathology of cases of nervous disease.[20] Having urged that there is a degree or kind of functional change (*b*) which is not the result of any pathological process, I am all the more wishful to urge the distinction. I continue to speak of the first degree (*a*), and urge the distinction from this point.

It may be epigrammatically said, and with truth, that an epileptiform seizure is "only a symptom." Nevertheless, it is always symptomatic of one *physiological* thing, a "discharging lesion." Otherwise put, an epileptiform seizure is not a symptom of tumour, of "softening," or of meningitis; it is a symptom of the one thing—high instability of certain cortical cells, *however produced*—produced by any pathological process. Physiologically speaking, there is but one "cause" of epileptiform seizures, namely, high instability of some cells of some centre of the Rolandic region; but there are many "causes" of them if we mean pathological processes leading to that instability. So that in the case of A. (he being *subject to fits*), the question in pathology is not the vague one: "What is the *disease* of a certain part of his cortex"? but "What abnormality of the nutritive process has produced such an alteration in the composition of the material of cells in that part as to render them highly explosive, and how was that abnormality set up?" Or, more generally and regarding all cases of epileptiform seizures (and epileptic too), we put the question: "How are local persistent discharging lesions established and kept up?"

The first question is: "What is the most general nature of the abnormal nutritive process of cells of discharging lesions in epileptiform seizures?" This we ask ourselves, whatever the particular gross pathology or morbid anatomy, tumour, "softening," etc., may be in any case.

The cells of the discharging lesion, although *quasi*-parasitical, are not strictly parasitical; for another purpose I urged that the nervous arrangements into which they enter remain integral parts of the nervous system, and now I urge that they are nourished along with the rest of the body. Their nutrition does go on in some base fashion as certainly as that that of their stable neigh-

[18] In the abstract of "Brown Lectures," *Lancet*, December 25th, 1886, Mr. Victor Horsley is reported: "The conclusions ..... all round, therefore, were very emphatic, to the effect that convulsions due to cortical discharge are evoked in various groups of muscles by nerve energy proceeding from that centre in each hemisphere which is in relation to each group of muscles, and that in generalised epileptic convulsions both cerebral hemispheres are involved.

[19] JOURNAL, March 29th, 1884.

[20] I have urged this many times for at least twenty years. "Functional changes must not be confounded with pathological changes, although of course the two necessarily co-exist" ("Study of Convulsions," "St. And. Grad. Rep.," vol. iii, 1870). I mention this as I am said to have put forward the "theory of discharges" as the *pathology* of epilepsy—a thing I earnestly repudiate. In 1870 I did not make two degrees of functional changes, and spoke only of what I now call the first degree (*a*).

bours goes on in a proper way. By nutrition of some kind the cells of the discharging lesion attain high tension and very unstable equilibrium, and occasionally discharge suddenly, excessively, and temporarily; their stability is after their discharge below normal; by continuance of this abnormal nutrition they reattain high tension, or no more fits would ensue. What is the "base fashion" of nutrition of these cells? It does not follow that the cells are *more* nourished, although they are certainly *worse* nourished. So to put it, they may be less nourished in quantity and worse nourished in quality. The nutrition must be such that it alters the composition of nervous matter of the cells, and in such a way that it becomes more explosive.

I have so often spoken on this subject that I will only mention the hypothesis that the nutrient fluid bathing the cells is comparatively stagnant, and that in consequence there is inferior nutrition. I suggest that there is "substitution nutrition," phosphorus compounds becoming more nitrogenous, or nitrogenous compounds more nitrogenised. I may illustrate by the well-known case of glycerine becoming explosive (nitroglycerine) when some of its hydrogen is replaced by nitric peroxide. The Composition of this substance is altered, but its Constitution remains the same. It may possibly be that the mass of the explosion of nerve cells, both in health and convulsion, is of non-nitrogenous matter, as is the case in the normal discharges of muscle. But the suggestion is that a nitrogenous substance is the "pivot" of the metabolism of nervous matter both in physiological and in hyperphysiological states.[21] I suppose the greater nitrogenisation of the material of nerve cells makes it highly explosive, but that the constitution of that material and the morphological structure of the cells remain the same. If this hypothesis be valid, there are presumably degrees of substitution (as there are in the three chloracetic acids, for example), and consequently degrees of high explosiveness. It may be that when a patient's fits "get worse," the original fulminate becomes more fulminant by still greater nitrogenisation, as well as that more cells become part of his fulminate. The abnormal nutrition leading to change of composition is a pathological process, whilst the high explosiveness it produces is a functional change. Surely it is the change of function which should be called a functional change. Whether these speculations be valid or not, it is a warrantable inference that the nutrition of the cells of the discharging lesion goes on, that it goes on wrongly, and that some material of the cells does become highly explosive.

Now I consider differences in sizes of cells with regard to their normal and morbid nutrition.

For theoretical reasons, and after the observations of Betz and Mierzejewsky, I suggested [22] that those centres of the motor region which especially represent small muscles (eyes, face, and hands) will have a greater number of small cells than those which especially represent the large muscles of the limbs. I think this is generally true of the "motor region." And I suggest that it accounts for the great frequency of onset of epileptiform fits in the hand and face, for reasons to be given presently. The "leg" centre contains many large cells, but it also contains some small cells. Bevan Lewis's researches, to which I am very greatly indebted, show that the parts of the motor region representing small muscles have most small cells. He says—and this bears on the remark I made on the leg centre—"that alongside the largest pyramidal cells are numbers of others of the *smallest* dimensions" (italics in original) [23] so that the discharging lesion of the "leg centre" may be made up of small cells.

Although it is convenient for some purposes to say "small muscles," the expression is not exact. For I urge once more that nervous centres do not represent muscles, but movements. I will substitute the expression "small movements" for "small muscles," and that of "large movements" for "large muscles." As these terms, the best I can think of, are vague, their meaning must be taken as defined here. Most of the movements of the hand are "small" according to the definition I now give of "small movements." The parts moved have little mass, and in most of the operations they serve in there is little more added mass. The muscles are small and numerous, and in most operations by the hand their movements are of little excursion, of short duration, and rapidly changing. Short, prompt, and frequent liberations of small quantities of energy will be required for these successions of "small" different movements; I submit that the nervous arrangements for these movements of the hand have small cells, and very many small cells. Most of the muscles of the shoulder are "large movements," according to the following definition. The muscles are large and few; there is much mass to move, the whole arm to lift, and the added mass is often great in some of the operations they serve in, as in lifting weights the hand takes up. In most operations they serve in, the movements are of large excursion, of comparatively long duration, and are comparatively little changing. They will require persisting and slow liberations of large quantities of energy by, I suggest, comparatively few and large cells. We may have "large" and "small" movements of different parts of one limb in a single operation. When the arm is put forth there are "large movements" of the shoulder, upper arm, and forearm; and when the fingers, thus put forward, elaborately explore an object, there are "small movements" of the hand.

But although parts which most often engage in "large movements" have large muscles, yet there may be "large movements" of parts having small muscles. If we grasp an oar and pull a boat, the whole of the musculature of the arm serves in a succession of similar large movements (and then subordinately to other movements). For these, I suppose, will be discharges of large cells, even for the movement of the hands grasping the oar; there is, indeed, then but one unchanging movement of the whole hand, a large movement *as defined*, all the small muscles serving together as if one muscle.

A principle of representation is here in question; we have not only to do with sizes of cells, but also with numbers of cells, and therefore with the volume of different centres. Schroeder van der Kolk, illustrating by the case of the sturgeon, which has large muscles and few cells in its spinal cord, pointed out that there is not a mere relation between quantity of grey matter and size of muscles, but that the grey matter is greater in proportion to the complexity of movements of muscles. This shows that we have never to forget that centres represent movements of muscles, not mere muscular masses. I have several times drawn attention to Herbert Spencer's statements bearing on this question.[24] One remark he makes is: "In proportion to the number, extensiveness, and complexity of the relations, simultaneous and successive, that are formed among different parts of the organism, will be the quantity of molecular action which the nerve-centres are capable of disengaging." Spencer takes count of both impressions and movements; I am illustrating by movements alone. The much greater volume of the middle motor centres is in accord with the fact that they represent vastly more numerous different movements than the lowest motor centres do; the muscles represented by both levels are, of course, the same, being in each case all the muscles of the body; hence the middle motor centres contain many more cells and fibres than the lower centres do. Presumably the same principle applies in detail. According to Horsley and Beevor, the thumb and index finger, which have a great number of different movements, chiefly "small movements," have a large area of representation in the motor region. As to the trunk area of the cortex, I quote what Horsley and Schäfer say:[25] "It certainly is not a little remarkable that the numerous and powerful muscles of the spine should be governed from so small a portion of the cerebral cortex, *but it is to be remembered that the movements of which the spine is capable are comparatively few and simple*" (no italics in original). The supposition is that parts having many small and greatly changing movements are especially represented by small cells and by many small cells, and that parts having but few and little changing ("tonic") movements are represented by large cells and by few large cells.

The size of cells is a very important matter with regard to their nutrition. Both in health and in disease small [26] nerve cells will be nourished more quickly than large ones when both are bathed

[21] "Whether the chief product of the metabolism of any tissue be a proteid substance, or a fat, or a carbohydrate, proteid substance is the pivot, so to speak, of the metabolism, and nitrogenous bodies always appear as the products of metabolism." (Foster, *Physiology*, pt. ii, p. 828).

[22] *Medical Press and Circular*, August 23rd, 1876.

[23] *A Text-book of Mental Disease*, p. 106.

[24] *Psychology*, 2nd ed., vol. i, pp. 35, 55, 67.

[25] *Phil. Trans.* vol. clxxix, 1888, B.

[26] "Other things equal, the smallest cells will soonest become unstable. A mass of nervous matter in many small cells will 'present a much larger surface to the contact' of nutrient fluid than the same mass in a few large cells" (Harveian Lectures, *Med. Times and Gaz.*, January 11th, 1879). My attention was first directed to this subject on reading Spencer's *Biology*, where he expounds his theory of growth. A brief statement of Spencer's theory will be found on p. 220 of *The Evolution of Sex*, by Geddes and Thomson, from which I quote. "In spherical and all other regular units the mass increases as the cube of the diameter, the surface only as the square." I refer the reader to Ross's great work, *Diseases of the Nervous System*, vol. i, p. 13, where he deals with the significance of differences in sizes of nerve cells with regard to normal and abnormal nutrition and its consequences.

in the same nutrient fluid. (From diminished nutrient supply the small cells will atrophy sooner than large ones.) The smaller cells will become highly unstable sooner than large ones during morbid nutrition. It certainly is the fact that most epileptiform seizures begin in parts having "small movements," in parts represented by areas of the cortex having most small cells. As I have pointed out, they may begin in the large muscles of the upper arm; it would be begging the question to say that in these cases the smallest cells of the "shoulder centre" are those which first become highly unstable. The size of cells bears also on rates of discharge.

Four hot iron balls will become cold much sooner than the same mass of iron in one ball having the same quantity of heat as the four balls together have. From discharge of four small cells, which are together equal in mass to that of one large cell, there will be, I submit, a liberation of energy in a shorter time than by the large cell, supposing equal quantities liberated in the two cases. Hence another reason for fulmination of the highly unstable cells of discharging lesions if these cells are small. As being somewhat illustrative, I may refer to different sizes of grains of powder used as ammunition; slowly burning (pebble) powder is required for large cannons, quickly burning (fine-grained) powder for firearms.

To repeat the several hypotheses, Epileptiform seizures begin most often in parts of the body having "small movements;" these movements are represented by nervous arrangements having many small cells.[27] Small cells present a more extensive surface to nutrient fluid than the small quantity of grey matter in large cells, and will be more quickly nourished than large ones are. Nerve cells become highly unstable from an abnormal nutrition, such that, although their structure and the constitution of their material is unaltered, that material becomes of more nitrogenous composition, and thus more explosive. Small cells become highly unstable more readily than large ones do; thus discharging lesions are supposed to be especially of small cells. A rapid liberation of energy overcomes greater and more numerous resistances than a slower liberation of an equal quantity of energy. Small cells liberate their energy in a shorter time than large ones; hence the currents developed by fulminates of small cells overcome greater and thus more numerous resistances than would fulminates of large cells, and hence produce more convulsion and greater range of convulsion.

We have assumed that the nutrition of the cells of the discharging lesion is continuous, and have supposed in effect that the nutrient fluid is comparatively stagnant. But how is this comparative stagnation brought about? This brings us to pathology, commonly so-called. We have to distinguish between what I may call the coarse pathology of a case and its immediate pathology.

[In the remainder of the lecture the production of discharging lesions by tumours and by arterial occlusion was considered.][28]

[27] The movements, speaking most generally, represented by the cerebrum are, I suppose, numerous different punctuated movements (many and different "small movements"). Presumably those represented by the cerebellum are, in comparison, few and little different movements, movements as it were gliding into one another (few and similar "large movements"). It is interesting to observe that the structure of the cerebellum is more uniform than that of the cerebrum, and that those of the cells of the cerebellum which are presumably motor are large and of nearly equal size. The movements for bracing up the spine in standing or walking, and the separate movements of the legs and arms in walking, will require comparatively continuous supplies of large quantities of energy. (I believe, however, that the cerebrum and cerebellum are both engaged by contemperation in at least all extensive operations.)

[28] See JOURNAL, July 21st, 1888.

AFTER April 1st, Professor Curschmann, of Leipzig, will replace Professor Unverricht, of Dorpat, in the editorship of the *Fortschritte der Medicin*.

AT a meeting on February 24th, the Council of Public Health for the department of the Seine had under consideration a report from M. Alexandre, chief of the sanitary service, urging the establishment of a departmental laboratory, in which researches on the contagious diseases of animals could be carried out. It is proposed to ask the departmental authorities for a grant of money for the purpose.

THE Royal Medical Benevolent College has received a legacy of £400 from the late Mrs. Price, of Leamington; and also £250, assigned to it by the trustees of the late Mr. Daniel Procter, of Manchester, who left a large residue at their disposal for charitable purposes.

THE

GOULSTONIAN LECTURES

ON

SOME CEREBRAL LESIONS.

Delivered before the Royal College of Physicians of London.

BY G. NEWTON PITT, M.A., M.D., F.R.C.P.,

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LECTURE II.

1. CASES OF MENINGITIS AND TUBERCLE SECONDARY TO EAR DISEASE. 2. CASES OF CEREBRAL ABSCESS AND SINUS THROMBOSIS DUE TO OTHER CAUSES THAN EAR DISEASE.

Meningitis (25 cases).—In patients under 10 this is the most fatal complication of ear disease, 8 out of 11 patients below this age dying from meningitis. The distribution as to age is under 9 years, 9; between 10 and 19 years, 6; between 20 and 29 years, 5—that is, the younger the patient the more frequently does meningitis occur. The meningitis was usually secondary to some other complication.

The following is a list of lesions which had preceded the meningitis: Pus outside the dura mater on either the anterior or the posterior surface of the petrous bone in 5 cases, a sloughing condition of the dura mater in 2, perforation of the dura mater by an extradural abscess in 1, thrombosis of the lateral sinus in 4, of a dura-matral vein in 1, cerebral abscess reaching the surface in 3, bursting into the lateral ventricle in 2, fracture of an abnormally thin petrous bone in 1, and the passage of pus along the internal auditory meatus from the ear in 3. Whereas cerebral abscess only ensues in cases of old ear disease, meningitis may come on within two days of the onset of otorrhœa, and is the probable lesion when there is a complication in the early days of ear trouble, as may be illustrated by two cases (XL and XLIX) of foreign bodies in the ear, in which death ensued within four and eight days respectively.

Another group of cases are those in which (3) suppuration spread through the internal auditory meatus from the internal ear. The exact course which the pus took inside the bone was not made out definitely, although in at least one case a minute dissection was made. Probably the course was not the same in all. These cases are of importance because they show that infection may travel from outside through the internal ear to the posterior fossa; in each instance a fatal meningitis was set up.

The symptoms which were recorded of the 23 cases of meningitis may be grouped as follows, bearing in mind that of 6 there are only a few brief notes, but the remaining 17 are fully reported. The onset was sudden and the course rapid. The temperature was raised in all (19); it averaged about 101°, but in some was towards the end very high. In 15 there was headache, which was not of extreme severity in most cases; earache, in addition, was noted in 8 cases. The patients were listless or lethargic in 9, drowsy in 8, and finally drifted into coma in 11 instances; 8 suffered with vomiting, and 6 were extremely restless. The optic discs were recorded as normal 6 times, while neuritis was only noted in the 3 following cases (XLVI, L, XIV).

Neuritis was found in a child a year old, ten days after a fit. At the inspection four days later there was found a general cerebro-spinal meningitis secondary to the ear disease. This is the only case in which the neuritis was due to the meningitis. In the second case double optic neuritis was found in a girl 12 years old, six days after the onset of the illness. Pus was let out from both surfaces of the petrous bone; the neuritis at first diminished and the girl improved, but she relapsed and ultimately died from meningitis, due to the rupture of an extra-dural abscess into the arachnoid space. The neuritis was probably due to this extra-dural abscess.

The third case was a girl aged 8, who had thrombosis of the lateral sinus, and there was an abscess in the centrum ovale, which burst on to the surface of the brain after an exploratory operation. The neuritis in this case was due to the thrombosis. Optic neuritis is, therefore, rarely due to meningitis, and the reason is not far to seek. Five or six days must elapse before neuritis develops, and still longer when the meningitis is limited