THE LUMLEIAN LECTURES ON CONVULSIVE SEIZURES.

Delivered before the Royal College of Physicians of London.

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LECTURE I.

Definition of Terms.—Classification of Convulsions.—The Three Evolutionary Levels in the Nervous System.—Middle Level Fits. "Epileptiform."—Highest Level Fits: "Genuine Epilepsy."—Lower Level Fits: "Ponto-bulbar."—Varieties of Lowest Level Fits.—Abnormal Affections of Consciousness in different kinds of Fits.—The Discharging Lesion in Epileptic and Epileptiform Fits.—The Interconnecting Fibres.—The Kinetic Route.—Representation of Movements.—The Right Motor Region.—The Cerebellar System.

THERE are two pleasant preliminaries, the sole parts of my address I can enter upon with a light heart. The first is most earnestly to thank you, Mr. President, for the honour you conferred on me in asking me to deliver the Lumleian Lectures. There is one thing for which I cannot blame myself. I have, as was my duty, taken all the pains I could over the task you were so good as to assign to me. The second preliminary is to mention my great obligations to Dr. Ferrier. There are very few men of the day by whom both the art and science of medicine have been so greatly helped. It is only since his remarkable researches that medical men in this country have studied convulsions in a thoroughly realistic way. Without further particular acknowledgments I make the general one, that all over the field of neurology I am profoundly indebted to Ferrier.

Convulsions and other paroxysms are owing to (1) sudden, (2) excessive, and (3) temporary nervous discharges.¹ The term "nervous discharge" (used before me by Spencer) has been much objected to; when I say that it is used synonymously with "liberation of energy by nervous elements," it will mislead no one. There are nervous discharges in all the operations of health. I should use the term "explosive discharge" for the abnormal liberation of energy in convulsions were it not that physiologists speak of "explosive decomposition of the muscle's substance" which gives rise to contraction of the muscle in health; and thus it may be that normal nervous discharges, those in the operations of healthy people, are "explosive." So then I use the word "excessive" for the discharges which have the three characters mentioned, the words "sudden" and "temporary," or the word "paroxysmal," being understood; when dealing with convulsions, it will not be always necessary to add the term "excessive." I shall, however, occasionally use "explosive" for states of cells of a discharging lesion, qualifying it by the word "highly," and sometimes the term "high instability." I do not speak of "disorderly discharges," for if in some ways the expression be correct, it hides from us the fact that the most brutal-looking convulsion is only the sign of a departure by a vast excess and by a caricature from normal nervous discharges.

I have always assumed, and shall continue to suppose, that convulsion results from excessive discharges of nerve cells, meaning, of course, liberation of energy during rapid decomposition (katabolism) of some matter in, or of part of, those cells. I shall frequently speak of cells concerned with excessive (primary) discharges as constituting a "discharging lesion," and sometimes of them as making up a "physiological fulminate," or occasionally, using Horsley's term, of their being together an "epileptogenous focus." Some material of the cells which make up the discharging lesion has, by morbid nutrition, become of very high tension and of most unstable equilibrium (briefly of high instability), and occasionally discharges excessively. I do not assert that excessive (primary) discharges producing convulsions always depend on a persistent state of high instability of cells; to give but one example to the contrary, excessive discharges beginning in a

healthy rabbit's respiratory centres are induced by rapidly bleeding the animal to death, or by quickly asphyxiating it.

The discharging lesion, though I speak of it as persistent, meaning that it is of the same locality throughout each case, yet varies in its condition.² The discharges are occasional. After their excessive discharge the cells are no doubt far below the degree of stability (properly comparatively slight instability) of normal cells; they will regain a highly abnormal degree of instability—again become highly explosive—by further morbid nutrition. These qualifying remarks should be borne in mind, and especially when the discharging lesion is spoken of as a fulminate—it is occasionally fulminant.

I make three classes or kinds of convulsions. Convulsions, or I will say, fits, differ in kind, according as centres discharged differ in rank; or, speaking more definitely, as the centres first engaged in paroxysms make up different evolutionary levels of the central nervous system. I have several times suggested that there are three levels of the cerebral (central) nervous system; each is sensori-motor, and each represents impressions and movements of all parts of the body. I speak briefly of what I suppose to be the hierarchy of centres of the nervous system as a basis for the classification of fits.

(1) The lowest or first level is roughly and incompletely defined as consisting of cord, medulla, and pons,³ and more completely, and yet still roughly, as being that sensori-motor division of the central nervous system, to and from which pass nerves (all cranial and all spinal nerves) for every part of the body. This level, speaking of its motor elements, represents simplest movements of all parts of the body by a series of lowest motor centres (lowest motor centre being a proper name for a centre of the lowest level) from those in the aqueduct of Sylvius for simplest movements of the ocular muscles to those of the sacral cord for simplest movements of the muscles of the perineum. This universally representing level is cerebro-cerebellar; it is at once the lowest level of the cerebral system and of the cerebellar system. For the present I ignore the higher levels of the cerebellar system, and go on to speak of two higher levels of the cerebral system. These levels are, as the lowest level is, sensori-motor, but I find it possible to illustrate by motor centres only, not, however, believing that these so-called "motor centres" are purely motor. (2) The middle or second level (its motor province) of the cerebral system is composed of centres of the Rolandic region (so-called "motor region" of the cerebral cortex), and, possibly, of the ganglia of the corpus striatum also. It represents complex movements of all parts of the body from eyes to perineum (re-represents). (3). The highest or third level (its motor province) of the cerebral system is made up of centres of the prefrontal lobes (highest motor centres, motor division of the "organ of mind"). It represents most complex movements of all parts of the body from eyes to perineum (re-represents). The highest centres (sensory and motor divisions of the highest level)—the "organ of mind," or anatomical substrata of consciousness—are the acme of the evolution; they have the same kind of constitution as lower centres; they are sensori-motor as certainly as the lumbar enlargement is. (Of course each level is bilateral).

That the lowest level is a very distinct division of the central nervous system will, I think, be granted. The separation of the frontal lobe into middle motor centres ("motor region"), making up the motor province of the middle level, and highest motor centres (prefrontal lobe) making up the motor province of the highest level, is, of course, hypothetical; there is no obvious morphological separation. I do not suppose that the evolutionary distinction is so abrupt or so decided as that between the middle and lowest levels. I will mention some differences in the two regions of the frontal lobes. (a) It seems certain that the middle motor centres ("motor region") are those cerebral centres directly connected with the lowest motor centres, as the facts of "Wallerian wasting" show; there is, however, possibly some "wasting" of fibres from the prefrontal lobes as low as the pons. This wasting has been differently interpreted. (b) The middle motor centres contain most large cells. (c) The middle motor centres are experimentally "excitable," and the prefrontal lobes are not.

² The rapid reader must not take "persistent discharging lesion" to be "persistently discharging lesion."

³ I have spoken briefly on what I believe to be the importance of reckoning the cord, medulla, and pons, or, rather, certain elements of these morphological divisions, as one, the lowest level—I believe it is the "spinal system" of Marshall Hall—(JOURNAL, July 14th, 1888). I do not pretend to be able to define the upper limit of this level.

¹ A term introduced by Dr. Edward Liveing in his masterly work *On Megrim* is "nerve storm." This term has met with much favour, and the conception it stands for has been of great value in elucidating some very complex problems in neurology.

This is a very important difference; it may be taken to mean that the præfrontal lobes are not, as I suppose, motor. The distinction currently made is vastly greater than the one I have submitted. Believing that the whole central nervous system (the organ of mind included—the mind, of course, not included) is a sensori-motor mechanism of three levels, the distinction I make is not of kind but merely of degree—it is that the præfrontal lobe is only greatly more complex, etc., than the "motor region." But, according to the received doctrine, whilst the latter alone is motor, the former differs from it in two ways; the præfrontal lobe has no motor constitution, and is part of the "intellectual centres." Distinguishing the psychical from the physical, I would say that psychical states are not functions of any centre, but are simply concomitant with functioning of the most complex, etc., sensori-motor nervous arrangements—those of the highest level ("organ of mind") of which level the præfrontal lobe is the motor division.

There are, I submit, three kinds of fits corresponding to the three evolutionary levels. It is convenient to speak of the three kinds in an order different from that used in stating the levels. (2) I mention epileptiform seizures first because their localisation is not doubtful. (They were first described by Bravais in 1824.) They are "middle level fits"—that is, they are produced by excessive discharges beginning in parts of the middle level (motor province) of the cerebral system ("motor region"). My hypothesis is that (3) fits of epilepsy proper ("genuine epilepsy" of some nosologists) are "highest level fits," and that many of them, not all, are produced by excessive discharges beginning in parts of the præfrontal lobes, highest level (motor province) of the cerebral system. Although the præfrontal lobes are not experimentally excitable, I suppose it will not be denied that their cells katabolise and liberate energy in their normal activities, and it is not unreasonable to suppose that cells of parts of them may, by pathological changes, become highly explosive, so that they occasionally discharge excessively. So that, if the præfrontal lobes are divisions of the "intellectual centres," as no doubt they are, and not, as I think, motor too, excessive discharge beginning in parts of them may produce epileptic fits.

Of course, this is speculative. I am not aware that anyone pretends to know the seat or the pathology of cases of "genuine epilepsy." I do not use the term "cortical epilepsy," because both epileptic and epileptiform seizures are, to my thinking, cortical fits. (The difference in meaning assigned to the two terms epileptic and epileptiform must never be lost sight of.) (1) I think that there are "lowest level fits." These are fits produced by excessive discharges beginning in parts of the lowest level, a level which is common to the cerebral and the cerebellar systems. I suppose that most of them are owing to excessive discharges beginning in centres of the bulbar and pontal regions of the level, hence I sometimes use the term "ponto-bulbar fits." With regard to epileptiform and epileptic fits, I deal almost exclusively with cases of patients subject to fits—with, so to speak, "chronic cases."

I have so far spoken only of three Kinds of fits. No doubt there are Varieties of each kind. Whilst the kind answers to the level, the variety of each kind answers to the particular part of the level in which the excessive discharge begins. It is certain that there are varieties of (2) epileptiform seizures; each is marked by a particular place of onset of the convulsion. There must be at least as many varieties of (3) epilepsy proper as there are different "warnings" of the paroxysms. Presumably there are varieties of (1) ponto-bulbar fits.

Then, of course, there are Degrees of each variety dependent directly on degree of the primary discharge, and indirectly on that of the secondary discharges. (2) For example, there are degrees of that variety of epileptiform seizures marked by the first spasm being of the thumb. There are many ranges of it, from convulsion almost limited to a thumb, thence onward to universal convulsion. (3) Everybody recognises that epileptic attacks occur in two vastly different degrees, *les petits maux* and *les grands maux*, and there are sub-degrees of each of these degrees. (1) There are degrees of convulsion in lowest level fits produced by Brown-Séquard's method in guinea-pigs. There are, no doubt, degrees of respiratory fits produced in certain lower animals by rapid bleeding, by ligation of the great arteries of the neck, and by sudden stoppage of respiration.

To repeat, "fit" is a term used to include convulsive paroxysms of all kinds dependent on excessive discharges beginning in any part of any one of the three levels, the epileptic and epileptiform

beginning in one half⁴ of a level. There are three kinds of fits, (1) ponto-bulbar, (2) epileptiform, and (3) epileptic. There are, the supposition is, varieties of each kind, and degrees of each variety of each kind.

I have used the wide term "fit" advisedly because my method is not merely an empirical or clinical one. It is not only an endeavour to find out whether a convulsive paroxysm a patient has is like or unlike that of the type "genuine epilepsy" of nosologists or any other type, but is also an endeavour to discover how it shows a particular departure from normal states of his nervous system.

Is there any difficulty in recognising the difference in the two mental attitudes, or, as I shall now say, the distinctness of the two view-points? From the empirical or clinical view-point we look to see how this or that fit approaches this or that nosological type. From the scientific view-point we look to see how these or those paroxysmal manifestations are produced, asking ourselves, "What is the level and what is the particular part of it in which the excessive discharge producing this or that set of manifestations begins?" or, regarding all kinds of fits, the more general question, "What different effects can an excessive discharge 'get out of' the different levels?" The use of the general term "fit," compels careful segregation of kinds and analysis of individual cases. In this frame of mind we note the manifestations whether they have "the characters of an ordinary epileptic fit" or not. Whilst for purely scientific purposes I care very little for an answer to the question, "Do excessive discharges beginning in ponto-bulbar centres produce paroxysms resembling those of the epilepsy of nosologists?" I care very much for one to the question, "What effects do excessive discharges beginning in ponto-bulbar centres produce?" This question is, as yet, only to be replied to by experimenters who artificially produce fits in lower animals. I should be very much astonished if it turns out that excessive discharges beginning in any centres of the lowest level do produce convulsions having the same characters as those produced by such discharges beginning in centres of the higher levels. It would be marvellous if excessive discharges beginning in centres lowest in rank produced fits like those (epileptiform seizures) which are produced by excessive discharges beginning in parts of the more evolved centres, the middle motor cerebral centres ("motor region").⁵

As I shall not have time to deal with lowest level fits, I will here mention what I think are some fits of this kind, not classifying them, but making a rough arrangement into three groups: 1. *Respiratory fits* (respiratorily beginning from primary discharge of the main (medulla) respiratory centre.⁶ I think that fits of laryngismus stridulus come in this category, but Semon thinks they are cortical seizures.⁷ Respiratory fits are easily induced in animals, and are described in all works on physiology when asphyxia is considered. Kussmaul-Tenner fits are respiratory fits.⁸ It is said that convulsions occur in newborn animals

⁴ It is convenient to use the word "half" (lateral) for nervous system and "side" for body.

⁵ I formerly used the term epilepsy generically for all excessive discharges of the cortex and their consequences. At that time I did not think there were any fits in depending on excessive discharges beginning in any part of the ponto-bulbar centres. Using then the term epilepsy generically, I submitted that any part of the cerebral cortex might become highly over-unstable and discharge excessively. So that under the term epilepsy used generically there were epilepsy proper, epileptiform seizures, and migraine (the last mentioned being then spoken of as a sensory epilepsy), and, indeed, any paroxysmal symptoms attributable to sudden excessive discharges of any part of the cortex. I now use the term epilepsy for that neurosis, which is often called "genuine" or "ordinary" epilepsy, and for that only. Of course in all quotations I preserve the term epilepsy when used regarding any class of fits.

⁶ Long ago (*St. And. Med. Grad. Trans.*, vol. iii, 1870) I had the same mental attitude. After speaking of observations of the local onset and march of spasm in cases of fits from cerebral tumour, at a time when the cortical "motor region" had not been defined, I wrote: "We do not care to say that a tumour of the brain (or minute changes near it) had 'caused epilepsy,' but that changes in a particular region of the nervous system—say in the region of the middle cerebral artery—led to convulsions in which the spasm began in the right hand, spread to the arm, attacked next the face, then the leg, etc."

⁷ *Brain*, April, 1886.

⁸ See an able paper by Dr. Gay in *Brain*, January, 1890, for much valuable information on laryngismus stridulus, and for arguments against the view I take of the causation of the paroxysms.

⁹ The fits produced in lower animals by rapid bleeding, by ligaturing the great arteries of the neck, and by asphyxia, are alike respiratory fits; they all depend on stimulation of the respiratory centres by lack of oxygen. Asphyxia experimentally produced in animals does not produce cerebral convulsions, but, on the contrary, renders the cortex inexcitable, whilst at the same time it increases the excitability of the ponto-bulbar centres. Again, as Franck says (*Fonctions Motrices du Cerveau*, pp. 86, 87), "l'anémie soit totale, soit partielle de l'encéphale, n'est nullement la cause des convulsions épileptiformes." Arrest of the heart by excitation of the vagus stops these fits.

after division of the cord below the medulla when they are asphyxiated; if so, these are certainly lowest level fits if not respiratory; it is possible that they are owing to discharge of subordinate (spinal) respiratory centres. 2. *Fits produced by convulsant poisons* (fits from nitrous oxide and curara are respiratory fits). Fits are experimentally produced in animals by absinthe and camphor. According to Magnan, convulsions are produced in animals by absinthe when the cerebrum has been removed. Many years ago Dr. George Johnson showed that convulsions occur in man from poisoning by camphor. Possibly some fits in renal disease (some so-called uræmic fits), and, it may be, fits occasionally part of a constitutional disturbance after urethral lesions, are ponto-bulbar fits from home-made poisons, as those just mentioned are supposed to be from foreign poisons. 3. *A condition for fits consequent on certain injuries of the cord or sciatic nerve* in guinea-pigs (Brown-Séquard). These are so well known that mere mention will suffice. There are often fits attending the onset of infantile paralysis; these are very difficult of explanation. I submit the hypothesis that they are lowest level fits produced by action on the ponto-bulbar centres of ptomaines, the result of disintegration of nervous matter of anterior horns; if so, they come in Group 1.

In all severe lowest level fits it is supposed that the primary discharge of ponto-bulbar centres not only induces discharge of other lowest motor centres, but also that by intermediation of sensory ("ascending") fibres it discharges centres of higher levels. (I never thought of implication of higher centres in these or any other fits by intermediation of sensory nerves until after consideration of the researches of Victor Horsley and Binswanger).¹⁰

It is necessary now to speak of abnormal affections of consciousness with regard to kinds of fits. I presume that there is loss of it in severe fits of all kinds. Consciousness is not a function of the highest cerebral centres; it is simply concomitant with their functioning. There is no physiology of the mind any more than there is psychology of the nervous system. On the basis of mere concomitance, mental symptoms (synonymously abnormal states of consciousness) are, strictly speaking, only signs to physicians of what is not going on or of what is going on wrongly in part of a patient's material organisation. Thus cessation of consciousness at, or close upon, the onset of an epileptic fit is of value to physicians as a sign that the correlative physical process, the excessive discharge, begins in some part of the "organ of mind," or equivalently, highest centres of the cerebral system; the physical process in these and all other kinds of fits is our proper concern as medical men. Consciousness is lost late in epileptiform seizures, and in those of but little range there may not be even defect of consciousness; this agrees with the empirical evidence that the excessive discharge begins in lower (middle motor) centres; probably excessive discharges are induced (upwards) in the highest sensori-motor centres by intermediation of sensory fibres when consciousness begins to cease in an epileptiform seizure. Availing ourselves of abnormal affections of consciousness as signs of states of the central nervous system, we next, so to speak, put them on one side in order to study the process in fits in a purely materialistic manner.

We must bear in mind that not only is consciousness absent in negative functional states of the highest centres, but also that it ceases during the diametrically opposite functional state, excessive discharge beginning in those centres; there is loss of consciousness not only during, but also for some time after, a severe epileptic fit; in post-epileptic states there is temporary exhaustion of elements of the highest centres, and, corresponding to that exhaustion, there is absence of consciousness. There are, however, degrees of negative affection of consciousness. There are degrees from that slight defect in some fits of epilepsy (*les petits mal*) to seemingly entire loss of it in severe epileptic paroxysms; and there are degrees from that existing with trivial confusion of thought after a very slight epileptic fit to seemingly loss of all consciousness in deep coma after a very severe one. I now return to the physical process of fits.

It will be observed that I have spoken of the excessive discharge productive of fits beginning in this or that level; further, of its beginning in some part of a level. In recapitulation, the primary

discharge in all kinds of fits is of some part of but one of the levels. And now I add that in epileptic and epileptiform seizures, of which alone I speak in the remainder of this lecture, the excessive discharge begins in some part of one half (lateral) of a level; thus, so to say, in these two kinds of fits the discharging lesion is "doubly local." If the discharging lesion be, as I suppose, of but a few cells, very little of a convulsion is directly due to it. Most of the convulsion is produced by intermediation of fibres between the cells of the discharging lesion and other cells of its own level and of other levels; there are induced, consecutive discharges of normal stable cells. Hence the interconnecting fibres of each level and the fibres connecting the several levels with one another, and the fibres connecting the lowest level with all parts of the body (lowest level of the whole organism), have to be considered. (I am straining the meaning of the word fibre, making it stand for any kind of nervous pathway ensuring physiological union, definite or indefinite.) Consideration of these connections is essential for clear ideas of the full process in fits, the only visible part of which is convulsion; it is especially important with regard to the way by which in epileptiform seizures a very local (a "doubly local") discharging lesion causes wide-spreading and even universalisation of convulsion. Again, the study of the interconnections of the levels is a necessary preliminary to the comparison and contrast of the effects of "discharging lesions" and of "destructive lesions," an essential thing in the scientific investigation of diseases of the nervous system, as I urged in my Gullstonian Lectures (1869). I speak only of connections of motor centres of the levels.

Each level being bilateral is a twin series (right and left) of centres. There are connections (commissures) between "identical" centres and between "non-identical" centres of its two halves—presumably between centres of the two halves as they correspond for co-operation of the parts of the body they represent in joint operations by the two sides of the body. There are also connections between the centres making up the lateral half of each level. The fibres of the two connections spoken of are intrinsic fibres of levels. I speak next of Extrinsic fibres—that is, of those interconnecting levels. Considering for a moment all the levels, the motor path,¹¹ or, as I shall say, kinetic route, extends from the highest motor centres to the muscles, which in a certain regard, being dischargeable, are centres too. This route (strictly the three series of motor centres are parts of the kinetic route) is in three segments: from highest to middle centres, first segment; from middle to lowest, second segment; and from lowest to muscles, third segment. I can, however, consider in detail only the second segment of the kinetic route, that connecting the middle and lowest levels. I shall, for convenience, speak of the motor centres of the right half of the middle level. There are three sets of motor fibres, kinetic lines of the second segment, uniting all right middle motor centres to all lowest motor centres—at least to all motor centres of the left half of the lowest level, if not, as I imagine, to those of the right also.

First Set of Fibres of the Second Segment.—Those which have been traced (on the Wallerian method by Charcot and others) from the right "motor region" along the right corona radiata, right internal capsule, through the right crus cerebri, right halves of pons and medulla into the (left) lateral column of the cord as low, Sherrington has found in one case, as the origin of the coccygeal nerve roots. These fibres are (and so are the second and third sets) extrinsic of the levels; they belong to neither level, and yet they belong to both in the sense of interconnecting the two.

Second Set of Fibres.—Those of the direct pyramidal tract. They have been said to be traceable no lower than the mid-rorral region. Tooth has traced them by the Wallerian method in one case as low at least as the second lumbar; these are fibres of the inner part of the right anterior (Turck's) column.

Third Set of Fibres, comparatively recently (1884) discovered by Pitres, and seen by Schäfer, Sherrington, Hadden, Tooth, France, and others.¹² These fibres have been traced, on the Wallerian

¹¹ Dr. Gowers (*Dis. of Nervous System*, vol. i, p. 118) gives a diagram of the "motor path." He makes two segments, "cerebro-spinal" and "spino-muscular." The kinetic route is a modification of his scheme.

¹² Mr. E. P. France (*Phil. Trans.*, B. 48, 1889) has not found after lesions of the marginal convolution in monkeys (made in some very important researches by Schäfer, Horsley, and Sanger-Brown, *Phil. Trans.*, B., 1888), nor in any other case in these animals degeneration of the direct pyramidal tract; but in all cases in which the degeneration in the crossed pyramidal tract was well marked he found degeneration much less in amount, but in the same position, in the other half of the cord (side of lesion). Horsley and Schäfer have, so to say, completed the "motor region" by their discovery of trunk centres in the marginal convolution.

¹⁰ Horsley (*Lancet*, December 25th, 1886, abstract of Brown Lectures) believes that "all the convulsive, tonic, and clonic phenomena may originate from the ordinary bulbo-spinal centres such as exist for carrying out normal mechanisms. Tonic or clonic spasm, then, may be produced by any motor centre, but the combination and sequence of tonic-clonic could originate only from the cerebral motor cortex." This is important with regard to the question of ponto-bulbar fits.

method, into the right lateral column.¹³ The degeneration in the (right) lateral column in cases of (left) hemiplegia is recognised by Charcot. So we see that the interconnection of the middle and lowest motor centres is very complex.

Some years ago¹⁴ I inferred from the then known connections of the right corpus striatum (internal capsule) with both halves of the cord (by the first and second set of fibres) that both sides of the body are represented in the right half of the brain (I still say "right" for convenience), but the degenerated fibres being of different columns (the left lateral and the right anterior), that the left and right sides of the body were differently represented in the right half of the brain. (This, I have since stated, seems to me to be but an expansion and modification of the principle of Broadbent's well-known hypothesis as to the double representation of the bilaterally acting muscles.) I should have thought the discovery of the third set of fibres rendered my hypothesis more tenable. But there are serious difficulties. The second set of fibres (those found in the right anterior column) are supposed by some great neurologists to cross to the left half of the cord.¹⁵ The same has been said of the third set of fibres found in the right lateral column. So that it may be that all three sets of fibres pass from the right middle to the left lowest motor centres. It is, however, not really known where the second and third sets of fibres end. No wasted fibres are found in the anterior commissures. Hence, I provisionally keep to the hypothesis mentioned, and now say regarding it that the three sets of fibres may show that the right middle motor centres are connected with lowest motor centres of the left and right halves of the lowest level, and thus that the right middle motor centres represent, by intermediation of the twin lowest motor centres, movements of muscles of both sides of the body. The expression "movements of muscles" introduces a matter which must be considered before I can illustrate the hypothesis.

Having considered the whole central nervous system with the rest of the body represented by it to be a sensori-motor mechanism, I now wish to urge that the motor centres of every level represent movements of muscles, not muscles in their individual character. The same muscles, that is, all the muscles, are represented in Simplest, in Complex, and in Most Complex movements by respectively the lowest, middle, and highest motor (better "movement") centres. If so, it is possible, as I believe happens in hemiplegia, to lose one series of movements of muscles, and to retain another series of movements of the very same muscles. Thus, to take a case of left hemiplegia in which the right middle motor centres, if not destroyed, are cut off from lowest motor centres (to neglect the second and third sets of fibres), the condition is not properly described as "loss of power of the muscles" of the left arm and leg, but as loss of complex movements of the muscles of those limbs. The simplest movements of the very same muscles remain represented by the left lowest motor centres. The muscles certainly are unaffected, and when rigidity comes on the simplest movements of them are developed by over-activity of their lowest motor centres. To repeat, there are, in cases of hemiplegia with rigidity, the double opposites, loss of complex movements and over-development of simplest movements of the very same muscles. The fixed rigid state of the left arm and leg, which clinically we speak of as a symptom, as if it were something *sui generis*, is only the algebraical sum of the co-operating and antagonising "pulls" of the simplest movements of the muscles of these limbs. If the hemiplegia be very slight, we may say that the muscles of the left arm and leg "are only a little weak," but the strict description is that there is then loss of but a very few complex movements of all the muscles of these limbs, with persistence of the rest of the complex and of all of the simplest movements of those same muscles. Similarly, convulsion is not to be looked on as convulsion of muscles; in an epileptiform seizure the convulsion is a contention of complex, and also of simplest, movements. In this contention the in-

dividuality of each movement is lost. I will consider a more complex case, that of a man imperfectly hemiplegic permanently, and yet subject to occasional epileptiform convulsion of the region paralysed; the paralysis is loss of but some of the complex movements of all the muscles of the arm and leg, and there is persistence of the whole of the simplest movements of all those muscles; when the fit comes there is a temporary contention of the remaining complex movements and of the simplest movements of all those muscles.¹⁶

The distinction between muscles and movements of muscles is exceedingly important all over the field of neurology; I think the current doctrine of "abrupt" localisation would not be so much in favour if it were made. The occurrence of convulsion of a muscular region which is already imperfectly and yet permanently paralysed, is unintelligible without that distinction. And without it we shall not understand how it can happen that there is loss of some movements of a muscular region without obvious disability in that region. This bears closely on the realistic study of the physical conditions in aphasia and insanity. We are concerned with both these morbid affections in this inquiry. The anatomical loss in cases of aphasia is of certain complex movements of the tongue, palate, etc.; there is a paralysis in that sense. In cases of insanity (post-epileptic states, for example) there is, I submit, corresponding to the negative element of the psychical symptomatology, paralysis in the sense of loss of some of the most complex combinations of impressions or of most complex movements, or of both; for one example of those most complex, etc., movements of the hands, which are represented in the anatomical substrata of tactual ideas. In brief, a negative lesion of any part of the nervous system ("organ of mind" included) causes, and always causes, paralysis in the sense spoken of, sensory or motor, or both, and causes nothing whatever else.

It may seem absurd to say that when there is no obvious disability in a muscular region, there may be loss of some movements of that region. This is, however, what I do affirm confidently. We shall be particularly concerned with this dictum when dealing with post-epileptiform aphasia. An eminent physician, referring to a previous statement I made¹⁷ of the dictum, described my aim as being to prove two things—that a motor centre does and also that it does not represent movements. I never consciously attempted that very marvellous feat. Then, as now, I am content with the supposition that the non-disability of a muscular region when some movements of it are lost by destruction of part of a centre is accounted for by that muscular region being represented by other movements in other parts of the centre or in other centres. On this supposition of Compensation, we can explain recovery from hemiplegia (without relying altogether on the hypotheses of subsidence of "shock" or diminished pressure) dependent on very small destructive lesions; there is recovery when a few movements are permanently lost. That the seeming paradox holds for some cases is undeniable, as the experiments of Semon and Horsley on the cortical representation of the vocal cords show. These experiments give a crucial verification of Broadbent's well-known hypothesis for one case. Moreover, France¹⁸ has found degeneration in the lateral column of the cord after lesions of the gyrus fornicatus in monkeys. This shows, by the way, that that gyrus, although no doubt (Horsley and Schäfer) mainly sensory, is not purely sensory. Although in the monkeys mutilated as mentioned, there is no discoverable disability in any muscular region, I submit that the degeneration France describes is proof of loss of some movements, skeletal or visceral, or possibly of negative movements (inhibitory). It would be remarkable if there were any conspicuous disability, considering the enormous compensation given by the intact "motor region." I can now return to the question of representation of both sides of the body in each half of the brain.

I suppose that the right "motor region" represents complex movements of muscles of both sides of the body (1) as they serve in bilateral actions, (2) as they serve in alternate actions (or, I should

¹³ They were called by Sherrington "re-crossed fibres;" but he now thinks (*Journal of Physiology*, January, 1890) that this name is unsuitable. Sherrington, in a case of small and superficial lesion of one-half of the brain at the lower end of the fissure of Rolando has traced degenerated fibres into both halves of the pons and medulla.

¹⁴ *Med. Times and Gaz.*, October 23rd, 1889. In the abstract of one of my Gullstonian Lectures (1889), after reference to Broadbent's hypothesis, there appears: "Taking one side of the brain, the right, the lecturer thinks the muscles acting unilaterally, both of the left and of the right side of the body, are represented in the right side of the brain, but that the muscles of the left side of the body are especially represented there. 1st. More in quantity, for they are more affected when the hemisphere discharges. 2ndly. First in time (instability), for they are affected before those of the right side."

¹⁵ Gowers, *Diseases of Nervous System*, vol. i, p. 114.

¹⁶ The distinction between movements and convulsion is of extreme importance, and is not always made. In some slight epileptic fits there are mixed up, so to say, with convulsion movements properly so called, as those of chewing, spitting, clutching the throat, etc. It is very necessary not to mistake writhing movements of the arms with suspended respiration for convulsion of those limbs. I regret that time will not allow me to consider this part of my subject, for the question raised bears closely on the interpretation of post-epileptic mania, and thus indirectly on the study of insanity in general.

¹⁷ *JOURNAL*, May 10th, 1873.

¹⁸ *Phil. Trans.*, B. 41, 1889.

say, with preponderance of activity on one side), and (3) as they serve in unilateral actions. But I suppose, too, that those movements which are "bilateral" are represented most nearly equally in right and left halves; that those which are "alternate" are represented less equally in the two halves (those of the left side more in the right half), and that those which are "unilateral" are represented most unequally in the two halves (those of the left side most in the right half). We must bear in mind that movements are spoken of, for the so-called alternate muscles may serve in bilateral movements; they may serve with a great degree of preponderance on one side approaching unilateral movement; and the most unilateral muscles may serve in bilateral movements.

It may, however, be asked, Where is the paralysis corresponding to abrogation of the second and third sets of fibres from the right "motor region?" The existence of some weakness of the right side (the so-called non-paralysed side) has been urged by Brown-Séquard, Pitres, Friedlander, and Gowers; but when in the difficult circumstances attending investigation of the state of the right side no disability is demonstrable, there may yet be loss of some few movements of the muscles of that side. The loss of these few movements is masked by the compensation given by the left half of the brain, which represents most numerous movements of the right side. If we take the case of excessive discharge beginning in some part of the right middle motor centres, we find that, in severe epileptiform fits, both sides of the body are convulsed. In this case compensation is "inverted," that is to say, centres which would compensate a loss are compelled to co-operate in an excess. The comparison and contrast of hemiplegia and an epileptiform seizure are, however, by no means simple, as we shall see when the process of universalisation of an epileptiform convulsion is considered.

Although when speaking of the hierarchy of nervous centres I excluded from consideration the higher divisions of the cerebellar system, it is necessary to say something of them. There are in some cases of tumour of the median lobe of the cerebellum, seizures very like, if not quite like, those of ordinary surgical tetanus. I have suggested that the convulsion of surgical tetanus is owing to cerebellar discharges; but no doubt the poison causing that morbid affection affects lowest centres too. It has been suggested that the tetanus-like seizures in the cases of cerebellar tumour mentioned, are owing to pressure upon, or to changes induced in, the corpora quadrigemina or medulla oblongata, or both. But surgical tetanus is, at all events, in order of development of spasm over its regional distribution the "complementary inverse" of epileptiform seizures. And when we consider nervous diseases, each as a flaw in a whole nervous system, the study of "corresponding opposites" is most important. There is a more direct reason for taking note of the cerebellum in the present inquiry. What the structures are connecting the cerebellum with the lowest motor centres I know not. If there be a connection—if the lowest level is at once the lowest level of the cerebral and of the cerebellar system—we may expect the higher levels of the cerebellar system to be concerned in post-epileptiform states; the question is important, especially with regard to increased tendon reactions in post-epileptiform paralyses. I have suggested¹⁹ that the cerebellum is concerned with the rigidity and exaggerated tendon reactions of (left) hemiplegia²⁰ when that paralysis is the result of a destructive lesion of the (right) internal capsule, that there is not only exaltation of function of the (left) lowest motor centres from loss of control, but also that the cerebellar influx upon them is no longer antagonised. This hypothesis has been objected to on the ground that in transverse lesions of the upper dorsal or cervical cord (the cerebellum being thus excluded) there is nevertheless rigidity of the legs with exaggerated knee-jerk and foot clonus. I was obliged to admit that the cerebellum is not necessary for the production of the exaggerated jerk and rigidity.²¹ But Charlton Bastian,²² who has advanced proof where I only speculated, has found that, in man, on complete transverse lesion of the regions of the cord mentioned, the muscles of the legs are flabby and the knee-jerks absent; what is very striking, he finds that the condition of rigidity of the legs and exaggerated knee-jerk in cases of incomplete transverse lesions changes to one of flaccidity with no tendon reactions when that lesion becomes

completely transverse. If Bastian be correct, as I think he is, the hypothesis of cerebellar influx, on which we are in fundamental agreement, is supported.

THE TREATMENT OF SUPPURATIVE INFLAMMATION OF THE MASTOID PROCESS ASSOCIATED WITH DISEASE OF THE MIDDLE EAR.

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My object in writing this short paper is to advocate no very novel mode of treatment, but to illustrate by a few cases the advantage of adopting as early as possible such radical surgical measures as not only remove the patient from the risk of septic infection and intracranial suppuration, and prevent the possibility of a recurrence of the inflammation in or about the mastoid process, but also serve to cure the inflammation which has become chronic in the middle ear and probably influenced the function of the internal ear, and in a very considerable proportion of cases to restore to the patient an amount of hearing power so great as to render any deficiency imperceptible to a non-professional observer. This last advantage has impressed me very greatly, imbued as I was with the belief that the presence of the small bones of the middle ear and the membrana tympani were necessary for fairly acute hearing. Over and over again, as the following cases will show, patients have, after the removal of the mastoid antrum and the contents of the middle ear and the subsidence of the inflammation, recovered very good hearing power. These cases will also serve to illustrate the frequent presence of a large antral cavity in such as suffer from chronic abundant middle ear discharges, and especially when associated with inflammation of the mastoid process.

Given a large antrum filled with foul caseous and purulent material, and discharging its overflow of extremely irritating material into the sensitive mucous lining of the middle ear, it is perfectly obvious that this condition cannot subside or disappear if left to Nature, while its presence is not only a source of great danger to the life of the individual, but also to the integrity of the internal ear. This antral cavity, when well developed, is of considerable size, and is in immediate relation with the posterior and middle fossae of the skull, the lateral sinus, the middle ear, the external auditory meatus, and the outer surface of the skull, in all of which directions pus may make its way through an aperture in the bony wall of the cavity, or it may determine the development of an acute suppurative periostitis between the cartilage of the auditory meatus and the bone or over the outer aspect of the process, or between the dura mater and bone in the middle or posterior fossa, and associated or not with the last it may produce thrombosis of the lateral sinuses. I am convinced that it is this antral distension which forms the chief source of the above, and that it is comparatively rarely that the penning up of pus in the middle ear is the chief factor, for the reason that the external auditory meatus is not easily completely occluded.

The mastoid antrum varies within very wide limits, not only in form and size, but in its relation to adjoining surfaces. It would appear, as one would expect, that suppuration in and about the mastoid process takes place more readily in such subjects as have well-developed antral cavities, and this is especially the case in those who are affected by recurrent attacks of inflammation. Pathologists appear to be unaware of the importance and the anatomy of the mastoid antrum. How rarely does one see its condition described in the record of a fatal case of intracranial mischief. Usually the observer is satisfied with opening the roof of the middle ear and finding pus, or with exposing the mastoid cells in the apex of the process. My experience would lead me to conclude that the antrum is a much greater source of danger to the cranial contents than are the mastoid cells.

As I have already stated, by no means the least advantage of the operation to the patient is the opportunity it gives of removing a *cul-de-sac*, which without operation could never be drained, and without the removal of whose foul and irritating contents the inflammation of the middle ear, and the absence of function of the internal ear, could never be removed. It also enables the middle

¹⁹ *Medical Examiner*, April 5th, 1877, and March 29th, 1878.

²⁰ Not rarely both knee-jerks are exaggerated; this perhaps helps to show that the third set of fibres ends in the right lowest motor centres.

²¹ *Medical Times and Gazette*, February 12th, 1881.

²² *Quain's Dictionary of Medicine*, p. 1481.