THE LUMLEIAN LECTURES ON CONVULSIVE SEIZURES.

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LECTURE III.

After-Effects of Epileptiform Fits.—Paralysis due to Cortical Exhaustion.—Analysis of a Case.—Variations in Degree and in Range of Post-epileptiform Paralysis.—Hemiplegia, Post-epileptiform and from Destruction of Motor Part of Internal Capsule contrasted.—Post-epileptiform Defects of Speech.—The Negative and Positive Elements of these Defects.—The Psychological and Physical.—The Concern of the Right and of the Left Half of the Brain in Speech.

I HAVE now to consider the After Effects of excessive nervous discharges in cases of epileptiform seizures. This part of our subject is of extreme importance; the presumption is that from the study of the comparatively simple post-epileptiform conditions we shall obtain a basis for the interpretation of post-epileptic conditions. It is for this reason that I go into some detail as to the former.

There is not rarely after epileptiform seizures local temporary paralysis;¹ and sometimes aphasia with it. For the present I speak of paralysis only. In this inquiry we have no direct concern with paralysis, temporary or permanent, owing to destruction (by clot, softening, or any other pathological process) of any part of the brain, not even if the malady set in with a convulsion.

I must particularly mention cases in which there are both (1) permanent paralysis—for example, imperfect hemiplegia; and (2) occasional epileptiform seizures, the convulsion beginning in some point of the muscular region paralysed and involving part or the whole of it, and sometimes also parts beyond that region. In these cases the two opposite functional states—destruction of some nervous elements which represented complex movements, and high instability of some others which still represent other complex movements of the same muscles—are side by side in one part of the cortex (or the latter of cortex and the former of fibres passing down from it). After a paroxysm in such a case there may be temporary increase of the paralysis. For my present purpose, which is only an attempt to explain the nature of the sequence (paralysis after convulsion), I shall exclude these cases, and deal only with what I may call uncomplicated cases—that is, with those in which there is only paralysis immediately when epileptiform seizures have ceased, such paralysis being temporary (unless, of course, the fits recur at short intervals). Something has to be said, however, regarding certain of the so-called uncomplicated cases.

In some cases in which there is no discoverable paralysis in the intervals of the fits, except for a short time after one, there may be indirect evidence of a negative functional state of a few fibres of the second segment of the kinetic route. That evidence is foot clonus and exaggerated knee-jerk on the same side as that in which the convulsion occasionally starts. I mean, of course, cases in which these abnormal tendon reactions are producible at all times, although in a greater degree just after a fit. It may be that severely careful tests would show some *quasi*-trifling paralysis—loss of a very few complex movements. I shall here ignore this slight inferential paralysis, and speak of such cases as “uncomplicated.”

The term post-epileptiform paralysis will be used to include all paralyses the immediate sequels of epileptiform seizures. Arbitrary divisions may be made, speaking of range only. (1) Terminal paralysis, as of a hand; (2) monoplegia, as of an arm; (3) hemiplegia; and (4) a range, which is not generally admitted—some degree of slight universal paralysis. I hold the hypothesis (essentially that of Todd and Alexander Robertson) that there is exhaustion of central nervous elements, including fibres of the second segment of the kinetic route, and that this is produced by

the sudden and excessive discharge in the prior paroxysms. These nervous elements are exhausted, not otherwise injured, so that recuperation is prompt, and the corresponding paralysis is temporary. There is a sequence of two opposite functional states, superpositive of the first degree (a) in the paroxysm, and then, after the paroxysm, negative. But there is often more than a negative functional state in the post-epileptiform condition. We may find increased tendon reactions implying a superfunctional state of the second degree (b) of lowest motor centres. The duplex nature of these post-epileptiform conditions must be borne well in mind.

The term “exhaustion” has been objected to; general bodily prostration is, of course, not meant; but, as said, exhaustion of nervous elements in a particular part of the central kinetic route. There are, I suppose, degrees of exhaustion, and no doubt of different numbers of nervous elements of the kinetic route in different cases. But illustrating by the extremest degree, the supposition is that nervous elements of the route, after their excessive “exercise” in the fit, are left “fatigued” to the degree of utter impotence. The nervous elements exhausted in post-epileptiform paralysis are, I suppose, in the same state as are the motor nerve fibres of the sciatic nerve going to the cut-off leg of a frog after strong faradisation of that nerve trunk; the leg is convulsed by the faradisation (stage analogous to epileptiform seizure), and is next paralysed (stage analogous to post-epileptiform paralysis) because its motor fibres are exhausted by the unnaturally high functioning they have been artificially compelled into. A closer analogy may be stated, although the case to be instanced is a complicated one. The motor nerves of a frog, poisoned by a very large dose of strychnine, lose function partly by the direct action upon them of that poison, but partly (this is what is relevant) through exhaustion—“the exhaustion of over-use due to the intense activity of the nerve during the stage of spasm.”²

Not only the term, but the hypothesis itself is objected to. I will consider other hypotheses. The paralysis has been ascribed to cerebral congestion consequent on arrest of respiration in the preceding seizure. It is difficult to see how cerebral congestion, even if we grant that it can produce paralysis, could be so exquisitely local as to produce what we sometimes observe, paralysis of one arm only, or even of but part of one. Again, absolute paralysis of a limb is found after seizures nearly limited to that limb, in which seizures there was no arrest of respiration and (if that is thought to bear on the question) no loss nor even defect of consciousness; the patient may talk throughout an attack in which an arm is involved, and after which it is temporarily paralysed. There is the hypothesis that the temporary post-epileptiform paralysis is owing to a small extravasation of blood in the brain, caused, I suppose it is meant, by congestion consequent on arrested respiration in the preceding seizure. I admit that very small clots may cause local paralysis, and that such paralysis may be transitory. But the post-epileptiform paralysis is—that is in my experience—always of the parts which were first and most convulsed in the prior fit; it would be marvellous if a small hæmorrhage happened locally in one-half of the brain, so as to produce temporary paralysis of the parts first and most convulsed, and happened in the same place in every fit the patient had. Besides, if arrested respiration could cause cerebral hæmorrhage by leading to sudden great congestion of the brain, we should find very local paralysis after fits of the epileptic kind, and we do not; according to current opinion there is no paralysis at all after seizures of this kind. I shall exclude the two hypotheses just dealt with.

Post-epileptiform paralysis has been ascribed to inhibition by some medical men whose hypotheses deserve respectful consideration. Gowers believes that discharges in epileptic fits sometimes inhibit; he thinks that temporary paralysis is found in some cases after a purely sensory discharge which does not next discharge motor centres, but inhibits them. In some cases of epileptiform seizures the patient tells us that his arm “falls dead,” there being no spasm in it, whilst the face of the same side is being convulsed. This, so far as I can learn, is a paroxysmal, not a post-paroxysmal paralysis, but possibly it remains for a short time after the convulsion of the face has ceased. I express no decided opinion as to the validity of the inhibition hypotheses. I think it possible that there may be discharge spreading slowly in a motor centre of the middle level, excessive enough; to cause slight after-exhaustion of some of its elements, although not one strong enough to overcome the resistance of lowest motor centres, and thereby to produce actual convulsion. I have spoken of “feelings

¹ This paralysis was long ago described by Todd under the name “Epileptic Hemiplegia.”

² See Wood's *Therapeutics*, 7th edition, pp. 258-9.

of convulsion" in which there is no actual convulsion, but do not know whether there was any degree of post-paroxysmal paralysis of the parts "ideally convulsed." I go on to consider how far the hypothesis I hold, essentially that of Todd and Robertson, accounts for the facts of different cases.

There is exhaustion of the cortex after artificially induced epileptiform fits in some lower animals, as Franck and Pitres³ have shown; they use the term "épuiement cortical post-épileptique." They write: "Le phénomène de l'épuisement cortical consécutif aux accès d'épilepsie partielle (what I call epileptiform seizures) est très facile à constater." The "épuiement" is transitory; it lasts a quarter or half an hour. By artificial excitation of the part of the cortex in question, as the exhaustion diminishes, simple movements are first producible, and later epileptiform seizures, but not, for some time, is an attack provoked so intense as before the "épuiement." I think that the researches of these distinguished physicians countenance the inference I have drawn that the exhaustion in post-epileptiform conditions, although local, is yet widespread in the "motor region;" that besides "running down" of the cells of the fulminate there is also running down of the collateral normal (stable) cells which were compelled by the fulminate to discharge excessively. Franck writes: "Cet épuiement est tout local, non point qu'il se borne exclusivement à la portion circonscrite du champ moteur excité en premier lieu, mais il est limité à la zone motrice de ce côté." Exhaustion in the sense of general bodily prostration is not meant, for Franck and Pitres point out that excitation, without effect on the exhausted part of the motor region, when transferred to the motor region of the opposite hemisphere, produces severe convulsions.

I will here mention an objection to the hypothesis of exhaustion made to me by a physician whose opinion I respect highly, that during post-epileptic coma there may occur a convulsion affecting all parts of the body, which I say are then paralysed. But what I mean by paralysis in post-epileptic coma is loss of some most complex movements, represented by the highest centres of all or very many parts of both sides of the body; there is retention of other most complex movements represented by those centres; most of the complex (middle centres) and simplest movements (lowest centres) being also retained. Further, it is quite certain that an arm which the patient is quite unable to move in the slightest degree after an epileptiform seizure may be suddenly seized with another severe convulsion (I am supposing a case in which neither in nor after the paroxysm consciousness is lost). This, however, is only reasoning by analogy, and I admit that the interpretation of the last-mentioned case is to me most difficult.

I will now give an illustration of post-epileptiform paralysis. It may be taken to be an artificial one, but it is essentially that of a patient whose case I reported.⁵

A man, B., was *seemingly* (1) quite well when he arrived at my house after a walk of about a mile. (It is convenient to speak of this as the "first stage," not, of course, of the fit, but of the dramatic occurrence I am relating.) (2) (a) A fit began in the toes of his left foot.⁷ (b) The whole leg was gradually involved, the spasm passing up the limb. In about eight or ten minutes the convulsion ceased, when (3) the leg was found to be (a) paralysed, and (b) there was exaggeration of its knee-jerk and clonus of the foot. (4) In six hours or less he was *seemingly* well again, certainly he was then rid of his paralysis; and next day (I did not test his tendon reactions before) his knee-jerks were normal and there was no foot-clonus. Let me consider the four acts of this drama.

(1) The patient was not really well when he arrived at my house. He had a persistent discharging lesion, presumably of a few cells of his leg centre (perhaps only of some of those of the hallux centre); so to say, he always carried it about with him, or, to speak more precisely, it was a persistent *quasi-parasitical* hyper-functionable part of himself.

(2) (a) The discharging lesion did function and that excessively, and possibly produced the initial spasm (of the toes) by its sole discharge; but (b) next, as a fulminate, by overcoming the resistance of (discharging) other cells of the leg centre it compelled these normal stable cells to discharge—compelled them to co-operate in its

excess.⁸ So much for the discharges (primary and secundo-primary) of the cortex (middle motor centres). That there was a super-normal activity from cortex excessively discharging to and of the muscles of the leg convulsed is certain. The route of the numerous and rapidly succeeding nerve impulses from the hyper-physiological cortical process would be certain fibres of the corona radiata, of the internal capsule, of the crus cerebri, pons, and medulla of the right side, then of fibres of the left lateral column (possibly also of the second and third sets of interconnecting fibres, both of which I shall ignore here) up to the anterior horns (some lowest motor centres); so far the second segment of the kinetic route is concerned. The resistance of the cells of these lowest motor centres would be overcome (secondary discharges), and the impulses (presumably increased in number) would then be of fibres of the nerve roots from those centres, thence of the continuing fibres of nerve trunks and their branches to the end plates of the muscles (third segment of the kinetic route); the resistance of the muscles would be overcome, and there would finally be great "explosive decomposition" of muscle substance (tertiary discharges).

(3) The paralysis signified exhaustion of nervous elements previously excessively functioning in the paroxysm. There would be exhaustion of cells of the cortex, not only of those of the discharging lesion, but also of those collateral stable cells which it, as a fulminate, compelled to discharge excessively. There would be exhaustion, too, of the fibres passing down from both sets of cells. The hyper-kinetic route, or at least part of it, would now be a hypo-kinetic tract. There would be exhaustion as low down as certain lowest motor centres; these centres were therefore exalted in function (second degree of superpositive functional change), hence the increased tendon reactions.

(4) The paralysis had passed off; the patient could walk well. Next day the knee-jerks were normal. There was recuperation of the exhausted nervous elements. No doubt the cells of the discharging lesion (after the fit, stable below normal) began at once by gradual morbid nutrition to re-attain high tension and very unstable equilibrium—"began to prepare for the next fit."

So far as I have seen, post-epileptiform paralysis is always most of the parts which were first and most convulsed in the preceding paroxysm, as in the case of the patient B. This correspondence is denied by some eminent physicians; further observations will settle the matter. It is an important one, for, according to the view I take, post-epileptiform paralysis adds no evidence to that afforded by the prior convulsion as to the seat of pathological changes in the cortex. If, however, there is not the correspondence I suppose, the case is very different and the situation is a most difficult one.

I think there is a relation of proportionality between the severity of the epileptiform fit (severity of the discharge) and the subsequent temporary paralysis (amount of exhaustion). I should have thought this a truism if it had not been expressly denied. It is averred that whilst after the epileptiform kind of seizure there is often decided paralysis, there is not, as a matter of fact, any whatever after the severer fits of the epileptic kind. I demur to the "fact," believing that whilst after a limited epileptiform seizure there is often great *local* paralysis, loss of *many movements* of a small part of one side of the body, there is after a severe epileptic fit *widespread* paralysis, loss of *few movements* of most, if not of all, parts of both sides of the body. Indeed, I submit that the whole condition of bodily impotence after a severe epileptic fit is paralysis, and that, speaking gene-

⁵ In the real case (*op. cit.*) the left arm was slightly convulsed. The current hypothesis would be that, in causing this part of the fit, the discharge spread from the leg centre to the arm centre. This I cannot disprove. Yet I think it an equally legitimate hypothesis that the discharge causing the slight movements of the arm in this case was of those elements of the "leg centre" representing subordinate movements of the arm. No doubt, of course, in a severer fit other centres of the motor region would be discharged. As implied in several parts of these Lectures, I do not accept the current doctrine of Localisation. The minute investigations of the monkey's cortex by Horsley and Beavor go strongly against it. To me the "leg centre" is only a part of the "motor region" where most special movements of the leg are represented, and where subordinate movements of the arm and of other parts of the body are also represented (the same, *mutatis mutandis*, for other centres). The researches of Sherrington (*Journ. of Physiology*, vol. x, No. 5) seem to me to be counter to the current doctrine. He writes, ".....after lesion in the leg area, encroaching little if at all upon the cortex of the arm area, the degeneration in the cord stopped short in great part in the cervical region, especially in the cervical enlargement. Again, after lesion in the arm area, encroaching little if at all upon the cortex of the leg area, the degeneration in the cord extended down through the dorsal into the lumbar and even throughout the sacral region of the cord." He suggests, however, that the fibres descending from the arm area into the lumbar region are visceral.

³ *Arch. de Phys.*, 1883.

⁴ *Op. cit.*

⁵ *Fonctions Motrices du Cerveau*, pp. 90, 91.

⁶ *Medical Times and Gazette*, February 12th, 1881.

⁷ Referring to the real case (*op. cit.*) the patient said that in all his seizures the first "sensation" was to the outer side of the ball of the great toe; very likely I missed a very earliest stage, one possibly of spasm limited to the big toe.

rally, there is really more paralysis than is found after severe epileptiform seizures.

The relation of an epileptiform fit to the sequent paralysis, or, more exactly, the relation of the two opposite functional states, discharge, and subsequent exhaustion, is not a simple relation.

According to the hypothesis I hold, there should always be some, however little, paralysis after an epileptiform seizure, at least some in the parts first and most convulsed, however slight the attack may have been. Trifling degrees of paralysis are easily overlooked. This deserves remark. Once more I urge that more or less paralysis from negative central lesions is always loss of more or fewer movements; it is not to be thought of merely as *loss of power in muscles*. There may, for example, be loss of the most special (the most "delicate") movements of the muscles of the hand with retention of other, the next most special (the "coarser") movements of those muscles. I will illustrate by a case of imperfect post-epileptiform paralysis.

After a slight epileptiform seizure starting in the hand, the patient may have difficulty in picking up a pin, and may yet be able to grasp strongly. There is clumsiness of movement, or perhaps some may say "loss of muscular sense." These terms must not let us overlook the fact that the slightly abnormal motor condition of the hand is a double condition, one of two opposite elements. There is in the case supposed difficulty in picking up the pin, because a few most special movements of the hand are lost (so far paralysis, negative element); the pin has to be picked up by the next most special movements remaining, which are *now* the most special of the movements (positive element); they do not serve so well as the most special would have done; hence the operation is "clumsy."

From such *quasi*-trifling paralysis as that indicated, loss of a few most special movements of the muscles of the hand, there are met with after severer epileptiform seizures beginning in the hand degrees of loss of next and next most special (or, equivalently, of the next and next more general) to loss of all movements of the muscles of it, and of some movements of the rest of the arm too; there is then no "clumsiness," for no movements of the hand remain to do anything.

The last remark introduces another part of our subject. It implies that post-epileptiform paralysis varies in a double way, (a) in degree (number of movements lost) and (b) in range (number of parts of which movements are lost). I think, in harmony with what was said in Lecture II, on compound order of spreading of convulsion, that degrees of difference between what may be called little and great paralysis after epileptiform seizures are not simple degrees of more or less amount of paralysis, nor simple degrees of more or less range; there are degrees of both, there are differences of Compound Degree. After the slight epileptiform seizure alluded to in the foregoing, there was slight paralysis limited to the hand. If that patient had had a somewhat severer fit, he would have had after it more paralysis of the hand (loss of more movements of it) and greater range of paralysis (loss of some movements of the next part, say the forearm).

That there was exhaustion lower than the cortex in B's case, exhaustion also of fibres of the second segment of the kinetic route, that is, up to the lowest motor centres, is signified by the exaggerated tendon reactions. I have spoken of the lowest motor centres and of the third segment of the kinetic route (fibres from them to the muscles) and of the muscles as not being exhausted in B. at the time when his leg was paralysed. It would, however, be remarkable if a few elements of these lowest parts of the kinetic route were not hypo-kinetic to some trifling degree after the hyper-kinesis during the convulsion—if there were not after their excessive exercise some exhaustion of a few of their elements. No doubt, being more strongly organised structures, the lowest motor centres and muscles are less easily exhausted by the discharge, and, if exhausted, recuperate soonest of all parts of the

⁹ Here is an illustration of the principle of duplex symptomatology of nervous maladies, and that the positive element is often owing to activity or to over-activity of nervous arrangements untouched by any pathological process. The movements by which the pin is picked up are the outcome of activity of healthy nervous arrangements. To blame these, to speak figuratively, for the clumsiness of the operation is to ignore that they are doing their best in the evil circumstances. The principle is most important, if it applies, as I think it does, throughout the nervous system, from the symptomatology of a case of paralysis of an ocular muscle to that of cases of insanity; in the former case it is quite certain that most of the symptomatology answers to activity of perfectly healthy nervous arrangements of the highest centres; the negative mental symptoms in cases of insanity alone correspond to disease; they signify dissolution of part of the highest centres; the coexisting positive mental symptoms signify evolution going on on a lower level of those centres. The very same principle is displayed in cases of aphasia.

route. After some epileptic fits (Westphal, Gowers, Beevor) the knee-jerks are temporarily lost; in these cases some lowest motor centres, the lumbar nuclei (as Gowers suggests) are probably temporarily exhausted. For my part, I think it likely that during the post-epileptiform paralysis in B., although his knee-jerks were exaggerated, there was some trifling hypo-kinesis of the lowest part of the route, that the hypo-kinetic tract was from cortex up to and of the muscles, although, no doubt, elements of the cortex and of the first segment of the kinetic route were most exhausted. The muscles in such cases act to faradisation (I did not test B. this way) as well as do those of the fellow leg. But if we were to take a case of paralysis, such as that of a man a few years ago under my care who had nineteen hundred and forty-five fits—each beginning in his left thumb, involving the left arm, fixing his chest, and turning his head to the left—in about fourteen days, and whose left arm was absolutely paralysed in the intervals, we might find by fine testing (what we did not find in that patient by ordinary testing) some slight degree of reduction of faradic reaction of the muscles, or, if not, of reduction of faradic or galvanic reaction in their nerve-trunks, of the part paralysed. In B. the exaggeration of the knee-jerk would not show that *all* the cells of the anterior horn which is concerned with that jerk were intact; a few of the smallest cells may have been exhausted.

In a case of amyotrophic lateral sclerosis there is wasting of some cells of anterior horns (hence the atrophy of some muscular elements) and increased activity of other cells of the same horns (hence the increased tendon reactions of the same muscles). This case shows that the functional condition of different cells of the same anterior horns may be diametrically opposite, and that the symptomatology in the muscular region which the horns supply may be of two correspondingly opposite elements. I shall put aside the hypothesis of exhaustion of a very few elements of the lowest motor centres, and of the third segment of the kinetic route, and of the muscles in such cases as that of B. for the present, but yet do not abandon it. I shall speak of the hypo-kinetic tract in such cases as that of B., as if it extended no lower than fibres of the lateral column, and as not involving any cells of the lowest motor centres in which they end. It would be reasonable to grant that it was at least of that depth in B's case.

It will be well to draw attention to resemblances and differences between post-epileptiform hemiplegia and the hemiplegia which is the result of destruction of the motor part of the internal capsule; for the moment assuming that the explanation given of the former is correct, and that the statements accepted by most physicians as to the latter are accurate. I will call the former Hemiplegia E, the latter Hemiplegia D.

First, for resemblances, taking to begin with, for comparison and contrast, recent Hemiplegia D. The regional (paralytic) affection is the same as in Hemiplegia E, and in each depends on a negative state of the same strand of fibres, those interconnecting the right middle motor centres and the left lowest motor centres. (I shall speak only of what I called the first set of fibres.) Now for differences. In Hemiplegia E (as in the case of the patient B.) there is exaggeration of the tendon reactions, in Hemiplegia D they are, we shall suppose, normal. Correspondingly for the negative lesions; in Hemiplegia E the negative lesion is of the whole length of the interconnecting fibres mentioned (and spoken of before as the second segment of the hyper-kinetic route become a hypo-kinetic tract) up to, but not involving, the lowest motor centres. In recent Hemiplegia D the negative lesion is quite local, a breaking up of the capsular part of the interconnecting fibres; the rest of the segment, its whole extent below the capsular break, being normal.

Now for *old* cases of Hemiplegia D; in these there are increased tendon reactions; in them a negative lesion secondary to the capsular one has been established; there is then a negative state (destruction) of the fibres (lateral sclerosis) below, and added on to, the local capsular lesion;¹⁰ that is to say, in the case of Hemiplegia E there is established at once the state of things which only occurs late in Hemiplegia D. So long as the exhaustion lasts (so long as the central kinetic route is hypo-kinetic) in Hemiplegia E, the situation is the same as in old cases of Hemiplegia D; it is the same for the time being whether the nervous elements in question are functionally dead, as in the former, or actually dead (broken up and wasted away), as in the latter.

In a popular use of the word cause, these negative conditions may be said to cause the increased tendon reactions, just as, popu-

¹⁰ Sherrington has traced the "descending wasting" in the lateral column of the cord of the monkey as low as the coccygeal nerve roots.

larly speaking, opening floodgates is said to cause water to flow. But using the word cause properly, it is an impossibility that the negative functional state in either Hemiplegia E or D can cause anything; a nothing cannot cause the something, the superpositive condition for exaggeration of the knee-jerks. If Bastian be right in his conclusions as to the effect of absolutely complete transverse lesion of the dorsal or cervical cord (see Lecture I), the hypothesis of a change in the anterior horns, produced by any sort of pathological process, is no longer tenable, as he has pointed out. I shall, however, consider other arguments against that hypothesis, which are supplied by cases of post-epileptiform paralysis.

It is agreed upon that the increased tendon reactions in cases of hemiplegia depend on exalted "excitability" (a superpositive functional state) of nervous elements of some anterior horns. There are different hypotheses as to the process by which that exaltation is produced, and thus as to the exact state of the cells of the anterior horns concerned. Two questions may be asked. Is the abnormal condition of the cells the result of any pathological process involving the horns themselves, or are the cells healthy although in greater activity? I reply to the first question negatively, and to the second affirmatively.

I take, for further consideration, an old case of Hemiplegia D, one in which lateral sclerosis is established. And now I consider rigidity as well as increase of the knee-jerk (limiting illustration to that one tendon reaction), both of which superpositive phenomena it is agreed upon imply exaltation of function of cells of anterior horns. One hypothesis as to the production of exaltation of function of the anterior horns (some of the lowest motor centres) is that the same process which leads to destruction of fibres of the lateral column produces, when come to the horns with which they are in physiological union, increased "excitability" of their cells; the same process, on this hypothesis, produces loss of function (destruction) of fibres and its physiological opposite superfunction of cells. The hypothesis of Charcot deserves to be received with profound respect. Referring to the condition of the cells of the anterior horns, as it corresponds to rigidity in hemiplegia, he considers it the theory which best accounts for facts we daily witness in cerebro-spinal diseases, that there is a purely dynamic lesion of their ganglion cells—a state analogous to that produced by strychnine. If this state of the cells be produced by an extension to them of the same process as that which has destroyed fibres, it (however slight the alteration effected may be, and however insusceptible of microscopical demonstration the change may be) must be considered to be the result of a pathological process.

This hypothesis cannot apply to Hemiplegia E; in this case it is not likely that there are caused by the excessive discharge a negative state of fibres, and a super-positive one of cells. All the more that after some epileptic fits there is found exaltation of the knee-jerk, and after others loss of it; differences, explainable on differences, in the severity of the discharge in the paroxysm, and on the different "depths" of exhaustion after it; there is a negative functional state in both, but lower down in the kinetic route, of cells of anterior horns, when the knee-jerks are absent.

My hypothesis is that both in Hemiplegia E and in old cases of Hemiplegia D, the cells of the anterior horns are perfectly healthy. They are, I submit, untouched by the pathological process causing the lateral sclerosis or by any other, and are simply over-active, partly from loss of control, and partly from non-antagonised cerebellar influx (second degree (*h*) of superpositive functional change). But I do not state the hypothesis of loss of control as I used to do. There is sometimes exaggeration of the knee-jerk in recent cases of hemiplegia, and this may be owing to sudden loss of cerebral (middle motor centres) control. I shall, however, speak as if the current opinions were correct—that both the increased tendon-reactions and rigidity "wait for" the descending degeneration in cases of Hemiplegia D. I now adopt an hypothesis put forward by Gowers, which is as to the effect that close upon the anterior horns there are small inhibitory centres, the anterior horns being, in his nomenclature, "muscle-centres."

In cases of recent Hemiplegia D, before the degeneration in the lateral column, these local inhibitory centres are intact, and the knee-jerks are not exaggerated. But I suggest that in such cases of Hemiplegia E as that of the patient B., they are exhausted as well as are fibres of the lateral column; after those epileptic fits, when the knee-jerks are lost, both the inhibitory centres and the muscle centres are exhausted. Now taking old cases of Hemiplegia D, and dealing with rigidity as well as with the exaggerated knee jerk, I would say that the "descending" process,

when it reaches the anterior horns, destroys the small local inhibitory centres; the anterior horn proper, "the muscle centre" now bereft of its inhibitory centre, is uncontrolled. The cells of the "muscle centres" are not invaded by any pathological process; they are healthy, but yet over active from loss of control. This hypothesis as to old cases of Hemiplegia D invokes but one mode of action—destruction. That hypothesis brings the facts of Hemiplegia E into harmony with those of long-standing cases of Hemiplegia D; it accounts for the absence of increased tendon reactions in recent cases of Hemiplegia D. Certainly cases of Hemiplegia E have to be considered as well as cases of recent and old Hemiplegia D in all hypotheses as to the nature of increase of the knee-jerk in cases of brain disease.

Of any degree of rigidity of muscular regions paralysed—during the paralytic stage or part of it I mean of course—after epileptiform seizures I know nothing. Possibly I have overlooked it. But Franck and Pitres have made very important observations on a muscular rigidity after epileptiform seizures produced in dogs and cats. Of course it might be said that this active muscular condition is owing to a slighter degree of the same cortical discharge which produced the obvious fit—that it is not post-paroxysmal, but a great attenuation of the paroxysm itself. But these distinguished observers tell us that the rigidity is altogether different from the convulsion in the attack itself. And what is decisive is that ablation of the whole motor zone does not cause disappearance of the rigidity.¹¹ It seems to me that the hypothesis I have taken from Gowers, exhaustion of inhibitory centres, and consequent over-activity of the muscle-centres of the anterior horns to which they belong, best explains these phenomena.

Although beyond mentioning it I have not in this Lecture spoken of cerebellar influx, I believe it to be a factor in the causation of the exaggerated tendon reactions in Hemiplegia E, and of these and the rigidity in old cases of Hemiplegia D—that in both there is loss of control and unantagonised cerebellar influx, and thus that the two positive symptoms are owing to the unhindered activity of perfectly healthy structures.

In some cases of epileptiform seizures at, or close upon their onset, there is loss or, at least, defect of speech with very little local spasm. (We have, of course, no concern in this inquiry with any cases of *le petit mal*.) I shall, however, speak only of temporary abnormal affections of speech, not in, but after the paroxysms—of what is present when all convulsion is over.¹² I explain post-epileptiform defects of speech in the same way as I did post-epileptiform paralysis, supposing there to be in the former exhaustion of nervous arrangements of "Broca's region."

Just as there are cases of epileptiform seizures with permanent paralysis of the side of the body in some part of which the convulsion starts and in which there is more paralysis, temporarily, of that side after a paroxysm, so in some cases of epileptiform seizures there may be permanent defect of speech and a temporary increase of that defect after a paroxysm. I shall omit consideration of these important cases. So far as I have seen, abnormal affections of speech are found after fits with right-side "signal symptoms." They certainly occur after left-sided fits, as Dr. Herman Weber has pointed out.¹³

I have only once known abnormal affection of speech to occur after a fit beginning in the foot (right); that case is of no particular value in localisation, as at the necropsy two large blood-cysts were found, causing extensive compression of each cerebral hemisphere. Temporary defect of speech after epileptiform seizures beginning in the (1) right hand or (2) right side of face or tongue, or both, is not rare.

The "abnormal talking" after some epileptiform seizures has long seemed to me¹⁴ not to be what could commonly be called aphasia; it often sounds like a *mélange* of an articulatory defect and of a speech defect. It may be that in these cases there is not only exhaustion of some elements of Broca's region, but also of elements of the corresponding lowest (bulbar) motor centres; if so, the situation is analogous to cases where, after epileptic fits, the knee-jerks are absent, from, presumably, exhaustion of (lumbar) lowest motor centres. I think the "abnormal talking" referred to most

¹¹ *Les Fonctions Motrices du Cerveau*, by Francois Franck pp. 88, 89.

¹² "There is a peculiar class of cases of epileptic hemiplegia in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech." (Todd, *Nervous Diseases*, lecture xv.)

¹³ *Trans. International Med. Congress*, 1881, vol. ii, p. 19.

¹⁴ "Study of Convulsions," *St. And. Med. Grad. Assoc.*, vol. iii, 1870; Reynolds's *System of Medicine*, vol. ii, second edition, p. 257; see also Gowers' *Epilepsy*, p. 101.

likely to occur after epileptiform fits beginning in the cheek or tongue. What may be distinguished as "pure aphasia" is itself, physically regarded, a paralytic affection in the sense of loss of complex ("articulatory") movements of the tongue, palate, lips, etc.; so the *mélange* spoken of, if the hypothesis suggested be valid, is physically a mixture of loss of some complex (middle level) and some simplest (lowest level) movements of those parts. The subject is not a simple one, because along with right hemiplegia from destructive lesions we sometimes meet with a difficulty of articulation (I used to call it "ataxy of articulation") when there is no obvious disability in the tongue, palate, lips, etc.¹⁵ There is one thing which must be mentioned in this connection. We should not consider that the inability of a patient who is more or less aphasic after an epileptiform seizure to put out his tongue when told is evidence of implication of lowest motor (bulbar) centres; for in these cases the tongue moves well in other and simpler operations. Moreover, this inability is often found in cases of aphasia from destructive cerebral lesions, and lasts too long in those cases for the easy explanation of "shock;" in them implication of bulbar centres is out of the question. This curious symptom is loss of a most special movement from a cortical or sub-cortical lesion; in that sense it is paralysis. It is no way regarded a speech or articulatory defect. To put out the tongue when told is what is called a "voluntary" movement, just as lifting the arm when told, or the card-sharper's shrug (Lecture II) is.

I shall ignore the hypothesis mentioned, and deal only with cases which would be admitted by all to be defects of speech proper, that is to be "of an aphasic character"—cases in which much speech of an imperfect kind remains, the utterances being clearly articulated. In most of these the prior convulsion starts in the right hand, and, I have thought, most often, not in the thumb or index finger, but in the hand generally or in the ulnar fingers. Of course there are many degrees of abnormal affection of speech of this kind, and it varies according to the time elapsed since the cessation of the fit.

I have not observed post-epileptiform loss of speech, but only defect of speech ("partial aphasia"). I shall use the term Defect of Speech E, or Partial Aphasia E. The term "defect of speech" is equivocal, as is also the term "partial aphasia;" it really covers two opposite elements, negatively loss of some speech, and positively retention of the rest of speech.¹⁶ The patient gets words out clearly, and may even get out simple appropriate replies, such as "Very well," and may answer correctly by "Yes" or "No," or both. This is the positive element: it is the inferior speech to which the patient is reduced. But he is *not* able to reply correctly except by "Yes" and "No," and by other simple and very general expressions; he is *not* able to converse properly on simple subjects; he is *unable* to explain anything at all complex. These statements give the negative element: the speech lost. It is of extreme importance to distinguish the two opposite elements of the symptomatic condition. It is plainly impossible that the post-epileptiform exhaustion (*vide supra*), which I suppose there is of part of Broca's region, can answer to the patient's utterances—to the positive element: that negative functional state of nervous elements of the region answers to the patient's loss of some speech. His utterances, positive element, however inferior as speech, answer to activities of nervous arrangement of Broca's region which are healthy—which are in all ways normal except possibly for slight "loss of control." Here is another illustration of the statement that part of the symptomatology of nervous maladies is the outcome of activity of healthy nervous arrangements.¹⁷

¹⁵ Dr. Charles K. Mills, of Philadelphia, relates, *University Medical Medicine*, November, 1889, a very important case of "Softening of the Face-area with Oro-lingual Monoplegia."

¹⁶ My present concern with aphasia is only a very limited one. I need not always supply the obvious qualifications to statements made in the text. The expressions "loss of some speech" and "retention of the rest of speech" must not be taken literally as if they meant that the patient had lost certain words or propositions of the (his) English language altogether, and had retained the rest of them intact. Such is an impossible condition of things when an aphasic patient corrects his mistakes. But the statements may be taken literally for the mere purpose of a limited illustration. I do not suppose that there are fixed nervous arrangements—some for these words or syllables (properly movements corresponding to syllables) only and some for those only. I would rather than hold this mechanical doctrine go to the other extreme, and say that there are no nervous arrangements for movements in any centres except at the time when these and those motor nervous elements are functioning together in a particular temporary grouping.

¹⁷ It is one thing to locate the negative lesion which destroys speech (renders a person unable to speak aloud), and quite another thing to say that "speech resides" in any particular part of the cortex. Words, or some other symbols,

We must bear well in mind that speech is a psychical process. Nowhere is it more important to distinguish the psychical from the physical. I say once more that psychical symptoms are to medical men only signs of what is wrong in a material system. Our task as physicians is to ascertain the nature of the physical process correlative with speech, or more exactly the nature of the anatomical substrata of words (syllables). No one denies that the physical basis is of cells and fibres, but this is a morphological account of it. The anatomical basis is, I submit, of morphological elements so grouped as to form certain sensori-motor nervous arrangements. These, I presume, are audito-articulatory—that is, they are nervous arrangements representing certain auditory impressions along with certain corresponding complex (articulatory) movements of the tongue, palate, lips, etc. We suppose the motor elements of these *sensori-motor* arrangements to be of Broca's region. In the case of Defect of Speech E our ultimate concern is with the two opposite functional states of these motor elements which answer respectively to the two opposite psychical elements of the double symptomatic condition, which is unfortunately named (defect of speech or partial aphasia) after the negative element only. The motor elements alone of the sensori-motor substrata of speech are damaged. I submit that in Defect of Speech E there is *correlative with its negative psychical element* exhaustion of some nervous arrangements of Broca's region, which is productive of loss of some of the complex movements of the muscles of the tongue, etc. ("articulatory muscles"). And I submit that the speech remaining possible is correlative with integrity of other nervous arrangements of that same region for other complex movements of the same muscles; these healthy nervous arrangements function during the inferior speech remaining possible to the patient, and produce the corresponding articulatory movements. I have now to support the conclusion that the inferred exhaustion of some elements of Broca's region in Defect of Speech E produces paralysis in the sense of loss of complex movements of the tongue, etc.

There is often with Defect of Speech E some temporary decided paralysis of (loss of many movements of) the right arm. This statement, however, although allowable clinically, is not a scientific one. We should not in a realistic inquiry group together a psychical symptom and a physical one as if when so considered they were symptoms of the same order. In harmony with what I have said, the things really comparable are respectively loss of some complex movements of the muscles of the tongue, etc. (corresponding to the negative element in the defect of speech), and loss of some complex movements of the muscles of the arm (in both of movements represented by the middle motor centres). In this sense the two co-existing things are paralyses. On this basis alone is their concurrence intelligible and their comparison and contrast possible. With regard to the defect of speech, it is to be vividly kept in mind that the fellow part of the right middle motor centres (centres of the left half of the brain) for other complex movements of the muscles of the tongue, etc., and that the right and left lowest (bulbar) motor centres for simplest movements of those muscles are intact.

Those who do not follow me in distinguishing the psychical from the physical will, perhaps, think that I am confounding difficulty of articulation, a physical symptom, with a mental symptom (with "aphasia"). The fact is that I am making an absolute distinction between speech, which is a psychical process, and its correlative anatomico-physiological process, which is purely physical. It is only the *physical bases* of words (or properly of syllables) which I assert to be sensori-motor nervous arrangements representing complex movements of the muscles of the tongue, etc., in association with complex combinations of auditory impressions. I only say that *correlative with the negative psychical element* in defect of speech there is the *physical loss* of some complex movements of the tongue, etc. Still, it may be thought that I have no clear ideas on the difference between aphasia, a mental defect, and, for example, "bulbar paralysis." In reality I have said that the "bulbar centres" (some lowest motor centres) are quite intact in Defect of Speech E. In bulbar paralysis there is loss of the

serve us during thought; when a man is thinking "gold is yellow," words in propositions are as certainly concerned as they are when he says that aloud. I submit that the highest centres ("organ of mind") must be engaged during speech, whether external or internal, notwithstanding that a negative lesion of a part of the middle motor level produces aphasia. Not believing in abrupt localisations, I do not mean by using the term "Broca's region" to limit the negative lesion productive of aphasia to that part of the lowest frontal convolution which enters into the "motor region." As is the custom, I shall neglect the concern which the highest centres must have with speech.

simplest movements of the tongue, etc., and the negative lesion is bilateral (of certain motor centres of both halves of the lowest level), whereas in all cases of aphasia there is loss of complex movements of the tongue, etc., and the negative lesion is of motor centres of but one (left) half of the middle level, and the lowest motor (bulbar) centres are intact. Is not this difference enough?

The objection may be made that, whilst in the case of post-epileptiform paralysis with partial aphasia there is obvious paralysis of the arm, there is, as a matter of fact, none of the tongue, palate, lips, etc. It will be well in further exposition to stop speaking of Defect of Speech E, and to take in illustration a simpler (although much more serious) case, one of *loss of speech* ("complete aphasia") from a destructive lesion, say from softening of Broca's region. I shall suppose, however, that there is retention of "yes" and "no."¹⁹ Here the negative element is loss of nearly all speech; the positive element being only retention of "yes" and "no." I shall call this case Loss of Speech D, or Complete Aphasia D; but the word "loss" is to be taken as qualified by "except for retention of 'yes' and 'no.'" We shall suppose there to be, as there nearly always is, right hemiplegia.

To say that the patient Aphasia D is speechless because he has lost the memory of words is to give no explanation of his inability to speak, any more than it is an explanation of the paralysis of his right arm and leg to say that it is owing to loss of volition. All psychological explanations of physical disabilities are merely verbal. Admitting for the sake of argument only that loss of memory of words is a correct *description*, our concern as physicians is with the physical condition correlative with that psychological loss. A part of the patient's brain has disappeared, probably the hinder part of the lowermost (left) frontal convolution (and no doubt more brain adjacent) has been changed to defluent stuff which is brain no longer; we shall speak of this as "destruction of Broca's region." I repeat that the lesion causes paralysis. I admit that there is no disability in the muscles of the tongue, palate, lips, etc., in their commonplace services, as in eating, drinking, swallowing, etc.; I admit it readily, because I only mean that there is loss of the complex articulatory movements of those muscles. There is nothing discoverably wrong with these muscles except for the slight facial and lingual paralysis which is a part of hemiplegia, right or left. The case illustrates that we may have loss of some *movements* of certain *muscles* without discoverable disability of those muscles. The seeming paradox in the particular case under remark is easily explained by Broadbent's well-known hypothesis.

In our speechless patient, not only are the lowest motor centres of *both* halves of the bulbar part of the lowest level for simplest movements of the muscles of the tongue, etc., intact, but also, what is most important, complex movements of these muscles of both sides remain represented in the right half of the brain (middle motor centres). Hence, on account of those remaining representations, there is no obvious disability of the muscles of the tongue, etc., in their commonplace services. Taking the simple representation by the lowest level for granted, the assumption is that the muscles of both sides of the tongue, palate, lips, etc., are represented by complex movements in each half of the brain, though no doubt more especially in Broca's region than in the fellow-region of the right. Here is an exemplification of Broadbent's hypothesis. Horsley and Schäfer write: "The *face area*, although we have so called it for convenience sake, actually gives rise to movements not only of the facial muscles, but also of the whole of the upper part of the alimentary tube (mouth, throat, and larynx)..... It is physiologically remarkable from the fact that many of the movements which result from its excitation are apt to be executed bilaterally, which is only exceptionally the case with excitation of the other areas (except that for the head and eyes)."¹⁹ Hence destruction of neither Broca's region nor its fellow-part of the right middle motor centres produces disability in the commonplace services of the muscles of the tongue, etc., although beyond all question some movements of them are lost. But destruction of part of both

halves does,²⁰ as some cases of double hemiplegia show, notably one recorded by Dr. Thomas Barlow.²¹

So that, so far from "confounding cases of aphasia with cases of bulbar paralysis," I am making a very definite distinction. So far am I from comparing loss of speech, considered as a negative psychical symptom, with the physical symptoms of bulbar paralysis, that I urge that there is not even a basis for any reasonable contrast of the two; psychical symptoms and physical symptoms are "not on the same platform." Regarding the physical condition correlative with the aphasia and that of the case of bulbar paralysis, the cases are alike in that there is in each loss of movements of the same muscles; on this basis I urge their resemblance as cases of paralysis. At the very same time I urge the differences between loss of complex movements of the tongue, etc., represented by certain *middle* centres of but *one* half (left) of the brain in cases of Loss of Speech D, and loss of simplest movements of those parts represented by certain *lowest* (bulbar) centres of *both* halves of the lowest level in cases of bulbar paralysis.

Furthermore, the tongue, palate, and lips not only act well in eating, drinking, swallowing, etc., in Loss of Speech D, but also when the patient says "Yes" and "No." He may have a stock utterance, as "Awful," "Oh, my God!" etc. He may when excited swear or get out other elaborate ejaculations; none of these are speech proper—they are not propositions, but compound interjections; the patient cannot repeat ("say") what he ejaculates. The articulation of all these utterances is perfect, and I presume that no one would deny that the articulatory movements are effected by perfectly healthy nervous arrangements. They are effected, I submit, by those of the undamaged right half of the brain, of course with subagency of lowest (bulbar) motor centres of both halves. Still neglecting, as everybody else does, the concern of the highest centres with speech and with morbid affections of it in disease, I submit that as regards speech the right half of the brain is the automatic half, and that the left is that half in which automatic action ceases into what we call voluntary action.

The positive condition of our patient, Loss of Speech D, is that he retains the propositional use of "yes" and "no," physically two complex articulatory movements of the tongue, etc.

Now I draw attention to the fact that the patient who never speaks, never does make (with the exceptions stated) the complex movements of articulation, which I say are lost. Instead of explaining that he does not do so *because* he has lost "the memory of words," I submit that he never makes them *because* the nervous arrangements for those movements have disappeared—that he has not got those complex movements. Once more I urge that what is psychically aphasia is physically paralysis.

Moreover, the part destroyed in the case of aphasia is motor; no one denies since the researches of Hitzig and Ferrier that the homologous part in lower animals is motor. Hence, there is nothing for destruction of it to produce in these animals except paralysis. Mark that although destruction of that part of the "motor region" on the left in man produces no disability of the muscles of the tongue, etc., of either side (beyond the slight right facial and lingual paralysis spoken of as part of hemiplegia), discharge of that part develops movements of these muscles of *both* sides; by subagency, of course, of certain lowest motor centres of both halves of the lowest level, develops a contention of complex and of simplest movements of those muscles. From discharge of the part on the left are revealed the movements, the loss of which, upon destruction of that part, is masked by compensation by the fellow part of the right half of the brain. Yet there may be discharge (impulses passing by *route* of the callosal fibres) of the fellow part on the right consequent on the primary discharge of the left. I mention again that Sherrington has traced degenerated fibres from a unilateral lesion of the lower end of the fissure of Rolando into both halves of the pons and medulla.

Hence I conclude that the patient hemiplegic and speechless (aphasic) has not only lost very many complex movements of the muscles of the right arm and leg, but that he has also lost many complex movements of muscles of both sides of the tongue, lips, etc.; the conclusion is that his whole physical condition is, and is nothing else than, paralysis *in the sense of loss of movements*.

The condition is fundamentally the same in the Defect of Speech E, but the negative element (loss of some speech) is less, and the positive element (retention of much speech) is greater. Here,

¹⁹ To speak is not merely to utter words, but to *propose*. The patient spoken of in the text, although not absolutely speechless, is so, except that he has only the use of the two most general of all propositions; I say "the use of" supposing that in the case taken for illustration the patient can assent and dissent by them. If he only uttered them at random, or if they were only signs of emotion, they would not serve propositionally, they would then be of no speech value.

¹⁹ *Phil. Trans.*, 1888.

²⁰ Such cases are sometimes called cases of cortical bulbar paralysis; they are double cortical; the bulbar centres are intact.

²¹ *JOURNAL*, 1877.

again, it will be well to illustrate by the case of a destructive lesion (a much smaller one than in Loss of Speech D), by softening, destroying but little of Broca's region; I will call this case Defect of Speech D. There is not rarely from such a pathological process a condition essentially like that in some cases of Defect of Speech E. Let us suppose that in the case of Defect of Speech D the patient frequently makes mistakes when he talks, either by using wrong words or by being reduced to very "automatic" utterances, such as "Very well," or that he often uses roundabout expressions such as "It won't come out here" (which, eked out by appropriate pantomime, meant "I can't make water"). And yet, to simplify exposition, I will take the patient's saying "hat" for "carpet" only, that to be a sample of the whole of his condition. Here we see the necessity of distinguishing the two elements, negative and positive. It is certain that the softened brain (really what is brain no longer; there is a hole filled with detritus of nervous elements) cannot be the cause of the patient's saying "hat;" that is the speech left, and is owing to activity of the part of Broca's region which remains perfectly healthy.²² This, his positive condition in speech, is sampled by his saying "hat." His negative condition is alone owing to "disease," and is sampled by his inability to say "carpet."

The negative element in Defect of Speech D is a minor degree of that in Loss of Speech D; the positive element is a major degree of the positive element ("Yes" and "No" only) in Loss of Speech D; in the former the dissolution of Broca's region is shallow and the level of evolution high, whilst in the latter the dissolution is very deep and the level of evolution very low, if there be any in that region. The two cases differ in double degree. In the Defect of Speech D there is much speech left, and there are very many mistakes; in the Loss of Speech D there is very little speech left, and there are no mistakes, the patient correctly assenting to and dissenting from (by "Yes" and "No") all statements made to him.

Now for the physical condition in Defect of Speech D. Answering to the slight negative element, there is loss of but a few complex articulatory movements represented in Broca's region; answering to the positive element there is retention of many other such movements; or we may say, limiting illustration to the samples, there is loss of the complex movements answering to inability to say "carpet," and retention of the complex movement answering to saying "hat." If anyone says that a word (properly a syllable) is not a movement he is agreeing with me; for I am contending that activity of nervous arrangements representing certain complex movements (with, of course, corresponding impressions) of the tongue, etc., is only the anatomico-physiological process going on during the appearance of what are the psychological things—words. I need only say that the softening in Defect of Speech D is the negative lesion equivalent to the exhaustion in Defect of Speech E. The utterances both in Defect of Speech E and D are the outcome of activities of perfectly healthy nervous arrangements. The negative lesion in both is dissolution; in each the speech, however imperfect, as certainly signifies evolution going on on a lower level, as the speech of healthy persons does evolution going on on the normal level.

I have admitted that the patient in Defect of Speech E and D may correct his mistakes—that after saying the wrong word "hat" he may immediately say the right word "carpet,"—and I will here admit that when he says "hat" he knows that the word is not the right one. But this does not invalidate the principle of the explanation. For convenience of exposition I made the arbitrary assumption that there are fixed and unalterable nervous arrangements for particular movements, in this case for complex movements of the tongue, etc., corresponding to syllables. I think the seeming difficulties could be met if, abandoning that assumption, I were to go into detail as to the constitution of nervous centres. At any rate, the attempt is to give a materialistic explanation of the physical condition in aphasia, without confounding the concomitant psychical states with that physical condition. I submit that the explanation is, at any rate, as good as the psychological explanation. If we do invoke the latter so-called explanation, we have not only to account for, to take the samples again, "loss of memory" for "carpet," but also for the "over-memory" for "hat." When the patient cannot say "carpet," why does he utter anything? Why does "hat" come out instead?

And admitting that the "loss of memory" for "carpet" is only temporary, and that next moment the patient can say "hat," we have to account for the temporary defect of "memory" and for its sudden restoration for that case. Moreover, if the patient says "hat" and knows that it is wrong, he can only know it by there being revived in him the word "carpet" at the very same time; certainly these two words must be revived mentally; there is then "verbal diplopia." If it is admitted that the psychological statements as to the aphasic conditions are correct descriptions, those who make them are just as much bound as anyone else to seek the abnormal material conditions of the several phenomena of "amnesic aphasia." And bearing in mind the proof given by Ferrier, Horsley, Schäfer, Beevor, Sherrington, and many others, that the region damaged is motor, a merely morphological account of the physical condition in aphasia will not suffice. If anyone says that he cannot understand how activities of motor nervous arrangements can correspond to words, I would remind him that, except the popular psychologist, no one pretends to understand how any material conditions correspond to any psychical states.

THE GOULSTONIAN LECTURES ON SOME CEREBRAL LESIONS.

Delivered before the Royal College of Physicians of London.

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LECTURE III.—ON CEREBRAL EMBOLISM AND ANEURYSM.

I PROPOSE considering to-day the cases of cerebral embolism and of cerebral aneurysm, which are for the most part included among them, that have died at Guy's Hospital, between the years 1869 and 1888, a period of twenty years.

In 79, that is, in 0.9 per cent., or nearly 1 per cent., of the inspections, cerebral embolism had occurred. In some of the patients two or more cerebral vessels contained clots; there were ninety-five blocked vessels found in the whole series of cases.

Forty-four of the patients were males and 35 females; there was nothing in the histories of the cases to indicate why men were more often affected than the women.

The youngest patient was aged 13, and the oldest 73; the remainder of the cases occurred between the ages of 13 and 66, the distribution being fairly uniform for the decennial periods from 10 to 60. Patients at both extremes of life seem to possess an immunity from this affection.

Cerebral Embolism.

No. of cases	10-19	20-29	30-39	40-49	50-59	60-69	70-79
...	18	15	15	10	15	6	1

In only 6 cases was the source of the emboli not discovered. In 4 of them the vessel had been blocked for a long time (two years, two years, ten, and many months), and it is quite probable that in these, cardiac vegetations or thrombi may have given rise to emboli, and may yet have disappeared and left no trace at the *post-mortem* examination.

In most of the cases there was no difficulty in tracing the source of the emboli; in 68 it was found to be in the heart. The mitral valve was the most common seat, being affected in 46 cases, in 7 of which the aortic valve was also diseased. The vegetations were limited to the aortic orifice in 13; in most (46) of these 68 cases, there was chronic disease of the valves, on which had been grafted a fungating endocarditis, the vegetations were generally friable, and in many ulceration of the endocardium and valves was also present. In 18 of the cases, ulceration had led to rupture of the chordæ tendineæ. The mitral orifice was markedly stenosed in 26, and was generally associated with recent friable (vi), or fungating granulations; in 5 the valve was free from vegetations, and in 4 of these the embolus had come from *ante-mortem* clotting in the left auricle, while in 1 no clot or granulating surface was found (Case CLVIII). The embolus ori-

²² To give another example, surely it is impossible that when the patient referred to in the text said, "It won't come out here," there was engagement of nervous arrangements which were "diseased" in any way whatever; the "disease" was the cause of his inability to say "I can't make water."