LECTURES

ON

THE DIAGNOSIS OF EPILEPSY.

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LECTURE I.

MR. PRESIDENT, GENTLEMEN,—The first thing I have to do is to thank you most heartily for the great honour you have conferred on me in asking me to give these lectures, and the next is to assure you that the task is in all ways a most pleasant one.

The subject I have chosen is the Clinical Investigation of Epilepsy. Epilepsy will be considered chiefly with regard to diagnosis. Such a subject is, however, far too wide. I must, therefore, omit large parts of it. To show more definitely how I shall handle my subject, some preliminary remarks on classification generally may be made.

There are two kinds of classification of diseases: one scientific, generally called theoretical, for the advancement of knowledge; one empirical or clinical, for practice. In the scientific or theoretical, so far as is possible, we classify diseases as they are *departures from healthy states*—as they show abnormalities of structure, function, and nutrition. Empirically or practically, we classify them as *they approach certain types*. We will first consider the scientific classification of epilepsy, or rather of epilepsies.

Scientifically, I should consider epilepsies on the hypothesis that the paroxysm of each is dependent on a sudden temporary excessive discharge of some highly unstable region of the cerebral cortex. There is, in other words, in each epilepsy, a “discharging lesion” of some region of the cortex cerebri. The discharging lesion is nothing more than a group of cells whose instability is raised far above normal. This is considering an epileptic fit as being a development of the impressions and movements represented in some unstable region and in other regions secondarily discharged; but yet, since the discharges are excessive, an excessive and, so to speak, brutal development of them. Thus epilepsies are, scientifically regarded, departures from normal states by excess. As implied, the “discharging lesion” is supposed to be local. The discharge of the highly unstable cells constituting it (the primary discharge) leads to secondary discharge of healthy cells in other centres (collateral or lower) with which there is anatomical connexion by fibres; the degree and width of the secondary discharges varying according both as the force of the currents developed by the primary discharge and the resistance opposed by fibres and by cells of the healthy centres vary. We must not forget the resistance offered to local discharges in our consideration of their effects. The “discharging lesion” may be likened to a fulminate which overcomes the resistance of less unstable compounds.*

It is in the study of epilepsies as departures from normal states that the researches of Hitzig and Ferrier have enormous value. Whatever interpretations psychologists and physiologists may give of them, the *medical* value of these researches is incontestable. By galvanic or faradic excitations of the cortex cerebri in dogs, convulsive seizures can be produced which are as closely like epileptiform seizures in man from disease in the same cerebral region as we could expect, considering the difference of the natural movements of the two animals.

I would take this opportunity of very strongly urging the importance of Ferrier's investigations of the sensory region of the brain. His conclusions harmonise so well with clinical observations, that I think they are of inestimable value in giving method to our investigations. It is quite certain that there are paroxysms consisting chiefly of sensory symptoms, which pair off with, and are strictly analogous to, unilaterally beginning convulsions. At any rate, Ferrier has, I think, given a new starting-point for the methodical, scientific investigation of a most important class of paroxysmal affections which are, in a scientific regard, epilepsies. There are also cases of epilepsy in which, although the paroxysms are for the most part motor (ordinary epileptic convulsions), the “warning” of the fit is by some crude sensation, *e.g.*, by “noise in the ear”, or by some “taste” (this is very rare indeed),

or by “coloured vision”, or by a “strong smell in the nose”. In each patient, the “warning” is always the same crude sensation; and very often the patient has, besides severe fits heralded by his crude sensation, slight paroxysms consisting of little more than that “warning”.

Since the cerebral cortex is part of the chief organ of mind, the tacit assumption in the above statements is that the whole of the chief organ of mind (the cerebrum) is, like all other nervous centres, made up of nervous arrangements representing impressions and movements. Another way of stating this is to say that not only have the so-called motor and sensory regions this kind of constitution, but the rest of the brain also has it. It is not said that mind, but that *the organ of mind*, is so constituted.* This is a necessary outcome of the doctrine of Evolution.

There are numerous epilepsies under the definition that any epilepsy is, on its anatomical side, a “discharging lesion” of some region of the cortex cerebri. The kind of paroxysm differs according to the particular region of the cortex affected; and since many regions, if not any region, of it may be affected, the number of different epilepsies, scientifically regarded, is great. There are some, as migraine, of which the phenomena are chiefly sensory; others of which the phenomena are chiefly motor. In some, loss of consciousness is first of all, or early, in the paroxysm; in others, it occurs late in it, or not at all. Besides, there are degrees of paroxysms of each kind, depending on the degree of the discharge of this or that part of the cortex, and on how much resistance it can overcome.

Before leaving this part of my subject dealing with classification—only another name for scientific investigation—it must be remarked that the scientific investigation of epilepsy owes very much to Broadbent. For my part, I think his hypothesis as to the mode of representation of certain movements of the two sides of the body in each half of the brain, is essential to the full interpretation of all kinds of severe epileptic seizures.

The above described scientific or theoretical classification, whilst of value in extending and simplifying our knowledge, is worthless for practical purposes. It would, I think, include migraine. To arrange migraine along with ordinary epileptic seizures—with cases of loss of consciousness and convulsion—for practical purposes, would be as absurd as a scientific zoological classification would be for fishermen, hunters, and graziers. Whilst scientifically migraine† is, I think, to be classed with epilepsies, provisionally at least, as being dependent on a “discharging lesion” of some part of the cortex cerebri—probably of some part in Ferrier's sensory region—it would be as absurd to classify it along with ordinary cases of epilepsy, for practical purposes, as to classify whales with other mammals for purposes of practical life. A whale is in law a fish; in zoology, it is a mammal.‡

We have first to find out that our patient's seizures are epileptic: by no means an easy thing when they are very slight and transitory. We have to consider individual cases as each is presented to us. A patient does not come complaining of a “discharging lesion” of some part of his cerebral cortex; but tells us that he has “had a bad fit”, or that he “occasionally loses himself for a moment”, and so forth. For practical purposes, we require quite a different classification from the scientific one, or rather we must have a mere arrangement; we must have types, not definitions, and consider cases presented as they approach this or that type.

In saying the foregoing, I am only repeating what Handfield Jones said long ago about the classification of epilepsy, and am using similar expressions. As Moxon insists (*Guy's Hospital Reports*, vol. xv), we must have a clinical nomenclature. The clinical or empirical types are only the most frequently occurring cases; there is really no such thing as “genuine” or “real” epilepsy, or “epilepsy proper”, except in this arbitrary sense.

I trust I shall not be supposed to underrate the scientific study of disease in advocating the separate clinical study of it for practical purposes. It is not likely that I shall fall under this imputation, since I am following in the path of one§ of the most scientifically minded men

* It is with considerable satisfaction that I read the following in a recent paper “It is impossible to conceive of what other materials the ‘organ of mind’ can be composed than of processes representing both movements and impressions.”—W. J. Dodds, *Journal of Anatomy and Physiology*, vol. xii, p. 462. The article from which the quotation is taken is a valuable contribution to the methodical study of the organisation of the cerebral centres.

† Dr. Latham thinks that the paroxysm of migraine is owing to arterial contraction in the region of the posterior cerebral artery; Dr. Livinge, that there is in the paroxysm a “nerve-storm” traversing the optic thalamus and other centres.

‡ Griesinger classifies migraine and some other periodical seizures under the term epileptoid.

§ Moxon states (Introductory Address, Guy's Hospital, session 1868-9): “You must know diseases, not as the zoologist knows his species and his genera and his orders, by descriptions of comparative characters, but as the hunter knows his tigers and panthers and wild boars.”...“Familiarity is the word, not mere knowledge.” In *Guy's Hospital Reports*, vol. xv, Moxon writes: “When we are dealing with the public or social function of names, we must keep in view the distinction between a

* The importance of considering resistances in our estimation of the consequences of nervous discharges in diseases of man and in the abnormal physiological conditions induced in animals by some drugs was, I believe, first stated in a methodical way by Sydney Ringer.

in our profession. A man who really dislikes scientific study, or, as he may say, hypothesis or theory, not merely professes a dislike in order to bring his practical ability into relief, is an one-sided man quite as much as those who do not, or rather cannot, care for music or poetry or art.

The following is the first division in our empirical or clinical arrangement of cases of epilepsy. There are two types:

1. *Epilepsy Proper:*

2. *Epileptiform Seizures.*

Partly in order to show that I do not advocate the absolute separation of clinical from scientific investigation, but only the temporary separation, I will consider several broad differences and resemblances betwixt the two divisions of cases from a scientific as well as an empirical standpoint.

a. *As to Affection of Consciousness.*—The distinction is not that consciousness is lost in one and not lost in the other, but that consciousness is lost at first, or very soon, in the paroxysm of epilepsy proper, and late or not at all in the epileptiform seizures.

To say that an epileptic paroxysm begins with loss of consciousness, is equivalent to saying that the epileptic discharge begins in some part of the highest of the cerebral centres. This is an irresistible inference, be those highest centres where they may.*

b. *As to Spasm or Convulsion.*—Convulsion begins more nearly bilaterally in the former; is more nearly universal at once, is more rapid, and is sooner over. In the latter, it begins very locally on one side—hand, side of face, or foot—and becomes universal more gradually, the spasm being comparatively slow, especially at the outset.

c. *As to Seat of Changes.*—The latter depends on disease in some part of Hitzig and Ferrier's region; the former—this is hypothetical—on disease in some part in front of that region, or behind Ferrier's sensory region; that is, on disease in some part of what I suggest are the highest cerebral centres. The so-called motor and sensory regions are only, I think, middle motor and middle sensory cerebral centres; the parts in front of the middle motor centres being the highest motor centres; those behind the middle sensory centres the highest sensory centres.

The vague term "disease" or "changes" has to be analysed; the term "disease" is frequently used without regard to the distinction betwixt the two things, physiological abnormality (*d*) and abnormality of nutrition (pathology) (*e*).

d. *As to Abnormal Physiological Condition of Centres.*—In each, of necessity, there is increased instability of cells, since there occasionally occur excessive liberations of energy; in each, there is a "discharging lesion". There is, then, no essential difference in the abnormal physiological condition in cases in the two divisions; yet the paroxysms of epilepsy proper are more "intense" (more spasm in a short time) than those of epileptiform seizures, and this possibly depends in part on the fact that the highest centres have most small cells (compare *b*).

e. *As to Pathology.*—In each there is increased nutrition; the excessive liberation of energy in the paroxysm of necessity implies increased taking in of nutrient substances having potential energy; increased nutrition is the other side of high instability. We have as yet little more than this very general, but inevitable, inference as to pathology in either. How the increased nutrition is brought about we do not know in most cases; in some cases (see *f*), it is determined by tumour.†

f. *As to Morbid Anatomy,* we can say that the former is rarely caused by gross organic disease; the latter are often so caused, syphilitic gross disease in particular. Sometimes, however, in cerebral tumour there are attacks difficult to distinguish from the epileptic vertigo of epilepsy proper.

Of course, the gross organic disease only "causes" the seizures in the sense that it leads to high instability of cells in its neighbourhood; it acts like a foreign body. The probability is that it leads to the hyperphysiological condition of instability (see *d*) by producing a subinflammatory change, and thus increased nutrition (see *e*).‡ This difference

science nomenclature and an art nomenclature. The aim of a science is the increasing and unlimited knowledge of the things it concerns itself with; a science seeks out and names with no other object than to know. The scope of any art is, on the contrary, limited and prescribed; it has its proper sphere of work, and it must borrow from the nomenclature of the sciences which it rests upon such names only as are useful for its practical purposes."

* By highest centres we do not mean locally highest; a better term would be "latest centres"; anatomically speaking, "latest developed centres" (individually and in race of organisms); physiologically speaking, centres which are the seat of the activities during which our latest mentation, or present highest consciousness, arises.

† I have suggested that increased afflux of blood to the cells unstable may result in some of those cases where there is no local gross organic disease from embolism or thrombosis. I do not mean that there is a new blocking in each fit, but that the blocking determines increased afflux of blood to a certain cortical region, and that, in consequence of increased nutrition, the cells therein have a highly raised instability. In other words, the hypothesis is that in some cases the "cortical regions" unstable in epileptics are "arterial cortical regions."

‡ MM. Pitres and Franck have made an important communication of facts bearing on the production of changes of instability, as the following quotation from *Le Progrès*

as to morbid anatomy in the two seizures can only mean, of course, either that gross organic disease most often occurs in some particular places, or that in some particular places it more easily leads to instability of nerve-cells.

Several of the foregoing are to a great extent but detached statements from different standpoints of what Bright and Wilks long ago asserted; viz., that epileptic or epileptiform attacks without loss of consciousness point to gross organic disease, such as tumour of the brain.

As implied, however (*a* and *c*), the essential difference betwixt the two kinds of seizures is chiefly as to the degree of evolution of the centre, part of which has become unstable. This leads me on to say that several of the above statements (*a*, *c*, *d*) as to differences in the two seizures are implied in the following highly interesting generalisation by Herpin: "En résumé: plus le début est long, moins la crise est violente; plus il est instantané, plus l'accès est intense."

I shall pass over the second division of seizures, and consider only epilepsy proper—what most nosologists call genuine, or idiopathic, epilepsy. I exclude the epileptiform seizures, partly to shorten my subject, but chiefly because I could do little more than repeat what I have elsewhere said about them. I learn from Charcot's writings that they were fully described by Bravais in 1824. Their study has been placed on a scientific basis by the researches of Hitzig and Ferrier. These researches show that epileptiform seizures are developments, although excessive and brutal developments, of the movements normally represented in some particular parts of the cerebral cortex; and thus the notion that epilepsy in a sort of entity attacking the patient, is finally got rid of. An epileptic is a person, certain of whose own cortical nerve-cells have become highly unstable; and in their discharge, the movements or impressions they represent are excessively developed.

More recently, eminent French physicians (Charcot, Lepine, Landouzy, Carville, Duret, Pitres, Franck, etc.) have given more definite clinical and scientific form to this most important—because least complex—group of epileptic seizures. Gowers, Dreschfeld, Byrom Bramwell, Ringrose Atkins, Bevan Lewis, and others in this country, have made most important additions to our knowledge of these. Charcot and others have added very greatly to our knowledge of the anatomy and physiology of the brain, by their discoveries of the cortical origin of monoplegias corresponding to monospasms. Their researches merit the highest of all praise, that of introducing method, by a kind of work which can be rated highly as clinical, or physiological, or anatomical, or pathological.

I am strongly of opinion that the epileptiform group should be studied first; and although I pass over it in these lectures for the reasons given, I shall now and then illustrate complex phenomena of epilepsy proper by corresponding simpler phenomena of epileptiform seizures.

Taking, then, epilepsy proper, we say that it is a paroxysmal loss of consciousness. That is only a statement from the psychical side; it is the first statement as to type. We have, speaking of the physical side, to add that there is with the loss of consciousness more or less spasm of muscles (including in that expression spasm of muscular coats of arteries and the like), or equivalent effects of cortical discharges. This is the second statement on the type. The third statement is, that the loss of consciousness is the first, or nearly the first, thing in the paroxysm.

There are degrees of involvement of consciousness, from slight confusion of mind to complete insensibility, or, after the attack, to coma. There are also degrees of the accompanying spasm from, for example, such as slight twitchings of the hands, or that signified by facial pallor, to universal severe convulsion. Presumably, there is an exact correspondence betwixt the psychical loss and the physical condition, but it would be difficult to show it from cases. But we must not suppose that the correspondence is betwixt degree of affection of consciousness, and degree of the physical condition of spasm; inferentially, the correspondence is betwixt the degree of affection of consciousness, and the degree of the physical condition of gravity of the discharge.

Médical, January 5th, 1878, shows. The quotation refers, first, to the results of destroying one of the centres in Hitzig and Ferrier's region. "Whilst the underlying white substance of the destroyed centre progressively loses its excitability, the grey substance of the neighbouring centres becomes turgid and inflamed, and if the inflammation be not sufficiently intense to alter the structure profoundly, a considerable augmentation of its excitability can be proved [a discharging lesion]. Epilepsy is then produced with the greatest facility. To make the animal walk, to exercise pressure upon the skin which has covered the cerebral wound, to pass a sponge over that portion of the brain which is uncovered, etc., often suffices to produce convulsive attacks. It is even very curious to see the grey substance react by convulsive phenomena under the influence of mechanical excitations which, in the physiological state, give place to no appreciable motor reaction. Under these conditions, partial epilepsy may remain limited to those parts of the body the centres of which have become abnormally excited, and respect the limb corresponding to the destroyed centre."

It would be very remarkable if there were not very many degrees of seizures, from those with most transitory and trifling manifestations, to severe universal convulsion, followed by paralysis and coma. It would be contrary to all analogy if there were not. In epileptiform seizures, it is common for the patient to have very partial convulsions before widespread or universal ones. Thus, a patient of mine had, first, attacks of spasm beginning in her right index finger, and "working" up to the elbow; months later, she had severe convulsions, with loss of consciousness, attended by much spasm. A patient may have spasm limited to the big toe, that is, to him apparently so limited, and at other times widespread convulsion. One of my patients used to show his twitching hand to his fellow workmen "as a curiosity", before he had severe seizures which appeared to him of moment.

To return from this illustration to epilepsy proper; we see that there are numerous degrees. At the one end of the scale is the case of a patient, whose seizures are so slight and transient, that a stranger to him might think he only bungled for a moment in conversation; and at the other end is the case of a patient who has loss of consciousness with severe and violent convulsion, followed by deep coma (apoplexy), quite undistinguishable at the first from coma (apoplexy) produced by fatal cerebral hæmorrhage.

The slight seizures are often, at the beginning of a case of epilepsy, so slight and transient, that the patient takes no notice of them; only when a convulsive seizure comes on does he find out what his slight transitory mental confusion, etc., meant.

Upon this basis of degrees, we now divide epilepsy proper. At first glance, we should expect the division to be founded on both the width and degree of spasm and the degree of affection of consciousness. This, however, cannot be done; there is not a discoverable exact correspondence (*vide supra*). We cannot make a clear division on this double basis. For this reason, and because there are degrees of affection of consciousness and degrees of spasm, the division must be not only arbitrary but rough. We make one which seems very rough indeed. It is into slight and severe, or, using French names, into *le petit mal* and *le grand mal*—freely translatable as "little fit" and "big fit".

I do not think the division is quite so arbitrary as it appears to be at first glance; the discharge beginning in the highest cerebral centres has, it is suggested, to overcome the resistance of the middle cerebral centres before it can produce peripheral effects (spasm, etc.). If there be such a thing as epileptic loss of consciousness without any peripheral manifestations, I suppose the explanation is that the effects of the discharge are limited to the highest centres; the currents developed by the primary discharge of some part of the highest centres pass by lateral or integrating lines to other collateral parts of the highest centres and discharge them. This is equivalent to saying that the currents developed, whilst strong enough to overcome the resistance of collateral parts of the highest centres, are not strong enough to overcome the resistance of, that is to discharge, the middle cerebral centres. And when not strong enough to discharge some of the middle cerebral centres, the currents may be strong enough to discharge others. The peripheral effects in slight seizures are manifested on small muscles; the discharge is then strong enough to overcome partially the resistance of the lower centres in Hitzig and Ferrier region, those representing small muscles and containing fewest large cells (Bevan Lewis). If this be so, we see that the correspondence is not betwixt the affection of consciousness and the peripheral effects of the discharge; there may be a discharge strong enough to abolish consciousness, and yet not strong enough to produce any, or very slight, peripheral effects.

By the expression severe, we mean those cases in which there is very much spasm and often universal convulsion with tongue-biting. I exclude these cases.

To recapitulate briefly: we have excluded one large division of cases (epileptiform), leaving epilepsy proper, and then from this remaining division we have excluded the severe seizures. We have only to consider slight cases of epilepsy proper. I must then give a type of a slight case, and this will be some indirect account of the severe ones excluded from consideration. To prevent a possible misunderstanding, let me remark that the terms slight and severe used empirically refer to quantity of manifestation, especially to range of spasm, not to gravity of the case. The slight seizures are said to be the worst for mind; this accords with the hypothesis that the currents develop by discharges of some part of the highest centres, or are, chiefly, limited to the highest centres.

The type of the slight cases is sudden and transient loss of consciousness, closely followed or accompanied by but little spasm and that of small muscles, such as is signified by pallor of the face (spasm of facial arteries), slight spasm of the eyes, hands, etc.

Two things are said about the spasm in these type cases; it is limited in degree (may affect parts in many different regions, however, and in this sense be widespread, or, more figuratively

speaking, widely scattered). In contrast, the spasm in the severe seizures is very great; it is not simply widespread, but universal; at any rate, in severe seizures, all the "visible" muscles, small and large, are affected. In some of the cases we call slight, there is apparently universal slight stiffness; there is a miniature universal convulsion, and possibly the "tremor" described by some patients is a miniature universal clonic convulsion; still, the most striking effects are manifested in small muscles. Instead of saying "small muscles", we should in strictness say "parts, the movements of which require little force for the displacements they have to effect".

There is a kind of spasm which is not obvious. A "vacant" look may depend on slight rigidity of the facial muscles. A patient is sometimes said "to look vacant"; a patient is said to "look fixedly". A loss of expression is essentially an unvarying expression, and very likely it is a matter of opinion whether a given facial aspect in a slight seizure is to be described as a "vacant look" or as a "fixed look". In the stiffened face of paralysis agitans, there is either "loss of expression" or a "puritanical expression", according as we take it. In double facial paralysis, we may take the physiognomy to be either "loss of expression" or a "sulky look". I only say that the so-called "vacant look" in a slight seizure may signify slight even-spread facial spasm, admitting that it may also signify relaxation of the facial muscles. In some seizures, there is at the onset a real expression, which cannot be put down either to a wave of facial spasm, or to a relaxation of facial muscles. There is a very definite emotional expression; one of fear is common. We not uncommonly hear a mother assert of her epileptic child's slight attacks, "She looks frightened, and runs to me". There is also the antithetical expression of indignation in other cases. There are minor degrees of each emotional expression, as astonishment, "looking startled", etc. It is important to notice these expressions; for possibly they will help us to account for the "form" of the actions which in some cases follow seizures.

Again, there may be more spasm in slight seizures than we see. If we look at the face and limbs only, we may see very little, although the respiration is altogether suspended, as is shown by dark lips; we infer that there is spasm either of the glottis or of other parts of the respiratory apparatus.

The slowness of spasm in some slight seizures is occasionally shown by the persistence of movements which widespread spasm would render impossible. In slight seizures, the patient may go on walking apparently well; he may sway about, but, so far as he does walk, so far is spasm absent from the parts concerned in locomotion.

Then, on the other hand, there are conditions which are, or simulate, loss of power; if the attack be sudden, the patient falls down; in some cases, he drops what is in his hands; the head drops when the patient is sitting, is not pulled down by spasm; he may look like a person going off to sleep in a chair. These conditions are not easy of explanation; to say that a man falls or drops things because he loses consciousness is an explanation which explains nothing, and may pair off with such an "explanation" as that chorea is due to "disorder of volition". He may fall, either because some muscles overcontract and destroy his equilibrium, or because he loses power in some.

There are certain movements occurring in slight epileptic seizure which, I submit, should be distinguished from spasm, either tonic or clonic. A common one is a movement of mastication; another is a swallowing movement; there is such an one as rubbing the hands; and there is sometimes writhing of the arms during suspended respiration. To call them movements is to tacitly deny them to be spasm.

Of course, all movements, as well as all spasms, imply nervous discharges; but the former imply slight discharges at particular intervals. I submit that the movements of mastication, swallowing, etc., occurring in a slight fit cannot depend on an excessive discharge; that is, on an epileptic discharge.

It is well to mention some of the names patients or their friends give to slight seizures: "Sensations", "turns", "spells", "thinkings", "stupidities", "reveries", "forgetfulness", "faints", "giddiness", "becoming absent", "quiet faints". I was consulted by one epileptic for "dropping off to sleep in the most extraordinary way".

Now the slightest of these so-called sensations are very often the sole seizures for weeks, or months, or years before the severe fits come on.

When slight seizures have preceded the severe fits, the patient may, until questioned, tell us nothing about the slight ones; he dates his illness from the first severe fit; or, if the slight seizures occur mixed up with severer ones, he mentions the severe ones only. He does not call his slight seizures fits, but "sensations", "symptoms", etc. He may say: "I've not had a single fit since I saw you, but I have had many sensations." These "sensations" are really slight fits, or, when considered in relation to the severe ones, incipient or abortive fits.

We speak first of the mental side of the slight cases—of the affection

of consciousness. As the definition or type of the slight seizure is vague, it may be well to give an example.

A boy, ten years of age, had been subject to fits for two years. At the first, they were so slight that his mother said they thought he was "in a deep study"; he used to sit "as if he were thinking". The attack was only of a few seconds' duration, and no alteration of complexion was noticed. Later on, in his fits he would turn up his eyes—slight spasm of very small muscles. Later still, there was, besides this, occasional shaking of the body (imitated before me by tremor). I saw one. Whilst sitting, the boy blinked his eyes; he did not alter in colour; he then drew in a deep breath, and rapidly all was over. The paroxysm was so sudden and short that I could not make any special investigations.

Now for cases which do not fully resemble the type, for cases which are subtypical. This boy lost consciousness, but it is to be insisted upon that there may be epilepsy proper without loss of consciousness. There may be defect of consciousness only. For example, a patient may say that he is not unconscious in his attacks, but add that he does not know where he is; that he hears people talking, but does not know what they say; this shows that he has not *lost* consciousness, but it shows also that he is defectively conscious; in popular language, he is "confused", becoming so suddenly, and remaining so for a few seconds only. "To be confused" and "to be defectively conscious" are synonymous expressions.

Then we have evidence from what we observe that a patient in an epileptic paroxysm is not absolutely unconscious. The patient may not only utter incoherent words, but may reply to questions vaguely, as, "What do you say?" or, more definitely, he may reply quite correctly. There is the difficulty here that the talking, vague or definite, may be just after a very slight and transient fit; but in an empirical or clinical inquiry for diagnosis the distinction is of little moment.

So our first exception or subtype is that the patient may be only defectively conscious, or, popularly speaking, simply confused.

More than this, there may be, according to the patient's report, no affection of consciousness at all. It is well known that a common precursor of affection of consciousness in a paroxysm of epilepsy proper (slight or severe fits) is a strange sensation at or near to the epigastrium, "in the chest", or "at the heart", some patients may say. Children call it "pain", or "stomach-ache". The sensation is variously described by adults as "sinking", "nausea", and it is frequently said to be accompanied by fear. Now, patients will tell us that, for months before they had any affection of consciousness preceded by an epigastric sensation, they occasionally had that sensation alone, and thought little or nothing of it; some thinking it to be owing to disorder of the liver, although it was sudden and transient (paroxysmal). This, then, would be a second subtype of epilepsy; it would be for a time all the paroxysm.

It may be said, your "definition" by type here breaks down utterly for practical purposes, for in this subtype consciousness is not affected at all. If literally taken, it does break down; we ought and must take such cases as subtypes. It is certain that these sensations may occur paroxysmally for months without anything further. The fact that such a paroxysmal sensation is often epileptic in nature is shown by the future of the cases: undoubted epileptic affections, attacks with convulsion and tongue-biting come on, and each of these severe paroxysms is preceded by the sensation. What was once all, is now the so-called warning of the severe fit. Strictly speaking, it was when it occurred alone a rudimentary or incipient fit of epilepsy, and when it becomes what we call a warning, since a severe fit follows, it is the inception of the severe fit. Besides, I should doubt whether consciousness was not affected to some trifling extent. However, I state what patients say, and at any rate the affection of consciousness is insignificant or most transient, or both.

Then there are cases of slight epileptic vertigo. By epileptic vertigo, I do not mean any sort of "confusion in the head", I mean real vertigo; a sensation of movement of external objects, or a sensation of turning of the body itself; at the beginning of some severe attacks of epilepsy proper, the patient does actually turn to one side, "turns right round", as the friends say. Hence I do not use the term epileptic vertigo as the name for a slight *degree* of seizure, but specially for a slight seizure of a particular *kind*, or for a seizure beginning in a particular way. I will not say there is no defect of consciousness in cases of epileptic vertigo, but it may be very slight and transient, and according to the patient and his friends none. I do not believe that consciousness is entirely unaffected in any sort of giddiness, however caused. Let a man try to think, when giddy, of something complex and difficult to him, to tackle some problem he could just manage to solve when well; he would fail when giddy.

So, then, whilst we take paroxysmal *loss* of consciousness as our type, there are subtypes. There is not only loss of consciousness, there is defect of consciousness, and there is also no affection of consciousness.

The paroxysmalness of the seizure and the sequel show that each is truly epileptic; they occur at different periods of the same case. Indeed, we may have each of the three degrees at different times in one case.

REPORT

ON

THE OUTBREAK OF DIPHTHERIA IN THE GRAND DUCAL FAMILY OF HESSE-DARMSTADT.

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I HAVE undertaken the more readily to publish a report in an English Medical Journal on the outbreak of diphtheria at the Grand Ducal Court of Darmstadt, because I feel only too deeply that, before all others, the people of England have a right to demand an exact account of the morbid process, and of all the circumstances connected therewith, by which they have lost a much-beloved member of their august royal family.

From the very beginning of the epidemic, a general interest has been evoked by the fact that a most severe form of diphtheria attacked, within a very few days, not fewer than six members of a family, which appeared to be much less exposed than others to obnoxious influences, and to the common modes of infection. Rapid limitation or destruction of the poison, which had been imported from without, might have been expected as another consequence of the advantages which their superior social position seemed to offer to them. Again—and this fact has not yet been much emphasised—it deserves attention, that the disease, in spite of its malignant form, has been limited to the Grand Ducal family, whilst not a single member of the numerous household or of the entire staff of nurses has been attacked. It will be seen that the facts enable us to draw quite special conclusions as to the nature of the contagium acting here, and as to its mode of dissemination. Finally, the intensity which characterised the diphtheritic process in all the patients without exception, and the increase of malignancy which took place *pari passu* with the propagation of the infection, are two most remarkable features. It is not at all rare for an entire family, or the greater number of its members, to be attacked by epidemic diphtheria; but, as a rule in such instances, there will be graver and slighter cases, the latter generally being even more frequent than the severe ones. In this instance, however, all the cases belonged, from the very beginning, to the worst forms which the disease can take, and in each individual case there was danger to the patient's life within a short time after the outbreak of the affection.

The commencement of the infection, and the propagation of the local process in the first affected members of the Grand Ducal family, were described, on the 25th of November, in the *Berliner Klinische Wochenschrift*. From this, the report passed into several German and foreign journals, and an abstract was given in the *BRITISH MEDICAL JOURNAL* of December 14th.

The first case (Princess Victoria, aged sixteen) was diagnosed by Dr. Eigenbrodt on the 6th of November. The patient was at once isolated, and the Grand Ducal family most carefully watched. There was no new case during the next five days, but after that, at intervals varying from five and a half to eight days after the first outbreak, the other members of the family were attacked, between the 11th and the 13th of November. Princess Alice, aged six, fell ill in the night of the 11th-12th of November; Princess Mary, aged four, in the course of the 12th; Princess Irene, aged twelve, in the night from the 12th to the 13th; the Hereditary Grand Duke Ernest Louis, aged ten, on the afternoon of the 13th; and finally, on the 14th, eight days after the beginning of Princess Victoria's, and two and a half days after the beginning of Princess Alice's illness, His Royal Highness the Grand Duke himself was attacked.

After this, no further propagation of the disease seemed likely to take place; the whole of this large household, the numerous staff of servants and nurses, and all others having intercourse with the palace, were spared. Suddenly—all the previously infected persons (with the exception of Princess Mary, who died during the night of the 15th) being already convalescent—on the 7th of December, twenty-three days after the last infection, Her Royal Highness the Grand Duchess fell ill, with high temperature, and all the symptoms of the most malignant croupous form of diphtheria. In the previous cases, the