THE

British Medical Journal.

THE JOURNAL OF THE BRITISH MEDICAL ASSOCIATION.

LONDON: SATURDAY, JULY 3RD, 1926.

A Lecture ON THE EPILEPSIES.*

BY

S. A. KINNIER WILSON, M.D., B.Sc., F.R.C.P.,
PHYSICIAN FOR OUT-PATIENTS, NATIONAL HOSPITAL, QUEEN SQUARE;
NEUROLOGIST, KING'S COLLEGE HOSPITAL; CONSULTING
NEUROLOGIST, METROPOLITAN ASYLUMS BOARD.

THE condition commonly called epilepsy, a "disease" characterized by fits, is familiar to every practitioner of medicine and to many a passer-by in the street; it has formed the topic of innumerable dissertations through the centuries, and has been claimed as a therapeutic preserve by each successive variety of healer, from the divine to the psycho-analytic. As has been the case with other common nervous ailments, from time to time there has been a kind of spurt in the race for a panacea, followed only too often by a reaction after the effort; and to-day various rival and mutually contradictory methods for dealing with it are being advocated, the efficacy of no one of which can be confidently and constantly relied on.

To obtain that definiteness of treatment which must always be his goal, the physician must know the nature and etiology of the morbid condition with which he is dealing and the mechanism of production of its symptoms. Now it so happens that while the external manifestation of an ordinary epileptic fit is apparent to everyone, its inward or neural genesis and progression offer problems of great difficulty and complexity, on which the outward phenomena at the best throw but an imperfect light; from them we are compelled to infer a mode of physiological activity our theories of which are largely speculative, while the symptoms themselves are essentially so transient that the opportunity for objective study is very often too brief to be adequately seized. Like some other nervous affections, in fact, epilepsy is a condition superficially easy of diagnosis but etiologically and pathogenically obscure, and wide excursions in neurophysiology and experimental neuropathology have to be taken to gather such relatively meagre data as are at our disposal in this connexion. Forestalling for the moment what may subsequently be alluded to more fully, I may now state that even if a psychogenic origin be assumed, or proved, for one or other of the epilepsies, the physiological problem presented by the clinical symptoms of the fit is thereby left entirely untouched. To say that a given fit is psychogenic provides no solution; in the words of Hughlings Jackson, "all psychological explanations of physical disabilities are merely verbal," and, as anyone can see, a fit in the ordinary sense is a physical thing.

I propose in this lecture to discuss some of the general and physiological questions connected with epilepsy. Our first is concerned with nomenclature and definition.

What is Epilepsy?

In ordinary parlance, as sanctioned by long usage, epilepsy is understood to be a disease the outstanding symptom of which is the occasional fit. One has only to mention this conception to realize forthwith its thoroughly unsatisfactory nature. There are many nervous states

of organic origin in which epileptic fits are a symptom; on the other hand, the conception is vitiated by the admission into the category of epilepsy of conditions characterized by "attacks" of a class quite different from the fit as usually understood, but supposedly "equivalent" to epileptic fits because of their recurrence, transient nature, the patient's subsequent conscious ignorance of what takes place during them, and so on. The epileptic fit is nothing else than a symptom; no such disease as epilepsy exists, or can possibly exist; even by resort to such epithets as "essential" or "idiopathic" epilepsy we in no sense add a term of differentiating quality, since the pathological element constituting the disease-component (whatever it may be) is not thereby expressed. Further, assuming that a person who suffers from fits is the subject of a disease which may as well be called epilepsy as anything else, we are unable to say where such a diseased state begins and ends. As is known to every clinician, an individual may have one fit only in the course of his life, or a few, at the widest intervals, as in the case of a lady who had an epileptic fit at the age of 14, when her period of reproductive activity commenced, and another at 44, when it closed. To dub such cases epileptic in the accepted and always somewhat grave sense is clearly unwarranted, but the nature of the attacks shows that they cannot be called by any other name if the term is to retain any descriptive value. Other individuals exhibit at the outset attacks justifiably regarded by the clinical observer as faints, which subsequently merge by ill recognized degrees into the epileptic category. And it is scarcely necessary to point out that ordinary major epileptic fits are often for a time the sole discoverable indication that a serious organic diseased cerebral state is developing—vascular, inflammatory, toxic, or neoplastic.

Nor is the problem of delimitation easier in respect of the clinical varieties commonly called Jacksonian or epileptiform, and minor, or *le petit mal*. By objective observation on a single occasion of the outward characters of a series of muscular twitches it is practically impossible in many instances to say if they belong to the category of incipient Jacksonian epilepsy, or are rightly described as myoclonus or paramyoclonus, or as tic; additional data not concerned with the extrinsic features of the muscular contractions are often requisite for an exact diagnosis. Most of us have in recent years become familiar with curious varieties of tonico-clonic "involuntary" movements as a sequel to epidemic encephalitis which some Continental observers are already describing as "striatal epilepsy," as though (vain hope!) the solution of their pathogenesis were thereby facilitated; and many practitioners must know of cases classed by them as *petit mal* in which they have never been sure whether the clinical appearances really justified that terminology.

The truth is, as it seems to me, that we have been content to classify fits by certain extrinsic characters or qualities mainly because of our relative ignorance of the intrinsic features of the disorder of neural function which exteriorizes itself as a fit.

What Constitutes a Fit?

When we come to examine the connotation of the word "fit" we find ourselves in no less a quandary than in regard to the term "epilepsy," for in the ordinary acceptance of that useful expression it stands for conditions as

* The Annual Harveian Lecture delivered before the Harveian Society of London on April 22nd, 1926.

widely separable as a convulsive movement limited to a segment, or part of a segment, of a limb, or to a part of the face, and highly elaborate co-ordinated acts of the whole person, sometimes of considerable duration, comprised under the term "hysterical fit." The word cannot even be restricted to phenomena of a hyperkinetic class, for it might legitimately be employed to designate certain attacks of which motionlessness is the prominent feature. In other words, "fit" and "convulsion" are not synonymous, and the expression "convulsive fit" is not tautological.

Since, then, it is used to include attacks which vary enormously in type, degree, and duration, not to mention accompaniments, it might be supposed to have become almost meaningless, yet its appropriateness need not be impaired if adequate restrictions are made, and if we can trace the action of some definite physiological mechanism behind its varied phenomena.

General consent, I imagine, will be given to the view that nerve cells in a state of health store up energy *in potentia* derived from the specific nutritional material they absorb or ingest, and that in functional activity normal movements (the reference is solely to motor nerve cells in this connexion, although of course the view applies to all varieties of cell) are the outcome of liberation of this energy in kinetic form, to the accompaniment of expenditure of cytoplasmic material. Such normal liberation of energy is orderly, moderate, and controllable. We can, however, readily imagine a condition of motor nerve cells brought about by abnormal nutrition, and resulting in the development of a state of high tension and instability, whereby they will discharge suddenly, with an excessive liberation of energy, either when a postulated maximum of disequilibrium has been attained, or when a sufficient stimulus reaches them.

Following the profound discussion of this matter by Hughlings Jackson¹ in his Lumleian Lectures of 1890, I believe that the essence of a fit with convulsive movement consists in the exaggeration of a normal physiological process—that is to say, in the sudden, excessive, and yet purely temporary liberation of kinetic energy in a series of motor nerve cells, the visible consequence of which is a sudden and excessive development of many movements at once. On this hypothesis it is clearly possible for any constellation of nerve cells in the nervous system to become highly unstable or overcharged, and to discharge accordingly, so that the term "epileptic fit" might be employed, and legitimately so, for any paroxysmal symptoms attributable to the process outlined above. Nevertheless, at this point clinical expediency steps in, to warn us not to extend the meaning of the term unduly, even if only theoretically. Avoiding, therefore, too inclusive a conception of the word, we are at the same time arbitrarily limiting its designation when we confine "fit" to a hyperfunctioning of motor cells. The clinical characters, on the other hand, of suddenness, disorderliness, "caricaturing," and excessiveness are cardinal features resulting from the physiological process, and without these no symptoms can be justifiably regarded as belonging to the category in its strict sense. This contention will of itself serve to exclude a considerable number of "involuntary" nervous movements which are the sequel to release of neural function. None the less, as already indicated, it is often far from easy to draw clear distinctions between minor types of "involuntary" movement, especially if the characters just mentioned are not conspicuously present.

The Process of Discharge in a Fit.

Our next question concerns the determination of the discharge, and its radiation or spread. If we take an epileptiform or Jacksonian fit, we must concede that the focus of pathologically altered cells may be very small. For example, a patient may suffer from Jacksonian fits which always commence by twitching of the same thumb and forefinger; between the attacks he may be perfectly able to move that thumb and finger "voluntarily," hence there can be no material structural defect of the corresponding cortical motor nerve cells. Some pathological condition, however, in the immediate vicinity leads at intervals to such alteration in their cellular nutrition that

they become unstable and prone to discharge "involuntarily" once the adequate provocation arises. As already hinted, we can only speculate as to what this stimulus is, but three possibilities suggest themselves: (1) it may be afferent or sensory, setting off the charge as the detonator fires the powder of the cartridge; (2) it may be intracellular, as though there were "spontaneous combustion," when a maximum of tension and instability is reached; (3) it may consist in removal of some inhibitory factor normally restraining the cell from activity. If this third possibility is entertained the problem is merely put a step further back, for we do not know what neutralizes the hypothetical inhibition and leads to decontrol.

Once the "discharging lesion," as Jackson termed it, is in action, the tendency is in the great majority of cases for its effects at once to spread to neighbouring linked physiological groups of nerve cells, so that in the course of a very brief space of time collateral cells are in discharging activity. It is in no sense likely—indeed, it is hardly possible—that these are anything else than healthy and normal, hence the important conclusion is reached that very little of a Jacksonian fit is directly due to the discharge of abnormally nourished neural units, and that by far the greater part of it is produced by consecutive discharges of normal stable cells. If this be granted, the radiation of an epileptiform fit is entirely a physiological phenomenon, and offers proof of the fact (for which there is abundant evidence otherwise) that healthy neural mechanisms may become epileptogenous, without being in the remotest degree diseased.

In the case of the general, or major, epileptic fit the circumstances cannot be materially different. While on occasion a local commencement (turning of head and eyes to one side) is indicative of the cell groups first implicated in the liberation of energy, in many instances universalization of the convulsion is almost immediate, and the fit is said to be severe. The supposition here is that the initial discharge is particularly sudden and powerful, and that many series of bilaterally situated cell groups have become unstable and, as it were, explosive. With a great quantity of this explosive material radiation through various neural levels evidently occurs with extreme rapidity, and Jackson's words contain no exaggeration:

"speaking figuratively . . . there is the mad endeavour of the highest centres to develop the maximum of function of every part of the body . . . and of all parts at once; the phenomena of a very severe epileptic fit show that this endeavour is nearly successful; the patient is almost killed by the paroxysm, and is nearly dead (deeply comatose) after it."

The Neural Site of a General Fit.

It is of interest from the standpoint of neurophysiology to determine if possible the neural site of a general epileptic seizure, a question closely connected with the further matter of the type of movements in a fit. This latter subject is examined later, and is to be considered in association with what is now stated.

If we agree that the property of excessive and uncontrolled discharge is common to all cell groups on occasion, whatever their neural situation, it follows as a corollary that at any of the physiological levels of the nervous system epileptic phenomena may make their appearance. Owing, however, to the intimate physiological integration of these levels the symptoms can scarcely be confined to one only unless the initial "discharging lesion" is mild in degree and incapable of overcoming such resistance as collateral groups offer to excitation. Thus, as already indicated, in the case of a severe fit the chief feature is the rapid spread of the excitations through various levels and to differing mechanisms. It is probably thought generally, or is at least a tacit assumption, that ordinary major fits are the outcome of discharges of cortical nerve cells throughout the middle physiological level of both hemispheres (the Rolandic motor cortex)—as far, that is to say, as the visible movements of trunk and limbs are concerned. The view held by Hughlings Jackson was that general epileptic seizures begin in the frontal lobes (his "highest level"), whence the underlying process spreads to and implicates the middle level. Whether this is so or not is extremely difficult to decide; no constant symptom referable to release, or stimulation, of function of the

frontal lobes can be confidently diagnosed; indeed, unless we accept turning of the head and eyes as a frontal symptom (which experimental evidence suggests it may be), no other positive symptom of an ordinary epileptic seizure can be said to have a frontal localization by itself, certainly not the loss of consciousness. It is true that various clinical and pathological data may be taken to indicate (as was discussed at some length in my Croonian Lectures²) that a large part of the brain in front of the precentral gyri subserves motor functions; but any motor phenomena its excitation might conceivably produce (with the exception of the not constant head and eyes movement already alluded to) are possibly at once submerged by the spread of the physiological process to the middle level centres. (This point is also referred to below under the heading of "Type of Movement.")

As far as the familiar motor area of the Rolandic cortex is concerned, from the physiological standpoint it is electrically excitable, and from the anatomical it contains the Betz cells, which are the origins of the pyramidal or cortico-spinal tracts running to the ventral horn motor cells of the spinal cord. These cortico-spinal paths are the chief available routes for cortically initiated movements to exteriorize themselves via the spino-muscular units, and in the case of the movements of Jacksonian epilepsy this is definitely the kinetic route taken. When, therefore, the limbs and trunk are convulsed in a severe fit, the idea is that simultaneous powerful discharge of whole groups of motor cells in the bilateral Rolandic areas is taking place, the result being at first an absolute contention of movement, resulting in relative muscular rigidity—tetanic contractions strictly comparable to that obtained by faradic stimulation of a cortical motor point as long as the stimulus is kept up.

The question arises, however, whether the motor phenomena of a general epileptic fit may not be produced at other levels than the cortical. I have already said that a "discharging lesion," if of sufficient intensity, will scarcely confine itself to one physiological level, but will rapidly implicate others; hence discharges may conceivably be taking place, in this instance, at the spinal level. It is recognized, further, that epileptic fits can be produced experimentally in decerebrate animals in which the brain stem has been transected and (*inter alia*) the cortical motor areas and pyramidal tracts put completely out of action. When only the pons, medulla, and cord are experimentally left, fits are still produceable, and convulsions may in these circumstances develop *sub finem* just as the respiratory centres are failing. Of course, they are possible only because the spino-muscular units are still living, to be convulsed, but the anatomical paths from medulla to ventral horn cells, traversed by the excitations, are not with certainty known. So far as I am aware, strictly spinal epilepsy—that is, in the purely spinal animal—has not been found experimentally, though there is no theoretical impossibility of its occurrence. In view of these facts and others which are germane but cannot here be detailed, it has been several times suggested, in recent years, that the phenomena under discussion are the result not so much of excitation spreading from higher to lower levels as of removal of inhibition from higher to lower levels, and that the symptoms of the seizure—that is, the motor symptoms—are produced by release of function; that the epileptic process inhibits controlling centres (say, cortical centres), and that "lower" centres are "let go." Hughlings Jackson himself fully realized this possibility; in his Lumleian Lectures¹ he referred to various "inhibition hypotheses," on which, however, he expressed no decided opinion. It is clear, I think, however, that he was so convinced of the far greater complexity of movement representation in the cerebral cortex that he could not accept the possibility of discharges at low levels being comparable to those of high levels; in his own words:

"it would be marvellous if excessive discharges beginning in centres lowest in rank produced fits like those . . . which are produced by excessive discharges beginning in parts of the more evolved centres, the middle motor cerebral centres. . . ."

The problem is one calling for contemplation from every angle, otherwise we are in danger of reaching a rash conclusion through neglect of the complexity of the

factors. Due attention should be given to the following considerations.

1. While it is agreed that fits of a general kind can be produced in the decerebrate animal, Cobb and Uyematsu³ have shown that a much larger dose of a convulsive agent (absinthe, thujone) is required to cause a convulsion in these circumstances than for a similar convulsion in an intact animal, in which the cortex is active, and they conclude that the cortical motor cells are those most easily and readily stimulated by these convulsants. Pike and Elsberg⁴ also admit the greater susceptibility of the uninjured cortex to experimental absinthe fits, although their investigations demonstrated the possibility of the occurrence of both tonic and clonic convulsions in animals deprived of both motor cortical areas, provided an interval sufficient for the return of "good locomotor reactions" was allowed to elapse after the initial operation. These fits "did not differ essentially from absinthe convulsions that occur in unoperated animals"; and, according to Cobb and Macdonald,⁵ "the march of events during a convulsion produced by thujone is very similar to that seen during an epileptic fit." Data of this kind, valuable as they must be acknowledged to be, do little more than substantiate the view already outlined, that in appropriate circumstances healthy neural mechanisms below the level of the cortex may become epileptogenous; they certainly do not validate the conclusion that the neural origin of the major convulsions in intact man cannot be cortical. The motor display of major fits is undoubtedly accomplished through spino-muscular units, but more evidence is requisite before the contention can be sustained that the convulsions of organic cerebral disease are, as it were, allowed to occur by removal of some cortico-spinal inhibition.

2. The evidence associating the phenomena of Jacksonian epilepsy with local cortical disease is sufficiently satisfying to dispel any doubt as to their interrelation; that the "march" of such fits is in strict accordance with the accepted physiological localization of face, trunk, and limb centres in the cortical motor area, while no representation of an identical kind has been determined in respect of spinal motor centres, is of fundamental significance. Thus I have twice seen Jacksonian fits involving simultaneously solely the forefinger and thumb, and the corner of the mouth, on the same side, a combination readily explained by the juxtaposition of the corresponding cortical centres, but meaningless and inexplicable were the epileptogenous site at any lower physiological level.

3. Assuming, for the sake of argument, that the phenomena of epilepsy belong to the category of release phenomena, and that cortical disease only removes cortico-spinal inhibition, we are faced with the problem that the speculation raises as many difficulties as some may think it appears to dispel. We have to explain the violence and the caricaturing element in the motor discharges, which are altogether unlike those motor phenomena—for example, involuntary flexor spasms—that occur when spinal mechanisms are admittedly liberated from cortical control, as in spastic paraplegia. We have to explain the disorderliness of the epileptic movements, again quite unlike the organized, systematized, scarcely variable movements of released spinal reactions. There is the further difficulty, also, that this postulated temporary release from cortical control is followed by convulsive movements while more permanent release is not.

It seems impossible to avoid the conclusion that there is another element in epileptic discharges than merely a temporary cessation of inhibition from a higher level. We must be careful, indeed, as to our use of the term "inhibition"; transcortical "inhibition" is at least as probable as cortico-spinal inhibition, and the phenomena of epilepsy may conceivably result from decontrol and yet have a cortical site. I have gone more fully into this transcortical question in my Croonian Lectures² already mentioned, to which the reader is referred.

4. Perhaps the most significant point is whether, as a physiological fact, lower (say ponto-bulbo-spinal) centres are capable of exhibiting in action as elaborate motor manifestations as are derivable from the cortical motor ganglia, if the expression be allowed. I have already cited Jackson's view that it would be "marvellous" if this were

the case. Unfortunately, this is a matter in regard to which definite observations are unusually meagre and inconclusive. The question is simply this: Are the tonic-clonic convulsions produced by absinthe in the decorticate or decerebrate animal identical in all respects with those readily caused by smaller doses of the convulsant in the intact animal? Only minute observation can settle the point. In respect of man, my clinical experience has led me to maintain that in conditions approximating to physiological decerebration in man the fits that may be met with are essentially tonic in character, and not tonic-clonic, as in intact man. The data supporting this conclusion have been recorded elsewhere.⁶ Such evidence as is available, therefore, in respect of diseased states in the human subject does not warrant the speculation that the march of events in epileptic cases is the same whatever the neural level of the discharges; the elaborate sequence of the complicated phenomena of a major attack is not such as can be explained by implication solely of low physiological levels. This brings us to the subject of the type of movements in epileptic fits.

The Type of Movement in a General Fit.

The ascertainment of the types of movement to be seen in the case of a general fit is a matter of observation, their interpretation matter for discussion. We have to distinguish, also, changes in the motor symptoms during the course of the fit.

Perhaps it is well, in this connexion, to point out that in the fit our concern is with movements and not with muscles. In my Croonian Lectures I have given reasons derived from personal observation for believing that there may be a representation of muscular units in the cerebral motor cortex, but for our present purpose it suffices to say that the motor display is to be regarded as one of movement.

It is most important, also, to realize that "ordinary," co-ordinated movements may and do occur along with and separate from those that are called "convulsed." For example, apart from the latter, the epileptic patient may, during the fit, make champing movements of the jaws, may smack his lips, spit, make clutching movements at the throat; further, when the convulsive phase is over, and while still unconscious, he may make even more elaborate movements of his limbs, such as plucking at his clothes, etc., all of which movements present the characteristic features of purposive, "voluntary," cortical movements, except that they are outside his voluntary control. It is quite impossible to entertain any other conclusion than that these have a cortical site; their complexity and purposive and deliberate character exclude any other explanation.

Convulsive movements proper cannot be considered as anything else than caricatures of normal movements. They are violent, powerful, disorderly, and generalized, and it is not easy to recognize in them either rhyme or reason. Varying considerably in different individual cases, symmetrical or asymmetrical on the two sides of the body, implicating trunk more than limbs or vice versa, or both equally, they represent a maximum of movement in a minimum of time, and no voluntary mimicry can give other than a faint reproduction of their intensity and severity. The arms may be extended fully and hands clenched, or the former may be flexed; the thumb may be inside the clenched hand, or pressed against the forefinger, or the wrist may be powerfully flexed and the hand more open. The legs may move up and down in violent kicking movements, occasionally in crude alternation, but far more commonly in more or less symmetrical and synchronous flexion and extension. From my personal observation and study of very many hundreds of epileptic fits, both general and Jacksonian-becoming-generalized, I cannot satisfy myself either that all epileptic fits, roughly speaking, are alike, or that any definite scheme or organization of movement is revealed in their manifestations. On the contrary, I follow Hughlings Jackson in holding that a convulsion is a "contention of complex, and also of simplest, movements. In this contention the individuality of each movement is lost."

It is customary to speak of a tonic and a clonic phase

of the convulsions. The former precedes the latter in the average general fit; Jacksonian fits are mainly clonic; a special variety occurs (as already mentioned) in clinical conditions corresponding more or less closely to physiological decerebration, and is known as the "tonic fit," since clonic manifestations are not usually an accompaniment.

The tonic stage of a general fit is one of practically universalized immobility; a number of movements, mutually cancelling each other, are developed with remarkable abruptness and equally remarkable power; the result is a rigid state, a "single big useless movement," as Jackson said, followed at length by a succession of such rigid states, a clonic stage, a series of so-called movements "which do nothing but 'mark time.'" Eventually the patient, exhausted by the severity of this contention of movements, which get slower and slower, sinks into a condition, sometimes not a little alarming, of profound collapse and coma, brief though it may be in reality. On at least two occasions I have seen a condition in all its essentials as closely resembling death as I am ever likely to observe.

Holding these views of the physiological phenomena of a general convulsion, I can scarcely regard with patience various speculations indulged in by some, to the effect that the convulsed movements are identical with those of the foetus *in utero*, and that they represent an attempt on the part of the sufferer to retreat again for shelter from an unsympathetic environment into the stillness of the amniotic fluid. I do not consider that they have any "meaning" whatever; they are of no more "significance" than an explosion of powder. Even in respect of the "march" of movement in a Jacksonian fit, the clinical type of exaggerated, caricatured movement in no way resembles those that are called "voluntary" or purposive, while the "march" of the movements follows anatomico-physiological lines alone and presents no point of comparison, but many of contrast, with the co-ordinated movements of a limb in health.

Summarizing at this stage the conclusions reached, largely on clinical grounds, I may state them as follows:

1. The convulsed movements of a general fit resemble those obtained by tetanizing electrical stimulation of the motor cortex (middle level) in their crudeness and severity, and by the action of convulsants on cortically intact experimental animals.

2. In their elaborateness, disorderliness, and violence they do not resemble the involuntary movements known to occur when spinal levels are released by cortico-spinal disease. The reactions of the spinal animal are orderly, systematized, and scarcely variable with variations in the stimuli evoking the reactions.

3. Combinations of movement have frequently been seen in cases of Jacksonian epilepsy which cannot be explained by any known juxtaposition of centres at infracortical levels.

4. Along with the convulsed movements of general fits, "ordinary" movements may occur; these resemble "voluntary" movements, except that the element of "volition" is wanting; their neural site must be cortical, but is certainly not Rolandic. It is therefore conceivable that transcortical decontrol is responsible for some of the discharging movements.

5. As far as man is concerned, fits the origin of which is at some infracortical level are not in all respects identical with those of the ordinary major attack.

The Phenomenon of the Epileptic Aura.

Consideration of the question of the nature and origin of the epileptic aura is not calculated to lend any support to the view that the manifestations of epilepsy are all essentially infracortical. While not constant (as far as the mind can subsequently recall the initial stages of an attack), it is when present an integral part of the seizure and of localizing value, as every student of the subject is aware. We must keep rigorously in mind the fact that the aura is definable as a sensation, crude or elaborate as the case may be, either referred by the patient to some part of the body or limbs or belonging to one or other of the special senses. Being a sensation, it is a phenomenon of

another order than the convulsive movements; it is a psychical thing, in consciousness, and all that can be said of it physiologically is that we believe it arises during functional activity of some or other central, cortical, sensory mechanism. Because of its being in consciousness its cortical site must be granted (I say nothing here of the speculation that some thalamic activities are accompanied by consciousness of them). In Jackson's clear diction:

"Crude sensations (psychical) and convulsion (physical) are in no way comparable; the comparisons and contrasts are of excessive discharges of sensory elements *during which* crude sensations arise, with such discharges of motor elements *from which* convulsion arises."

I do not know that Jackson, who was always conspicuously careful in his choice of words, was justified in claiming the discharge of sensory elements, constituting in consciousness the aura, to be "excessive." On the contrary, it has always appeared to me that, apart from the occasional normal movements accompanying convulsive movements (alluded to above), the aura is the one thing about the epileptic fit which is not disorderly, distorted, or caricatured. Its crudeness or complexity depends on whether a low-grade or high-grade sensory mechanism is implicated, but we rarely, if ever, can say of it that it is confused, or made up of conflicting elements, or that it is of exaggerated intensity, or contains any excessive quality.

The problem of its origination is in reality that of hallucinations in general. A sensory aura is a hallucination, be it of the visual, auditory, muscular, gustatory, olfactory, viscerosensory, or any other sense class. Very valuable data bearing on questions of cortical localization have been furnished by a consideration of the sensory auras accompanying epileptic seizures occasioned by organic cerebral disease, instances of which might easily be provided from clinical experience. But we are concerned rather with the general problem of hallucinosis. The difficulty of deciding whether such phenomena are release phenomena or are irritative or excitative is, in my opinion, verbal rather than essential. Completely ignorant as we are of the process whereby function of a sensory mechanism is accompanied in consciousness by what is called a sensation, and, for that matter, of the point in a given sensory arc at which its activation becomes a conscious thing, we are not in a position to assert that a hallucination arises either because a sensory mechanism is "let go" from the control of some hypothetical inhibition, or because the mechanism is being activated by some usual or unusual stimulus. Even if inhibitory control is at first to be removed (by some equally unknown and speculative process), some excitation might still be necessary to "touch off" the prepared neural unit or system.

That hallucinations are common accompaniments of toxic or toxæmic states in no way helps us in our search for a physiological explanation of their development. When, as in the case of uncinate or temporo-sphenoidal epilepsy, a gustatory or olfactory hallucination is often associated with a visual or visuo-auditory hallucinatory state of a particularly elaborate, if certainly brief and fleeting, character, the most we can say is that this state arises in the course of the development of that abnormal physiological process which underlies all the phenomena of the fit, the aura included. Although all auras are psychical, rendering the separation of a so-called "psychical aura" meaningless, there is no good reason for supposing that this sensory component is in some fundamental way different from the motor components, as far as its development in the course of a fit is concerned, and none for imagining that its type, or recurring character, constitutes proof of the psychogenic nature of the epilepsy. Elaborate hallucinatory manifestations occur in persons who are half-drowned, half-hanged, half-suffocated; the hallucinations of uncinate epilepsy may be, and frequently are, as definite an index to the existence of organic cerebral disease as the twitching muscles of a Jacksonian case; the "déjà vu" phenomenon and the peculiar psychical condition of reduplicative paramnesia can occur as the aura alike of organic and of so-called idiopathic epilepsy. Elsewhere I have analysed these and analogous psychical manifestations of ordinary epilepsy at some length, and to this paper the reader is referred.

The Question of Consciousness in Epileptic Fits.

Some epileptic attacks are attended by loss of consciousness; others are not. Unconsciousness may supervene at the outset, or develop subsequently; it may be extremely brief in duration, or may continue for some time after all convulsions have ceased. Further, of itself it is no index to the severity of the fit; a severe Jacksonian attack may from first to last involve no loss of the senses, while a petit mal seizure may consist of little else than a transient conscious "blank," an "absence épileptique." With this considerable variability in the interrelation of unconsciousness and motor accompaniments the task of correlating their respective causes is not rendered easy.

The evidence for assigning the former to changes in the cerebral circulation is doubtless familiar to most physicians; it was excellently marshalled by A. E. Russell⁸ in his Goulstonian Lectures of 1909, which should be read by all who seek an explanation of the difficult problems which epilepsy offers. Starting with this view, a line of argument in respect of the pathogenesis of epileptic symptoms has been developed by the same author, more or less as follows: Alterations in the general circulation afford an adequate explanation of ordinary fainting fits and syncope attacks; they also explain the symptoms of more prolonged attacks in which cardiac, vasomotor, and cerebral phenomena can be observed; the suddenness of some faints, and the conversion of faints into fits, suggests the possibility of the epileptic fit itself having some origin analogous to that disorder of the cerebral circulation which causes unconsciousness. To this point I shall revert immediately; for the moment I may say that while transient cessation of that circulation is the most plausible explanation of sudden unconsciousness in epilepsy, certain difficulties remain. Consciousness is absent in negative functional cerebral states, such as concussion and coma; it is also absent during positive, excessive, functional cerebral states—for example, during the period of convulsions. Many clinical instances of the former are unaccompanied by any discharging or convulsive phenomenon whatever; conversely, a Jacksonian patient may exhibit many severe convulsive movements before consciousness is lost. It is undoubtedly difficult to elucidate both negative and positive states by reference to the same process—namely, disturbance of cerebral blood flow. General cerebral anaemia explains too much, as it were; with it the excitability of the cortex certainly diminishes, and absence, not excess, of movement should ensue. On the other hand, we might postulate (though we know no special mechanism for it) a cortical anaemia, producing unconsciousness, with retention of the circulation at lower neural levels, allowing activity of released subcortical neural systems; while such a hypothesis might conceivably provide an explanation of the features of the general fit (though, for reasons given above, I do not consider it can) it will not elucidate those of the Jacksonian variety. In view of the occurrence of degrees of unconsciousness in the epilepsies, and of the fact that negative and positive states do not proceed *pari passu* (as far as we can judge clinically), a simple theory of cerebral anaemia is insufficient for the variability of the phenomena.

Relation of Epileptic Symptoms to the Cerebral Circulation.

We may, however, in conclusion, pursue this matter a little further. The theory that the paroxysms of epilepsy are a sequel to and determined by contraction of cerebral vessels dates back at least as far as the time of Brown-Séquard. Hughlings Jackson⁹ himself favoured the possibility of the paroxysm being caused by a local vascular contraction; he thought that "successions of different movements are developed by contraction of arteries"; "a convulsive paroxysm is developed by a stronger and more continued contraction" of the same arterial branches as, through coarse disease, are imagined to be producing abnormal and persistent changes of nutrition in the distribution of that artery; "it is, I speculate, through the arteries that sequence of movements is developed, whether those movements be spasm passing up the arm and down the leg, or whether they be the orderly sequences of movements in health."

More direct evidence, however, has taken the place of speculation. Russell⁸ emphasizes the importance of the facts that the symptoms of the various stages of the epileptic fit are closely mimicked by those following heart-block in Stokes-Adams disease; that uraemic convulsions are indistinguishable from those of epilepsy and are almost certainly due to cerebral oedema, consequent increase of intracranial tension being followed by temporary failure of the cerebral circulation; that in some recorded cases of value temporary cessation of the heart's action (disappearance of radial pulse) has immediately preceded the development of a fit. A number of direct observations of the human cortex during an epileptic convulsion have been made by surgeons (Horsley,¹⁰ Kennedy and Hartwell,¹¹ Leriche,¹² Horrax¹³). Sudden blanching of the cortex and pial vessels, and arrest of cerebral pulsation, is at once succeeded by pronounced hyperaemia and venous engorgement during the convulsive movements, according to the mean of the recorded observations. Reference might also be made to the apparent association of fits with disorder of the cerebral circulation in certain cases of Raynaud's disease, of which a number have now been reported (see a paper by H. J. Norman¹⁴).

While it would be unwise to ignore the cumulative effect of these differing lines of evidence, I do not think, after having given much consideration to the problem, that they can fairly be taken as showing anything more than that changes in the cerebral circulation accompany, in all probability, the march of events in an epileptic discharge. In my opinion Russell is justified in concluding that, even if vascular disorder is causal, it only carries the pathology of epilepsy one stage further. But I am not convinced that the association constitutes a causal relationship. The cerebral and general circulation is under the control of a vasomotor centre in the medulla oblongata; alterations in the whole cerebral vasomotor apparatus may be caused by dysfunction of that centre. Sudden loss of consciousness may not be of vascular origin, though accompanied by vascular change; the same pathological stimulus may act both on the bulbar vasomotor centre and on cortical neural elements themselves. That this would appear to be the case in respect of the unconsciousness following trauma is strongly suggested by the recent experiments of Knauer and Enderlen¹⁵; and although the analogy with epilepsy is remote, it is well not to lose sight of the possibility of the circulatory phenomena of the fit—as observed when the cortex is exposed—being no more than a sequel to a central neural disturbance of function at the level of the medulla.

If, however, we are sufficiently convinced by the evidence sketched in outline above, we should still be at the stage of pure speculation in regard to the question of how transient cerebral anaemia—in some cases only, not in all—causes neural mechanisms to discharge; we should have to imagine a local cerebral anaemia for the phenomena of Jacksonian epilepsy; and I submit that many cases of local and sudden cerebral anaemia, as in monoplegias of embolic or thrombotic origin, are unaccompanied by any discharging phenomena whatever. Nor is the solution of the problem facilitated by ascribing the motor discharges to anoxaemia. Imperfect oxygenation recurring periodically, locally, and seemingly vanishing after an attack—how is such a process to be imagined? And how will any such theory account for the aura of a fit, which, as has been stressed above, unquestionably appears to partake of the nature of a normal, orderly, regularized, non-excessive function of a given sensory mechanism? Is neural activity accompanied by anaemia in the cerebrum of the normal individual?

The truth is that the gulf between the vascular and the neural is almost as impassable as that between the physical and the psychical. In this lecture, intentionally, no allusion has been made to clinical varieties of epilepsy of an unusual kind; if, however, the phenomena of so-called "epilepsia partialis continua" are considered, they may justifiably be used as an argument for the improbability of "discharging lesions" being based on persistence of an abnormal vascular state; they appear to resemble much more these of continuous excitation of a reflex neural arc by a persisting neural stimulus.

It seems to me reasonable to suppose that the crescendo character of the symptoms of the epileptic fit is due to

direct action of a neural system on collateral and lower neural systems; if a vascular factor is involved, I have tried to show the possibility of it itself being of neural origin. In some instances this may have a bulbar locus; in others there is as clearly a transcortical commencement for the attack. We come back, therefore, to the view indicated at the outset: any constellation of nerve cells in the neuraxis may become unstable; hence physiological varieties of "fit" are to be expected. It is evidently more correct, clinically and physiologically, to speak of "the epilepsies" than of "epilepsy," and this fact by itself should lead us to avoid giving our patients the impression that once a diagnosis of epilepsy is made it is tantamount to pronouncing their doom. On the contrary, increasing latitude in our conception of the condition should lead to modification of therapeutic pessimism.

REFERENCES.

- ¹ Jackson, J. Hughlings: Lumleian Lectures, *BRITISH MEDICAL JOURNAL*, 1890, i.
- ² Wilson, Kinnier: Croonian Lectures, *Lancet*, 1925, ii.
- ³ Cobb, S., and Uyematsu: *Arch. of Neurol. and Psychiat.*, 1922, vii, 660.
- ⁴ Pike, F. H., and Elsberg, C. A.: *Amer. Journ. Physiol.*, 1925, lxxii, 337.
- ⁵ Cobb, S., and Macdonald, M. E.: *Journ. of Neurol. and Psychopath.*, 1923, iv, 228.
- ⁶ Wilson, Kinnier: *Brain*, 1920, xliii, 220.
- ⁷ Wilson, Kinnier: *Trans. Ophthalmol. Soc.*, 1921, xli, 116.
- ⁸ Russell, A. E.: Goulstonian Lectures, *Clin. Journ.*, September, 1909.
- ⁹ Jackson, J. Hughlings: *Trans. St. Andrews Med. Grad. Assoc.*, 1870, iii.
- ¹⁰ Horsley, Victor: *BRITISH MEDICAL JOURNAL*, 1892, i, 693.
- ¹¹ Kennedy and Hartwell: *Arch. of Neurol. and Psychiat.*, 1923, ix, 571.
- ¹² Leriche: *Presse méd.*, 1920, xxviii, 645.
- ¹³ Horrax: Cited by Cobb and Macdonald (see Ref. 5).
- ¹⁴ Norman, H. J.: *Journ. of Ment. Sci.*, 1916, lxi.
- ¹⁵ Knauer and Enderlen: *Journ. f. Psychol. u Neurol.*, 1922, xxix, 1.

DIETETIC CONDITIONS WHICH INFLUENCE THE CALCIUM CONTENT OF SALIVA:

THE POSSIBLE SIGNIFICANCE OF THESE FACTS IN TUBERCULOSIS.

BY

C. LEE PATTISON, M.B., B.S.LOND., M.R.C.S.,

MEDICAL SUPERINTENDENT, KING EDWARD VII HOSPITAL, SHEFFIELD.

Introduction.

THE study of the etiology of dental caries and other defects of the teeth has usually centred around the chemical, physical, and bacteriological condition of the mouth, and, among other factors, saliva has been considered of importance in this connexion. However, in spite of the large amount of work by Miller, Röse, Pickerill, Marshall, Howe, Bunting, and many others, no relationship has ever been proved between the alkalinity of the saliva and the immunity to caries, as those upholding the acid-decalcification theory of caries might have hoped to establish.

Less work has been done on the calcium content of saliva, yet it is likely to be important. Among other possibilities it may hinder the removal of calcium from the teeth on the production of acid, or it may help in the hardening of the enamel after eruption, if this takes place, or it may aid in the arrest of caries.

The experimental work of May Mellanby¹ has proved that the structure of dogs' teeth, and probably also of human teeth, can be controlled by diet and environment. Foods rich in fat-soluble vitamin (for example, cod-liver oil, egg-yolk, milk, and most animal fats) tend to help the formation of well calcified, regularly arranged teeth, while other foods (for example, cereals, and especially oatmeal) tend to cause the development of badly formed teeth. Thus the total effect of a mixed diet is the outcome of a battle between these different dietetic influences.

On the basis of these facts Mrs. Mellanby and I started, three years ago, a clinical investigation to determine whether there is any relation between diet of the above-mentioned type and dental caries. Some evidence was obtained that a diet rich in fat-soluble vitamin not overburdened with cereal tends to prevent the onset and spread of dental caries more rapidly than does one containing little fat-soluble vitamin and more oatmeal and other cereal. These findings were published in a preliminary note (together with J. W. Proud, L.D.S.) in the *BRITISH MEDICAL JOURNAL* of August 30th, 1924.²