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MODERN VIEWS ON THE CLASSIFICATION OF EPILEPSY*

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In the twenty years since the electroencephalogram (E.E.G.) began to illuminate some of the many dark corners of epilepsy people have tried to discard the old name with its dependent prejudices and misconceptions. They have tried to limit its meaning, and they have tried to make it fit new facts.

To some, "epileptic fit" is tautological, since every fit must be epileptic, but there are others who exclude symptomatic fits, who refer to "genuine epilepsy," or who limit the use of the word to recurrent fits without apparent cause, making epilepsy synonymous with "constitutional epilepsy" or "genetic epilepsy." A fit can be induced in everyone by chemical or physical agents, and a fit may complicate many of the physical upsets usually experienced in living—teething, measles, menstruation, acute alcoholic excess, hydration, exhaustion, fevers, toxæmias, and so on. It is therefore foolish to equate epilepsy with convulsions and to call a single convulsion an epileptic fit. And yet we know from clinical and electroencephalographic evidence that there is no difference in the nature of a convulsion, however induced and however predisposed to it the subject is.

Scientifically all fits are epileptic, and yet all who have fits are not epileptics. There is a close direct relationship between the chance of precipitating a convulsion and the individual's innate tendency to convulse. The kind of brain injured is as important as the kind of brain injury. Thus the diagnosis of epilepsy rests upon a degree of susceptibility, and not upon the nature of the attack alone. It is a relative diagnosis. A patient is said to have epilepsy when the attacks recur without a demonstrable independent cause. Conversely, a patient who has fits in response to a known recurring cause has not epilepsy; he may have hypoglycaemia with convulsions, uraemia with convulsions, or syncope with convulsions. When a faint caused by a fall in blood pressure ends in a fit the diagnosis must be syncope, however often the event happens, not epilepsy.

How shall Epilepsy be Defined?

There must be no confusion of meaning or transference of an old word to new phenomena. The word "epilepsy" refers to a state experienced by the patient, and not to electroencephalographic events, although the E.E.G. has widened the range of changes recognized as being epileptic. It is probably wise to keep the use of the word a wide one, and qualify it with an adjective when necessary. With our present limited knowledge epilepsy can be assumed when there are recurring discrete disorders of sensation or behaviour of any sort caused primarily by cerebral disturbances. This use of the word excludes the isolated attack and all attacks caused by disorder elsewhere in the body, but

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it takes the use of the word, probably justifiably, to the doorstep of clinical psychiatry. Here it has the advantage that it avoids the use of such uncertain terms as "epileptoid" or "epileptic equivalent."

Epilepsy has been recognized as having a broad spectrum since the middle of the last century, and Jackson was followed by Wilson in referring to "the epilepsies" to make this point clear. The forms of epilepsy can be recognized in the patients' experiences, in observation of the patients' attacks, in studying their electrical counterpart in the E.E.G., or in a combination of all these sources of information. The history of this aspect of the subject has often been reviewed, and is stated shortly and clearly by Penfield and Jasper (1954).

Classification in the Past

Four main forms of epilepsy used to be recognized: (1) grand mal, the tonic-clonic major fit; (2) petit mal; (3) focal attacks; and (4) "epileptic equivalents" or psychic fits. There were numerous subdivisions, and also uncommon or ill-defined forms of attack which simply could not be fitted into a short classification, such as epilepsy partialis continuans, striatal epilepsy, and reflex epilepsy. There is still no classification of epilepsy to fit all the facts or to suit all observers.

Why Classify?

Epilepsy is a symptom with many forms and as many causes. It reflects disturbance of brain function, and has the same order of meaning as madness. It has come to cover so many causes of first, second, and third degree—predisposing and precipitating, physical and psychological, acquired and innate—and to include so many evidences that it has lost precise meaning and must be qualified. It even suffers from the handicap of the brain-mind dilemma now. Classification of its forms is essential for the correct use of language and understanding of it, since any substitute for it will suffer from the same handicaps; but classification is also essential to the proper handling of causes, prevention, treatment, and social management.

Kinds of Classification

Any classification must be useful, and should therefore reflect the needs of its user, and there can be many coexistent useful classifications of epilepsy each reflecting an aspect of the subject. Quite useful classifications of the state of epilepsy can be based on the degree of disability; the form of disturbance seen or felt; its first causes; its precipitating causes; its associated disturbances, whether physical or psychological; its physiological correlations, whether physical or chemical; its anatomical bases; its temporal qualities; the kind of epileptic subject; or its social-economic status. They will all be valid, they will all overlap, each will be a compromise, and though all will cause argument each will have a use to someone. What is important is that we should no longer consider epilepsy a homogeneous, or self-contained, subject. Its boundaries cannot all be mapped nor its contours traced.

Present Classification

We are here concerned with a classification which will form a basis for discussion of the anatomy and physiology of epilepsy, so that we can inquire into causes and cures. The classification we use must rest on the basic sciences, and, like all the applications of basic sciences to medicine, it will thereby lose definition and exact meaning. Such a classification is based on facts observed in the attack—facts seen with the naked eye, and through the microscope in the laboratory, the E.E.G.; “bedside” and “scientific”; clinico-physiological. All these words are as meaningless as epilepsy.

If the classification is to be based on events in the attack we must be careful to distinguish between the pre-ictal, ictal, and post-ictal events. Most post-ictal events are caused by confusion in the patient, the result of the attack, and pre-ictal events may be related to the cause and not to the attack itself. This distinction was repeatedly emphasized in the writings of Hughlings Jackson and William Gowers, which form the basis of all discussions of knowledge in this subject. The most recent and important classifications are those of Penfield and his co-workers (Penfield and Kristiansen, 1951; Penfield and Jasper, 1954) and of Gastaut (1954). They have many points of similarity to classifications by others, notably Gibbs and Gibbs (1941), who mainly used electroencephalographic data, and Symonds (1954), who was mainly concerned with clinical.

In tabulating the events which can occur in an epileptic experience we must remember that they “may include an example or a distortion of every feeling or movement of which the patient is capable, in an infinite number of variations and combinations” (Williams, 1956). There is no sharp distinction between simple events and elaborate events, or even between physical and psychical. A classification of these experiences which was published by me recently (Williams, 1956) follows:

Forms of Ictal Experience**SENSORY EXPERIENCES****Perceptual Illusions and Hallucinations**

Special Sensory—light, sound, smell, taste.

Common Sensory—tingling, pain, or loss of feeling.

Conceptual Illusions and Hallucinations.**Proprioceptive****Body concept:**

Partial: Limb: e.g., alteration in size or shape; a supernumerary limb; disturbed positions; disordered relation to whole—i.e., “different,” “unreal,” “sinister”; unawareness.

Total: e.g., depersonalization; detachment of self; autoscopia.

Exteroceptive

Partial: Visual: e.g., Lilliputian, objects receding; space huge or constricted; objects distorted; objects too vivid.

Auditory: e.g., sounds distorted; far away; unreal or impersonal; having personal reference.

Total: Derealization; “dreamy states.”

Mixed Proprioceptive and Exteroceptive (Total Conceptual Disturbances)

E.g., disorientations; derealization with depersonalization; “dreamy states” which include the person; bizarre examples—e.g., “I feel the room is vast and I am in the top left-hand corner.”

Temporal Disturbances

Disturbances of “subjective” time—e.g., time seems to stand still; time rushes by.

Disturbances of “objective” time—e.g., people seem to move slowly, to rush along, or “my thoughts rush by.”

Perceptual distortions—e.g., familiarity, *déjà vu*, *jamais vu*.

MOTOR ACTIVITY**Somatic (Skeletal)**

Incoordinate { Focal: muscle twitch.
General: tonic-clonic.

Co-ordinate

Partial—e.g., licking, laughing, sucking, grimacing.
General—quasi-purposive—e.g., running, striking.
Purposive—e.g., aggressive organized outbursts with inadequate cause.

Visceral

E.g., vasomotor, sudomotor, piloerection, cardio-respiratory, alimentary (belching, borborygmi).

EPILEPTIC HALLUCINATIONS**Special Sensation****Unitary:**

Visual: unformed or formed; stationary or animate; natural or bizarre; coloured or black-and-white; total or against normal perception.

Auditory: unformed sounds, music, words, or words with personal reference.

Olfactory: unformed.

Gustatory: unformed.

Complex:

Special sensory—e.g., visual hallucinations that speak. Multisensorial hallucinations.

Special sensory with perceptual change—e.g., “a feeling of blue balloons in the left leg.”

Special sensory with total conceptual change—e.g., living an hallucinatory experience in a dreamy state.

Common sensation—e.g., an extra limb.

Total sensation with vision and hearing—e.g., autoscopia.

Emotion. Mood or feeling tone; fear, depression, pleasure, unpleasure, anger—in various settings, degrees, and descriptions.

Memory. Always, and only as a pattern for an hallucination falling into one of the previous categories.

Thought. “Spontaneous” cognition did not occur alone in any case, though secondary cognition based on the hallucination is described. “Forced thinking” should be reconsidered.

The first simplification is to divide epileptic attacks into general and partial or focal.

General

There are two easily recognized distinct forms of the general epilepsies in which there is a bilateral synchronous disturbance to be seen in the E.E.G. from both hemispheres: *major epilepsy*—the tonic-clonic fit of grand mal, which is stereotyped, self-limiting, and needs no description; and *minor epilepsy* (petit mal)—the frequently repeated interruption of consciousness of childhood in which there is no motor component more definite than blinking, blankness, and pallor.

The features common to these general epilepsies are that consciousness is lowered or lost and that disturbances of movement when they are present, as in a major fit, affect the body diffusely and symmetrically. The E.E.G. shows changes, such as the fast spikes of grand mal and the spike-and-wave discharges of petit mal, which have the same general characteristics, that they are generalized, and are symmetrical and synchronous in the hemispheres. There is increasing evidence, which is outside my terms of reference, that the disturbances responsible for these generalized attacks affect primarily deep midline structures which maintain and regulate consciousness. Stimulation of, and damage to, these structures in the mid-brain alters the conscious state of behaviour, and also alter the electrical activity of the whole of both hemispheres. We do not know why the intense fast electrical activity of grand mal is associated with intense motor activity, or why the high-voltage spike-and-wave discharge of petit mal accompanies lowering of consciousness without movements. Also we do not know why myoclonic jerks without loss of consciousness have the same electroencephalographic accompaniment as petit mal with its loss of consciousness but without jerking.

In all respects—in their clinical form, the electroencephalographic disturbances, and their biochemical and pharmacological responses—grand mal and petit mal are totally different, and yet they often occur in the same subject, both are features of genetic epilepsy, and both involve disturbance of deep midline structures.

Focal or Partial Epilepsies

The E.E.G. has taught us a great deal about these forms of epilepsy. Deductions on the site of origin based on clinical observation were confirmed and methods of spread over the cortex have been studied, but there are two ways in which we have gained original knowledge—the recognition of foci of discharge in the so-called silent areas of the frontal and temporal lobes, and the bilateral occurrence of so many focal discharges. We used to believe that major fits were of two kinds—those with a generalized onset and those with a focal onset. It is now evident that when a major fit begins with, say, twitching or tingling of a limb, what in fact has happened is that a focal fit has initiated a generalized convulsion—there have been two attacks, the focal fit having initiated the major fit much as electrical stimulation might. This fact, which is so evident in electroencephalographic records, was really evident from observation of the attacks too, because a major fit always has the tonic phase first, the clonus being caused by interruption of tonus, while a clonic focal fit consists of much more rapid jerks of part of the body, and the march of events is clonus, tonus, and then clonus—obviously two different kinds of fit succeeding each other.

Another fundamental fact that must be recognized to achieve a logical understanding of focal fits is this: that, however elaborate may be the movements or feelings in an epileptic attack in which consciousness is preserved, they are always due to focal disturbance of brain function. The only difference between an attack with a primitive muscle twitch and one with co-ordinated, apparently purposive disorder of social behaviour, is that the first is caused by disturbance of function in the relatively primitive motor cortex, and the second in a more highly elaborated integrative part of the cortex. Those attacks we most readily think of as “focal”—the clonus of a limb, local tingling or numbness, flashing light, a smell, a buzzing sound, and so on—all happen in the first cell station of reception of sensations into the brain or the last cell station of emergence of movement. They are simple phenomena, which were therefore understood early in the study of epilepsy. The “psychic fits” or psychomotor attacks, the elaborate hallucinations, dreamy states, emotional experiences, or visceral disorders of epilepsy are all just as truly caused by focal discharge in the brain as the experimental surgery of Penfield and others has demonstrated. That being so, focal fits can be classified according to their clinical pattern—motor, sensory, psychic, and so on—upon the basis of their known origin. The second is probably the more useful, because, as the list of epileptic events shown above indicates, there are too many forms of epileptic experience to make their division a profitable one. A classification based on the part of the brain first affected, with a statement of the clinical events, is acceptable in all countries.

Focal fits may be cortical or subcortical, but much of the cortex, like that of the island of Reil and the undersurface of the temporal lobe, is buried.

Cortical discharges may occur in *primitive areas* or *association areas*, or may spread from one to the other. They may, as so often in the temporal lobe, be *bilateral* and they may march over the cortex in a *Jacksonian* way. We now recognize that that Jacksonian march may include very complex experiences in which division into physical and psychological becomes meaningless.

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VALUE OF THE E.E.G. IN DIAGNOSIS OF EPILEPSY*

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The value of clinical electroencephalography in the diagnosis of epilepsy depends on the accuracy of the technique in throwing light on two issues: (1) From which area or from which neuronal system does the epilepsy originate, and is there more than one such focus or system from which it originates? and (2) What is the nature of the pathological process, if one is present, of which the epilepsy is a symptom? Diagnosis is therefore both topographic and aetiological. Considerable advances have been made in the last few years in respect of the first issue, and careful repeated E.E.G. investigations planned with knowledge of the clinical problem involved will in most cases answer the question, From where in the brain does the epilepsy arise? Very often, of course, such investigations merely confirm the clinicians' expectations, demonstrating what could be anticipated from a knowledge of the aura and fit pattern which the patient exhibits. Only if neurosurgery is contemplated are such intensive investigations vitally necessary. With regard to the second issue, which is of such great practical importance, the E.E.G. technique is of more limited value in helping us to decide the nature of the pathological process of which the epilepsy is a symptom. Here again, however, some advance has been made. These two issues are the subject of this paper.

Improper Use of the E.E.G.

The E.E.G. is often used as a means of helping to decide whether a given patient's symptoms are epileptic or psychogenic or are due to some non-cerebral cause, such as syncope. I believe this is an improper use of the technique and that in fact the results of doing this can be misleading. A single routine normal E.E.G. is not evidence against epilepsy, nor indeed is the finding of epileptic discharges in the E.E.G. evidence that the patient's symptoms are necessarily epileptic in nature. Such discharges occur in the E.E.G.s of some non-epileptic persons, in many with degenerative brain disorders, in some psychotics, and in cases of behaviour disorder. The prescription of anti-convulsants on the finding of such discharges in the E.E.G. is unjustifiable and often illogical, and may lead the clinician to overlook more important issues for his patient.

The recognition of the epileptic nature of any symptom or group of symptoms is, I believe, a clinical task and must remain one. When the clinician requests an E.E.G. to confirm the diagnosis he is not so much interested in the question of whether the E.E.G. demonstrates focal discharge in the expected area—he is merely disappointed but does not alter his diagnosis if it does not—as in the question of whether the E.E.G. demonstrates the presence of a pathological process, either local lesion or a diffuse one, which is of such importance for the prognosis. However, when the question of surgical removal of a focal epileptogenic area arises both these questions are of equal importance.

Classification of E.E.G. Characters

Some 20 years ago Gibbs, Gibbs, and Lennox (1938) described the first classification of the E.E.G. characters to be found in epileptics. Their categories were described in terms of frequency-voltage patterns of discharge—the actual appearances of the rhythm—and they paid no attention to

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