EPILEPTIC VARIANTS.

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In the course of twenty years' hospital and private practice there have come under notice a large number of cases which present intimate or remote similarities to the phenomena of epilepsy as usually understood, but which nevertheless do not seem to resemble each other, at least not superficially. Unfortunately, in many instances one has to rely on the description furnished by either the patient or his friends (there is no reason, of course, to doubt these descriptions are veridical), since development of the symptoms concerned during the period of consultation and examination can scarcely be expected, helpful though it would prove. Close study of these conditions, which may conveniently be termed epileptic variants, is particularly fruitful in dispelling from the mind any idea of epilepsy as a self-contained and sharply delimited clinical entity. I pick up a recent hook on neurology for practitioners and therein I find the chapter on epilepsy opens with the dictum: "Epilepsy is a dreadful disease." Five words can hardly contain more than a similar number of errors; these five unquestionably embody two, for epilepsy cannot be regarded as a disease, and experience teaches that the majority of cases are certainly benign. We have laboured for years under the unenlightening procedure of elevating symptoms into diseases, giving them names, and forcing new clinical instances into old nosological conceptions. This has been notoriously the case with epilepsy: so stereotyped is the idea in the minds of many that it is a sinister "disease" that to any given case of "fits" this conception is forthwith transferred—in the absence, that is to say, of accompanying symptoms which may point otherwise. Against this mental habit a protest must be raised; one of the best ways of counteracting its harmfulness is by the consideration of epileptic variants.

At the outset it is, perhaps, impracticable to dispense with some kind of exposition of what should be conveyed by the term "epilepsy." In a recent Harveian Lecture,¹ as well as in a paper² read before the Assurance Medical Society I have explained at some length the views commending themselves to me in respect of epilepsy and its connotation. Without here repeating general conclusions therein adopted, I should like to restate in a somewhat different form what seems to me the appropriate way of facing the problem.

The clinical phenomena with which we are concerned must be considered physiologically: they represent escape from physiological inhibitions. Whether they indicate only "release," or also "irritation," is immaterial or at least

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¹ British Medica Association Lecture, delivered at a meeting of the Swansea Division, December 8th, 1927.
of secondary importance; if an irritative process is to exteriorise itself it must succeed in overcoming inhibition. Escape of function is no preserve of motor mechanisms, as when the limbs of the patient with spastic paraplegia exhibit involuntary flexor spasms, or when the limbs of the epileptic are convulsed; or as, indeed, may conceivably be the case with movements thought to be "voluntary," since even for the manifestation of these release from transcortical inhibition may be an essential preliminary. In many epileptic phenomena symptoms of another order are apparent—the aura, if present, is a sensation, therefore a psychical thing, a change in consciousness; sensory systems can undergo release, as in sensory Jacksonian epilepsy (in some varieties sensory manifestations may constitute almost the whole fit). Once the process is initiated, it may, or may not, spread: mechanisms, motor or sensory, or both, on the same level, or on lower or higher, may in turn submit and their resistance be overcome, until in a brief period all are affected, and generalisation is then said to take place. Alternatively, the process may be strictly limited, both on motor and on sensory side; the aura, occurring by itself, may constitute the sole symptom and form a larval fit. Again, loss of consciousness, as in numerous instances of le petit mal, can develop almost monosymptomatically. Evidently, then, epileptic possibilities are legion; no rigid semiological framework can be fashioned to embrace them all. This becomes still more apparent if we include, as I think we legitimately may, analogous phenomena consequent on excitation or inhibitory release of visceromotor and viscero-sensory centres, e.g., those of the medulla. Their clinical types may seem far removed from epilepsy as ordinarily understood, but they can be considered none the less as belonging to the epileptic category.

Frontal, rolandic, parietal, occipital, temporal epileptic syndromes might be distinguished; also mesencephalic, pontine, bulbar, cerebellar, and, it may be, spinal varieties. And cannot similar physiological processes conceivably affect visceral neural plexuses and ganglionic collections? Is "epilepsy" say of the solar plexus an absurdity? I am far from thinking so.

Common sense compels us, if we appropriate this wide conception of the condition, to speak of "the epilepsies," and to disabuse our minds of the idea that one and all must always display a somewhat forbidding aspect. Doubtless in many instances we discover at a glance that the epilepsy is symptomatic, as when fits develop during an attack of cerebral haemorrhage, in pronounced toxicosis, under conditions of intracranial pressure or with perceptible vascular disease: this fact alone should have taught us to see in all epileptic manifestations the action of mechanisms irritated or released into independent function, and in so-called idiopathic (that is, unknown) epilepsy our search should still be directed towards discovery of the underlying cause, which must exist.

At this point it is perhaps natural to interpose with the objection that the epilepsies would thus be made synonymous with or equivalent to other orders of phenomena with which they do not appear to have much in common. It
is not expedient clinically that they should be classed with tics, for example, or with reflex movements and spinal automatisms, or with hallucinations, and so on; nevertheless, the aura of an epileptic fit is a hallucination, one or other of its movement-complexes can resemble flexor or extensor spasmodic automatisms, and minor, repetitive varieties are often separable from tic only on prolonged observation. An epileptic head-nodding or drooping movement in children is extremely like a salaam tic. What are to be the criteria whereby differentiation is effected?

In its crudeness, violence, complexity, disorderliness, the major fit can scarcely be confused with any other syndrome, yet parts of it are neither disorderly, violent, nor crude: the aura shows none of these qualities, and not a few of the movements are quite "ordinary," i.e., coordinated, and in no sense convulsive. As for Jacksonian epilepsy, its motor display is largely sui generis in respect of both the character of the movements and their march, but slight, localised twitching cannot readily be distinguished from some kinds of myoclonus or of tic. Petit mal is recognised with ease because of its intrinsic features in perhaps a majority of instances, yet every practitioner knows what uncertainty surrounds the exact diagnosis in others. To establish standards of universal applicability even in regard to the main classes of epilepsy thus becomes almost a Sisyphean task. All of us may think we know a fit when we see it, but we should have great difficulty in endeavouring to specify the criteria which we as clinicians are using. Features such as recurrence, transient nature, interference with the stream of consciousness, are in no way pathognomonic. though they are probably foremost in the mind whenever we think of "attacks" or "fits" of any sort. The phenomena of the epilepsies edge off by easy gradations from the motor convulsive seizure to psychical, sensory, or visceral symptoms of a highly disparate kind, and at the same time refuse to be separated for any physiological reason. Clinical conceptions ought to be based on physiological unities, and if the same process can rationally be supposed to be in action behind these diverse clinical syndromes they belong to the group. There is no class of symptom in neurology that has suffered more from nosological artificialities than that still called epileptic.

I shall take first the clinical types that are characterised by variations in the motor phenomena, and submit the following classification as possessing clinical usefulness.

I. MOTOR VARIANTS.

1. Myoclonic or regional epilepsy.
2. Epilepsia partialis continua.
3. Tonic epilepsy.
4. Coordinated epilepsy.
5. Inhibitory or akinetic epilepsy.
Myoclonic or Regional Epilepsy.—This particular variant is far more common than is usually imagined. It takes the shape of irregular twitches of a limb in multimuscular groups, and in colloquial parlance is often termed "the jumps." "I had the jumps this morning" is a habitual remark. The occurrence of such jerking movements without loss of consciousness is as distinctive as it is little known. Not much complained of spontaneously, careful inquiry elicits a history of their existence often for a longer or shorter time before the first paroxysmal fit. Indeed, in some instances they form the sole epileptic indication, as in the following case.

CASE 1. V. S., female, age 18. Since the age of 14 has suffered practically every morning from more or less violent sudden jerks of the arms, sufficient to throw things out of her hands. Affecting both arms indifferently but not synchronously, these jerks make her knock things off the breakfast table or toss a comb or hairbrush across the room. The legs are unaffected. She has never had a fit in her life, whereas one brother and two sisters suffered from convulsions in infancy, and another brother has had fits since the age of three.

Examination reveals an intact nervous system.

Either unilateral or bilateral, they affect some definite region of the body: sudden adduction or extension of one or both arms, and sudden flexion or extension of the trunk, are the commonest movements. A statistical study by Muskens has established their presence at one or other period in no fewer than 103 out of 150 female, and 82 out of 150 male epileptics. A textbook commonplace is to the effect that the familial myoclonus-epilepsy of Unverricht is a great rarity, and the statement is repeated in recent communications (cf. Crouzon and Bouttier), yet my experience does not bear out the contention.

The following example is characteristic:

CASE 2. P. P., female, age 15. Has suffered from major fits at long intervals, having had only four in two years. In addition, she shows typical myoclonic jerks of arms and legs in the mornings, throwing things out of her hands or falling suddenly to the floor without a trace of unconsciousness. Examination is negative. A sister also is epileptic, and has myoclonus.

In view of the curious indifference of the average patient to this abnormal condition until or unless major fits develop its early diagnostic significance ought to be impressed on the practitioner of medicine.

Epilepsia partialis continua.—A second motor variant, doubtless somewhat rare, was described originally by Koshevnikow, as long ago as 1894, and has received but scant attention notwithstanding its physiological interest. It differs from the general myoclonic type in that the twitching is limited to one segment of the body, nearly always a peripheral part such as the wrist and fingers, is practically continuous between the paroxysmal fits, and on the whole partakes of the form less of movements than of irregular, individual, muscular contractions. Among those who have reported cases are Orlowski, Chorosko (who suggested the term "polyclonia epileptoides continua"), Spiller, and Bruns. I have seen several typical cases, of which one is here briefly related.
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Case 3.  E. W., male, age 30.  Has suffered from epilepsy since childhood, having on an average several fits every week. For about 15 years he has complained of irregular twitches of the right hand and fingers, more or less continuous. They are jerky and abrupt, producing usually flexion but sometimes also extension of the fingers, and either flexion or extension of the wrist. Quite distinct from tremor, they are equally separable from both chorea and athetosis mainly because of their twitching character; in this respect they are classifiable with the myoclonias. The major fits begin in this right hand, becoming generalised with great rapidity.

Of poor mentality, the patient shows on examination no signs of organic nervous disease.

In this instance no objective evidence points to a local lesion of organic or structural type in the left rolandic area: Spiller’s patient, however, was a syphilitic. One of the other cases I have seen was under the care of a colleague who considered exploration justifiable in view of the localised phenomena, and suspected a cortical or subcortical tumour. At the operation (at which I was present) no abnormal cortical condition was found. The interest of these peculiar cases consists in the long duration of the twitching, as though due to persistent stimulation of a cortical reflex arc, and in the absence of any trace of that exhaustion paresis or paralysis which is remarked with some frequency in Jacksonian epilepsy.

Tonic Epilepsy.—Fits characterised by tonic contractions only, to the exclusion of the clonic element, have long been known. Of their semiology and pathogenesis full discussion was given in a paper on decerebrate rigidity published some years ago. It was there argued that many so-called cerebellar fits belong to this tonic group, whose physiological localisation is in posture-effecting mechanisms of mesencephalic site (mainly). The cortical element in an ordinary fit is supposed to be phasic, that is, clonic, although certain investigations (Pike and Elsberg) have demonstrated the occurrence of both clonic and tonic convulsions in decorticate (motor cortex) animals, provided an interval allowing for the return of locomotor reactions elapsed after the initial operation. From the clinical standpoint, fits distinguished by the assumption of tonic attitudes and the occurrence of tonic muscular contractions are most likely to develop in consequence of lesions dissociating physiologically the cerebral hemispheres from the cerebello-mesencephalopontine levels, and the reader is referred to the article mentioned above for a variety of clinico-pathological instances. But brief tonic fits, in some cases not much more than a rather elaborate petit mal seizure, also occur and deserve mention. The following is a recent example.

Case 4.  N. S., female, age 21.  When younger, used to have typical myoclonic jerks in the mornings. For the last 18 months, has had fits about once a week. These attacks, repeatedly observed, invariably exhibit the following features: ushering them with a cry, the patient throws her head back and falls backward with a crash; trunk and limb musculatures are seized in rigid tonic contraction, arms flexed, legs extended; cyanosis develops while the tonic contraction is maintained for many seconds. No clonic movement has ever been seen. Relaxation then sets in and consciousness returns.

A few days ago, her head turning as she fell, she broke the bones of her nose and sustained severe ecchymoses of both orbits.

One of her father’s brothers was an epileptic lunatic, and another committed suicide. Examination furnishes no evidence of organic subtentorial disease.
In all cases of this kind it is perhaps advisable to suspect an organic and localisable basis for the affection, though doubtless such will not always be discoverable.

**Coordinated Epilepsy.**—By this term—possibly not the most appropriate that might be devised—the intention is to denote those cases where the movements seen during the attack are coordinated and seemingly purposive, exhibiting objectively to a large extent the features of "voluntary" movements. They may however be aimlessly repeated and in any case do not attain the end to which they may be supposed to be directed.

It is of prime importance to recognise, as I have pointed out elsewhere, that in many epileptic fits quasi-volitional movements occur along with and separate from those called convulsive. "The epileptic patient may, during the fit, make champing movements of the jaws, 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, trying to undress, etc." Most of us must be familiar with post-epileptic confusional states during which all sorts of automatismas of a complex kind are performed without his conscious participation therein. Similar coordinated movement may initiate the attack; I remember a case of left frontal abscess where the patient waved his right arm in circles—as though turning the handle of a barrel-organ—for several seconds ere he fell unconscious and convulsed.

In still other instances the content of the fit is not convolution but coordination of movement, as in the subjoined case.

**Case 5.** C. S., male, age 30, has suffered from fits for two years. His attacks occur without warning; he has had one when riding a motor cycle. In them he turns extremely pale and is unconscious of everything, but never falls; he raises his closed fist and blinks with his eyelids, then looks sideways as if to see round a corner. The fit is over in about a minute. He never knows that he has had one, till his wife says, "you've just been having one of your funny do's." The nervous system is organically intact.

A maternal uncle suffered from major epilepsy, also a cousin.

As is known, the motor phenomena of pet. t. ma. not infrequently are coordinated rather than convulsive or myoclonie.

Thus (1) **before, during, or after** the phase of actual convolution or contention of movements individuality of movement may still be preserved; and (2) occasionally only movements of coordination are seen. To me this is a feature of the epileptic seizure that is of profound significance. For it surely helps to break down any artificial semiological barrier between "functional" and other kinds of fit, and indicates differences of degree only, not of quality, between hystero-epilepsy and other epilepsies. Cortical motor centres of the highest level (higher than the rolandic or middle level of Hughlings Jackson) must be implicated if quasi-purposive movements characterise a given fit. Clinically speaking, no arbitrary distinction can be drawn between the elaborate motor phenomena of a hysterical fit and those of the post-epileptic state; both alike represent subconscious or not fully conscious activities released from transcortical inhibition.
These considerations lead to the interesting speculation of the relation of the time factor to the particular guise assumed by epileptic disorders of motility. If the motor elements of a convulsive fit could be extended or lengthened in time, would they not possibly show more coordination than they do? Is not the *swiftness* of the average major fit responsible for the seeming contention of movement, which results in the "single big useless movement," as Jackson termed it, of the tonic stage? What would happen if it were spaced out a little? The clonic movements, "which do nothing but 'mark time,'" exhibit more than a trace of coordination. Were the fit more 'drawn out,' from the standpoint of motion, its features might be so different as to lead to the supposition they were hysterical in nature. The late Sir William Gowers used the apt illustration, in this connexion, of how velocity alters the effect of momentum. "A bullet fired from a rifle makes a round hole in a pane of glass, which it would smash if thrown against it." Variability in length or duration of aura and of post-seizure state of automatism is determined by factors of which we have little or no knowledge, and is accountable for phenomena so diverse as to render individual fits hardly comparable one with another, yet the differences are non-essential. After the briefest of petit mal attacks so rapid as to be scarcely recognisable as such, I have seen a little boy run many yards to hide in any convenient corner—a performance that would have been branded as hysterical had not its real nature been discovered. This epilepsia procursiva, so-called, differs in no fundamental way from a fugue that lasts for hours, days or weeks.

*Inhibitory or akinetic Epilepsy.*—While the motor syndromes of the epilepsies are ordinarily considered to be essentially kinetic or hyperkinetic, this is in point of fact not universally true. The question arises whether under certain circumstances motionlessness may form a prominent feature of the fit, and it must be answered in the affirmative. Faints and syncopal attacks generally, and some kinds of hysterical fit, are distinguished by relative immobility of the patient; in the case of the former the limb muscles are usually somewhat flaccid and devoid of tone; in the latter less so, since rigidity and motionlessness may occur together. I have had occasion to observe certain attacks which can fairly be taken as coming under the epileptic category, in which similar immobility has been remarked. Years ago both Jackson and Gowers interested themselves in this question: Gowers was of the opinion that discharges in epileptic fits occasionally inhibit, as he found temporary paralysis in some cases after a purely sensory discharge. Jackson referred to cases of epileptiform (Jacksonian) fit in which the patient tells us that his arm "falls dead," there being no spasm in it, whilst the face of the same side is being convulsed. His speculation was that "there may be discharge spreading slowly in a motor centre of the middle level, excessive enough to cause slight after-exhaustion of some of its elements, although one not strong enough to overcome the resistance of lowest motor centres, and thereby to produce actual convulsion."
Brief reference may here be made to a case of uni\text{\textbackslash} lateral fits which was the subject of prolonged investigation and in which I was able to observe the patient repeatedly during the attacks. Its salient feature was the development of complete flaccid palsy of a temporary character after a sensory Jacksonian aura.

\textbf{Case 6.} W. C., age 24. Has suffered for six months from attacks of tingling in the left hand and arm, and the left side of the body, occurring every two or three days, followed by weakness of the limbs corresponding.

The fit is preceded by subjective visual phenomena ("bright flashing stars"), without headache, and commences with a strong sensation of tingling in the left fingers and hand; within a few seconds this passes up the left arm, then down the left side of the body rapidly into the left leg, foot, and toes, also appearing in the left face and left half of the tongue. Variable in duration, from a few seconds to a minute or two, it leaves arm, leg and face "dead," and "heavy as lead." When this numbness appears he loses power in his arm and leg—in a second or two this loss is absolutely complete, and the left limbs immobile. The numbness and powerlessness together endure for anything from two to twenty minutes. Recovery of power is rapid and usually simultaneous in arm and leg. Headache usually follows the attack, as a rule bilateral and temporal.

I made many examinations both during and apart from the fits. No definite change in reflexes and no unequivocal sign of organic disease was discovered in the normal periods; in the course of the attack objective loss of sensibility coexisted with flaccid palsy, and with exaggeration of the deep reflexes and diminution of the abdominal reflex on the left. In more than one attack patellar and ankle clonus developed, and a slight extensor plantar response.

This striking case (published in greater detail in the article that follows this) is clearly linked to those mentioned by Gowers, and is classifiable as inhibitory epilepsy. Some clinicians might perhaps call it a case of migraine, but to do so is merely to change its name; the phenomena themselves are not thereby explained, nor is their epileptiform character thereby invalidated.

The topic, however, has in reality wider limits than may at the first glance be apparent. Attacks of one or other kind in the course of which the patient falls or lies motionless, or becomes incapable for the time of volitional innervation, form a perhaps rather heterogeneous group. Consciousness may or may not be interrupted, slight voluntary movements may be possible, slight involuntary muscular contractions may take place. At this point the question of inhibitory epilepsies merges with the fascinating problems attaching to trance, catalepsy, narcolepsy, and cataplexy. I read a communication on narcolepsy and allied states before the Association of Physicians at their Belfast meeting last June, and this will appear in extenso in the next issue of \textit{Brain}; in it a full discussion of these problems, together with a description of various personally observed cases, will be found. Arguments are therein advanced to support the view that some at least of the narcolepsies and epilepsies are interrelated, and that the cataleptic attack, in which the patient sinks to the ground with paralysed and toneless muscles, offers definite resemblances to some of the phenomena commonly called epileptic. This obviates any need to pursue the question further in this article, yet I may take the opportunity of alluding to one or two points.
To illustrate concomitance or sequence of narcoleptic and epileptic symptoms the following case may be cited.

**Case 7.** A. G., male, age 21. Had some six epileptic fits between the ages of two and seven years, and three or four more between nine and 14. From the description furnished these would appear to have been characteristic major epilepsy. Since 14, has had no more. Recently however, for some months, has developed a different kind of attack, in which he “loses himself,” turns white, may or may not fall, sits or lies still, unable to move, without convulsion, tongue-biting or involuntary sphincter relaxation. These turns are brought on by uproarious laughing; if he laughs less immoderately he gets a “pain at his heart,” without the attack following. No signs of organic nervous disease are discoverable.

Here is a clear transition from convulsive epilepsy to inhibitory epilepsy, and I consider the case to be of high significance. The late manifestations are classifiable as cataplectic, the early as epileptic. It is obviously impracticable and unscientific to endeavour to divorce the two from the standpoint of pathogenesis, and the case furnishes us with a good example of how misleading mere nomenclature can become. Another recent case may be reported.

**Case 8.** G. P., male, age 52. For the last eighteen months has had what he calls “attacks of trance.” The aura is a peculiar smell, always unpleasant and usually likened to that of “burning fat.” This olfactory hallucination is of variable duration, and is followed by his becoming “weak all over” and “falling into a trance” in which he turns white, is unable to move, and as a rule is unaware of his surroundings. After four or five minutes he “comes to himself,” experiencing at the same time a “peculiar feeling” at the root of his nose, which he has difficulty in describing.

In the instance just given we have the same sequence of temporary generalised immobility after a sensory discharge (in this case of the olfactory order), falling in line with those referred to above. It is evident, I think, that the association constitutes no rarity. At the bidding of intrinsic (and as we shall see immediately, extrinsic) stimuli a process of inhibition, not of excitation, is initiated. This is true of numerous cataplexy cases in the narcoleptic category, and also of occasional cases in the epileptic group. Why one physiological process rather than another should be determined cannot easily be explained, and its discussion would take me too far from the subject at present before us.

**II. SENSORY VARIANTS.**

1. Reflex epilepsy.
2. Sensory epilepsy.
3. Affective epilepsy.

*Reflex Epilepsy.*—The term is in usage to signalise cases where an epileptic seizure of one or other kind develops on the heels of an extrinsic sensory excitation of one or other order: formerly known as Brown-Séquard’s epilepsy (that experimentalist having produced fits in guineapigs by excitation of cutaneous “epileptogenous” zones after preliminary injury to the cord), it may or may not be associated with spontaneous fits in a given case. Examples of this type are comparatively scarce, although by now a fair number are on
record. In one published by Woodcock, that of a little boy, the remark made by his sister was: "Whenever we undress him he has a fit." Undressed on one occasion in the observer's presence, as his stocking was taken off his right leg, he went into an epileptic fit of the classical type, clonic convulsions following tonic, and unconsciousness being complete. I have seen a case in which, when the hat-elastic under a little girl's chin slipped up, hitting the nose, she went off in an epileptic fit. This accident led to the discovery that flicking or tapping the nose always started the attack, which consisted of dilatation of the pupils, tonic spasm of arms and chest, respiratory arrest, cyanosis, and slight frothing at the mouth, lasting in all less than half a minute. (This case was also seen by Dr. Foster Kennedy and is cited by him in a paper alluded to below).

Another variety of reflex epilepsy was termed by Oppenheim acoustico-motor epilepsy, and is distinguished by the patient's falling to the ground on hearing a sudden sound. The following is a personally observed example.

CASE 9. J. R., female, age 19, has suffered since the age of seven from "falling attacks." She had been knocked down by a taxi two years previously but sustained no actual physical injury. The slightest unexpected noise causes her to collapse to the ground, without unconsciousness, and apparently without any of the usual concomitants of epilepsy, major or minor. So sudden is the attack that she is unable to save herself and has sustained bruises several times. Up to the time of her coming under my observation she had had no epileptic fit. Somatic examination is negative.

A case of this kind is no doubt abortive or incomplete, but it is paralleled and confirmed by one that I have recently been able to see through the kindness of my colleague, Dr. Gordon Holmes. In his case, that of a youth, precisely the same abrupt falling attacks have occurred as an immediate sequel to loud and unexpected sounds, and in addition typical major epileptic seizures have developed. I might also cite another case recorded at some length by Foster Kennedy, in which a boy of 17 would fall immediately to the ground on hearing a sudden noise, not by any means always a loud one. He had suffered from convulsions in infancy, and also had exhibited jerking movements of the left limbs and body when he was younger—seemingly a variety of Jacksonian epilepsy. In this case operation over the parietal cortex revealed an old, organized subdural clot, and was followed by gratifying improvement.

In respect of reflex epilepsy the examples given illustrate the same double process as in the case of the motor variants: an extrinsic stimulus of one or other kind either excites or inhibits. When in consequence of a loud sound a patient falls to the floor it is apparent his posture-maintaining mechanism has undergone inhibition, and the assumption is justifiable that the reaction takes place at mesencephalo-pontine levels. A certain analogy is permissible with the cataplectic seizures of the narcoleptic (see above); under the influence of stimuli of an emotional kind the limbs give way and the patient sinks to the ground, without loss of consciousness.

Sensory Epilepsy.—That paroxysmal manifestations should be confined to the afferent side is more than a theoretical possibility. We have already
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seen how much of a fit may be purely sensory—its aura is a sensation, simple or complex, and in some Jacksonian cases not only does a sensation march along physiological lines (precisely as do the motor symptoms) but it also may represent the whole of the disturbance. An example of sensory epilepsy is the following.

CASE 10. M. M., age 43, female. Has had for a period of years recurrent attacks, amounting sometimes to as many as 12-20 a day; at first occurring mainly at the time of the catamenia, they now bear no relation to it. A sudden feeling of faintness, and a slight flush, is succeeded by a strong olfactory hallucination, always pleasant; "it is just as if I had been passing a perfume shop." The lips then become numb, and this is followed by numbness spreading over the right side of the face, right arm, and right leg. There is a certain feeling of stiffness with the numbness, compelling the patient to make voluntary movements of the lips in order to get this feeling away. No true involuntary movement of any kind occurs. The duration of the fit is from three to five minutes. In the course of its development there always appears a faintly conscious background as of voices asking the patient "familiar questions of an ordinary domestic kind."

Careful and repeated examination has not shown that any organic basis for the symptoms exists.

The reader will observe how this case blends with the usual syndrome of uncinate or temporoprogenoidal epilepsy: as for its actual clinical type, the manifestations are limited, and confined to the sensory system. Some paroxysmal sensory disorders at higher or lower levels (e.g. some of the neuralgias) may well be classifiable with the epilepsies, and the speculation might prove of value as an aid to further observation.

Affective Epilepsy.—We may conveniently include among the sensory variants numerous cases of epilepsy characterised by the fact of their development consequent to extrinsic or intrinsic affective stimuli. They are to be distinguished from reflex epilepsy in the more technical sense by the definitely affective element of their excitants, though in another sense one might consider the process a "reflex." We should also take note that the variation is not one of epileptic content, but merely of initiation.

"Psychic epilepsy" is a term that has done hard service without ever having been either defined or delimited. "Psychasthenic fits," similarly, have received widely differing connotations (cf. Ernest Jones). It is obvious that expressions of this kind can be applied in psychopathology to fits of a hysterical nature, with which we are not here directly concerned. The reference is rather to ordinary major or minor epileptic fits activated by stimuli of the affective or emotional series. Cases of this sort constitute neither a novelty nor a rarity, and were fully recognised and described long before the war produced them again in some abundance. A typical war case is cited in a paper mentioned above; another case under personal observation may be selected.

CASE 11. H. D., male, age 21. Has had typical grand mal attacks for some two years, on an average one every week. A sister, age 30, has also suffered from typical major attacks for a number of years. One day she fell in a corner of the room in a severe
epileptic seizure; overcome with emotion, and with tears in his eyes, he went to her aid, and as he bent down, with words of endearment on his lips, he suddenly lost consciousness and had an equally severe fit on top of her.

This psychogenic or affective epilepsy is as a fact common enough, and instances need not be duplicated. In comparison with what may be termed unmotivated epilepsy, however, their number is insignificant, and I find myself in radical disagreement with Rows and Bond, who assert that in every type of epileptic case a disturbance of consciousness appears, "with which is associated an emotional state," and that "some reaction to express the emotion" can be found in every instance.

III. PSYCHICAL VARIANTS.

The term "psychical equivalents of epilepsy" is objectionable for more than one reason: if any evidence is forthcoming to suggest the same physiological processes of excitation or of inhibition lie behind the psychical symptoms as behind those of epilepsy in the ordinary sense, then the syndromes are not "equivalent" to epilepsy but are as a fact epileptic. Again, we have seen how much of an ordinary fit may consist of phenomena in consciousness (aura, sensory symptoms), belonging therefore to the psychical series, and it is inadvisable to differentiate "psychical" equivalents as though they stood for phenomena never found in the usual epilepsies. Indeed, to harmonise "psycholepsy" with epilepsy is not arduous if we adopt the general physiological principles sketched at the outset. Aldren Turner defines "psychical epileptic equivalents" as "the mental phenomena of the pre- and post-convulsive states, when they occur without convulsion or spasm." In my view this is tantamount to an admission of the identity, and not the equivalence, of the phenomena with other epileptic phenomena.

Thus conceived, the phrase must be taken to incorporate diverse symptoms occurring in petit mal, coordinated epilepsy, hystero-epilepsy, post-epileptic stuporose and confusional states, and thereby becomes devoid of any specific usefulness. Including dreamy states in the Jacksonian sense, epileptic mania, fugues and ambulatory automatisms, its perpetuation can serve no scientific or clinical purpose.

I take the chance, nevertheless, of referring briefly to an occasional epileptic concomitant, which has been supposed to form a psychical epileptic variant.

Bad Temper.—The occurrence of instances of bad temper in members of epileptic families is noticed from time to time, and has been subjected to statistical investigation by Davenport. In a number of instances collected by that observer the patient was a sufferer from both epilepsy and bad temper, while in other members of the family they occurred either separately or together. The "epileptic temper" has long been recognised and often described (Ribot, Binswanger, Raecke), and its exhibitions in prodromal and post-paroxysmal periods are familiar to the neurologist; whether substitution of bad temper for epilepsy constitutes the former an epileptic variant is not to be decided by
superficial consideration, since it occurs also in families with insanity as a feature, but without epilepsy. The accompanying diagram gives the details of a family under my observation, in which insanity, epilepsy, and bad temper are prominent traits. The interest resides largely in the appearance of bad temper in three members, none of whom has ever had an epileptic fit; but as insanity also is a familiar inheritance it rather complicates the issue.

**FIG. I.**

Davenport's conclusion is to the effect that since violent temper can be shown to occur familiarly without either epilepsy or insanity it should be regarded not as an "equivalent" of epilepsy but as due to a factor which has its greatest effect when acting on a nervous system, especially liable to the other.

**IV. VISCERAL VARIANTS.**

Obscurities arise when, finally, we approach the question of epileptic syndromes of viscerosensory and visceromotor origin. Here we must proceed warily, content to observe if we cannot yet explain. On general grounds, participation of visceral centres in epileptic manifestations must of course be conceded. The sudden pallor of many cases of petit mal, not to mention those of "nervous faints," the sphincter relaxation of minor or major fits, are instances in point. As the "discharging lesion" sweeps over different levels it would be curious to find selectivity exercised; the discharge is "brutal," as Jackson said, and does not spare mechanisms belonging to the sympathetic system, in their central connexions. What interests us for the moment, however, is the possibility of epileptic variants the outcome of physiological disorder localised largely if not entirely in sympathetic centres,
to the exclusion of sensorimotor phenomena of somatic type. One variety can be clearly differentiated: others, more fragmentary, can also be exemplified.

Vasovagal Attacks.—This name, which we owe to Gowers\(^1\) (1907), has never received the sanction of common usage; "nerve-storms," "pseudo-angina" have a certain vogue, and "anxiety neurosis" a greater one, yet for a number of reasons I prefer that of vagal or vasovagal attack. For years it has been my custom to collect cases falling under this heading, and I\(^2\) have drawn attention to them on several occasions, but the idea is not one, as far as I can gauge, that has taken its place among the conceptions of practice.

The expression "anxiety neurosis," as an inclusive term, is misleading, since it is based on the view that the manifestations, both mental and physical, which here concern us, are "the inevitable accompaniment of conflict within the personality" (Gordon\(^3\)). To this assumption I cannot subscribe. That the phenomena do in fact often enough thus arise I fully admit, but my contention is that in other circumstances they are adequately described as epileptic variants (in still others, they follow from actual visceral disease). The error has its origin in ignoring the well-established general principle that a given clinical syndrome may be of diverse etiology and of differing physiological localisation.

Speaking generally, the symptoms of the vasovagal attack are referable to transient disturbance of function mainly in the organs supplied by the pneumogastric nerve, and since implication of the motor vagus is doubtful the probability is they develop from disorder of the dorsal vagal nucleus in the medulla and of the vasomotor centre in juxtaposition. As a result, the patient complains of attacks of palpitation at the heart, difficulty in breathing, a suffocating feeling in the throat, hot flushes, icy coldness, sweating, shivering and trembling, nausea and other epigastric sensations, and often, also, an indescribably distressing sensation of impending trouble, of fear, even of imminent dissolution. One or more of these symptoms, doubtless, may occur as an episodic syndrome in the course of either functional or organic nervous disease, but in the general form already outlined they are periodic and but slightly varying, and their due recognition is a matter of clinical importance.

I shall select one or two illustrations out of a large number.

**Case 12.** N. J., female, age 28. Has suffered for nine months from attacks which are exemplified in the following excerpts from a description with which she furnished me at my request.

"I was sitting in the garden with another patient at the time and had a quiet feeling as though I did not wish to speak or take notice of anybody or anything, but seemed to be living inside myself. At the same time everything impressed itself upon my mind to an unnaturally forcible extent. I tried to take no notice but to rouse myself and think of something pleasant but could not do so as my thoughts seemed confined; even familiar objects around me, and the birds singing, seemed different and more intense."

"All the time I was conscious of a dread that something was going to happen, accompanied by a fear of death. I had a feeling of slight tingling or electricity all through me and was exhausted, cold and clammy, as though all the blood and strength had gone out of me. Then my heart began to beat rather heavily and quickly, which affected my breathing, making me take my breath in gasps. After that I started shaking all over and then a warm feeling came down over me, like the blood returning, especially in my..."
head and face, and tears came involuntarily. My feet remained cold all the time. When it had passed off I felt better in my mind but weak and shaky in my body, and depressed."

By way of amplification it may be stated that nausea and a peculiar feeling in the stomach were also noted towards the outset, and that the flush which developed as the attack subsided was usually accompanied by some perspiration.

A cousin of the patient suffered from major epilepsy. Examination of all the bodily somatic systems has been consistently negative.

**Case 13. C. C., female, age 25.** For several months has complained of attacks of "the nerves."

On investigation these turn out to consist of palpitation at the heart, difficulty in breathing, sweating, icy-cold extremities, and generalised trembling, together with an anguished feeling of impending death. The attacks vary in duration, from a quarter of an hour to as long as four hours, and after every one there is urgent and almost involuntary defecation.

Family history and objective examination are alike negative.

**Case 14. F. D., male, age 39, a member of the medical profession.** Has suffered from typical major epileptic seizures ever since his student days. These fits are severe, and are characterised by cyanosis, convulsions, tongue-biting, incontinence, etc. The aura (not constant) consists of a peculiar repetition in the left ear of a word or series of words (not always the same, and not always clearly recollected).

In addition, the patient has frequent "sensations." These also are ushered in by a well-defined repetition of words in the left ear, and consist of a sharp feeling at the heart, "something like a tug," a sense of great difficulty in getting breath, icy-cold extremities, intense fear (apparently of developing a fit); then their place is taken by flushing of neck and face, the heart beats more strongly and quickly, giving a sense of relief, the limbs shake, and pronounced upward eruption of wind always ensues.

There are no signs of organic nervous disease and no structural visceral changes.

**Case 15. I. H., female, age 10.** Has had "fits" for the last six weeks. These commence by a sinking feeling in the stomach, associated with giddiness and a form of micropsia; "everything looks quite tiny." Then come severe palpitation, icy coldness and deathly pallor. "I feel as if I were going to die, my heart keeps on throbbing so." Bad headache, cold shivers, and numbness of the right arm and right side of the face round off the attack, which lasts about a quarter of an hour.

On examination no organic nervous disease and no visceral cause for the symptoms were discovered.

This patient came again to hospital eight years later (age 18) with a history that the attacks had ceased for a period of several years. In their place another type now occurred, in which the aura was "blindness" and which from its characters was suggestive of migraine.

The above cases are chosen from a large number because they illustrate various points of significance. They may be amplified generally by reference in a little more detail to the symptoms as they unfold themselves *seriatim*.

1. The gastric or epigastric sensation is often described as though that organ were moving or turning over, associated with a sick feeling, or as though it were empty or void. "It is a creepy feeling, as if the stomach moves about"; "like being on the waves of the sea"; "as if there were a hollow in the stomach, or a hole"; "as if I were hungry and yet I don't feel hungry."

2. The cardiac sensation is highly variable, but actual praecordial or cardiac pain is unquestionably rare. Rather the patient notes fluttering, racing, thudding, thumping, of the organ: cardiac arrest is a seeming actuality. In one case, cardiac pain with radiation down the left arm took place.
Respiratory phenomena consist of tachyphoea, less often of bradyphoea, and of unpleasant subjective sensations such as a choking, suffocating, gulping feeling: "my pulse comes up into my throat and chokes me."

Concomitant vasomotor symptoms, equally variable, consist in a coldness like marble—"the coldness of death"—with shivering, trembling, shaking; a cold and clammy perspiration breaks out. Warm or hot feelings suffuse the limbs afterwards. "I feel as if all the blood had gone out of me and that I was left a stone; then I begin to glow all over."

It is of great interest to note that in not a few cases the patient voids large quantities of limpid urine after the attack: in one of the instances cited above defaecation occurred.

As for accompanying phenomena of the psychical series, we note the sensation of angoisse, of impending catastrophe, of imminent death. Words of the patients are far more graphic than a laboured description: "Oh, I'm going! I'm going!" "I think it's a death struggle and I say my prayers every time." "I feel, is this it? Is this death at last?" In other instances the feeling is brief and indefinite, yet usually with a sinister content. While fear of one or other degree is common, I have not come across any cases where a conscious cause for it existed in the patient's mind.

We also note that there is no loss of consciousness, though frequently a sense of unreality, "as in a dream," closely allied to the "dreamy state" of uncinate epilepsy. Sometimes the senses are preternaturally acute, on the other hand; the patient feels abnormally on the alert. Again, particular attention is drawn to the fact that on occasion he is conscious of a sensation of "being unable to move," as if in a trance. "I struggle inwardly, but nothing happens. I try to call out, but I say nothing. I feel if only I could move all would come right." "I hear everything they say to me but I cannot utter a single sound."

This latter circumstance links the condition in some instances to what I have shown is characteristic of numerous narcoleptic and cataplectic fits—viz., inability to move or to innervate, and constitutes a phenomenon of inhibition accompanying those of release, as in some other epileptic variants.

These vasovagal fits frequently arise in cases with a family history of epilepsy, migraine, or insanity. Thus one of my patients suffered from characteristic attacks for a number of years: his mother's brother was insane, and two of his father's brothers had epilepsy: his mother, and a brother, were both martyrs to migraine. Sometimes no evidence of neuropathic inheritance is forthcoming, as also in many epileptic individuals. I am not concerned here with the appearance of anxiety neurosis as a sequel to "conflict within the personality," hackneyed though such a development is thought to be. Careful investigation fails to reveal any trace of "conflict" in the cases narrated above: and the "coitus interruptus" theory is of course inapplicable, not merely in the case of juveniles, but also in others in which the practice has never been indulged. To my mind the theory that vasovagal phenomena can in many
instances be considered epileptic variants does no violence to the facts and is consonant with what we know of epileptic manifestations otherwise. I do not believe we should expect the reactions of visceral centres in the neuraxis to be identical with those of somatic centres; allowance must be made for differences, histological and physiological, in the ganglionic collections that are the seat of "discharging lesions." The "extended" nature of the vasovagal attack cannot of itself form an insuperable obstacle in the way of acceptance of the hypothesis. If a faint is a bulbar inhibitory fit, a vasovagal attack can be a bulbar fit of a less rapid, less "brutal," more coordinated kind.

Precaution is desirable, on the other hand, before a given case of this bulbar syndrome can be entered as epileptic. Under very differing etiological circumstances phenomena resembling those that have been sketched can make their appearance. Shock, anaphylaxis, disorders of intracranial circulation, commotio cerebralis, and so forth, are pathological conditions in the course of which bulbar symptoms can and do arise. Attention was directed by Lévy long ago to the case with which bulbar disarray (affolement bulbaire) develops episodically, while in recent years among the sequela of epidemic encephalitis have occurred complex types of respiratory disturbance only a minority of which are classifiable with lowest level (ponto-bulbar) fits, though few if any are strictly comparable with the vasovagal attack.

Other Visceral Fits.—Other types of recurring seizure in which symptoms referable to central visceral disorder make their appearance have come under notice from time to time, and some are rather difficult to classify, as the subjoined examples prove.

Case 16. C. S., male, age 11. A very intelligent boy, and a scholarship winner, he has had a series of attacks during the last 12 months which are heralded by deep and incessant yawning, for perhaps half an hour. He turns deadly pale and then starts to vomit, while violent frontal headache develops. The whole thing may last for rather less than an hour.

Objective examinations has repeatedly proved negative. There is no family history of epilepsy, migraine or other analogous condition.

Case 17. C. L., male, age 25. Has had peculiar fits for about one year, recurring on an average at fortnightly intervals. They are constituted by continual yawning, with constant voiding of urine, and are followed by violent giddiness, headache, and occasional vomiting.

There is a family history of epilepsy. No organic signs have been found on repeated examination.

The explanation of these cases is not simple. One might hazard the speculation that they represent some disturbance of third ventricle visceral centres concerned in the regulation of sleep and of water-interchange, with irradiation to visceral centres situated in the floor of the fourth ventricle.

As clinicians, we must be prepared to meet with syndromes, such as these, which do not fit readily into hard-and-fast schemata. Not enough is known of the normal functions of the sympathetic system to enable detection of its disorders to be recognised with precision. We may only suspect certain visceral syndromes to be the outcome of functional disarrangement of
abdominal plexuses; we still are largely in the dark as regards those derived from changes at higher visceral levels. It is our task, therefore, with open mind to observe symptoms, collect data, and collate instances, and imagination may supply the light whereby we shall see eventually how heterogeneous syndromes slip into their place in the physiological puzzle.

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